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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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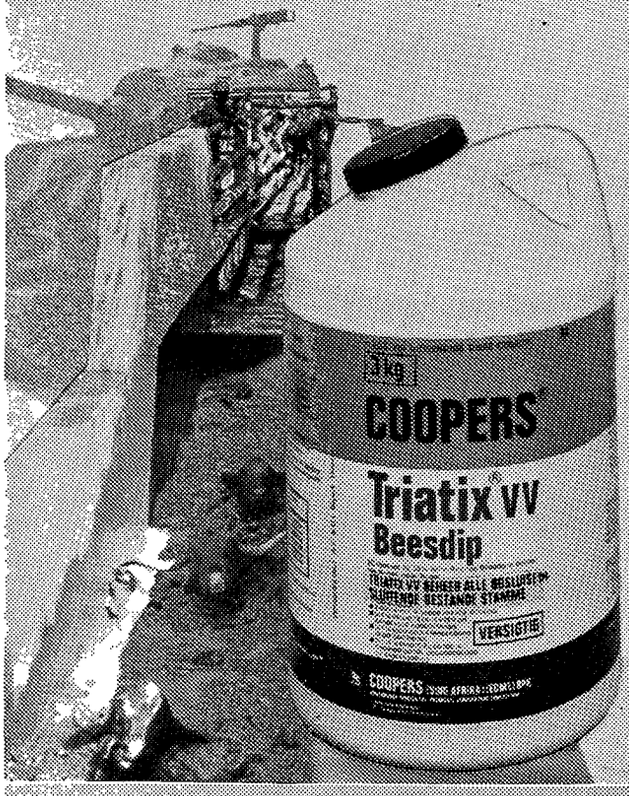

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CORRIGENDA

1. "Microbiological investigation of meat wholesale premises and beef carcases in Johannesburg" – P. J. Meara, Leah N. Melmed and R. C. Cook Vol 48 No 4 p 255–260: Table 8, p 259, the heading of the right hand column should read **Number of** *S. aureus/g*.

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REVIEW OORSIG

SOME ASPECTS OF THE PATHOGENESIS AND COMPARATIVE PATHOLOGY OF TOXOPLASMOSIS

G.V.S. TURNER*

ABSTRACT: Turner G.V.S. Some aspects of the pathogenesis and comparative pathology of toxoplasmosis. *Journal of the South African Veterinary Association* (1977) **49** No. 1, 3–8 (En) Dept. Vet. Prev. Med., College of Vet. Med., Ohio State University, 1900 Coffey Rd., Columbus 43210, Ohio, USA.

Some aspects of the pathogenesis and comparative pathology of toxoplasmosis are described. The general pattern of infection, with or without necrosis, and tissue cyst formation as it occurs in all species is dealt with. The wide pathological manifestations of toxoplasmosis as seen in sheep, cattle, pigs, horses, dogs, cats, chinchillas and man are reviewed.

INTRODUCTION

Toxoplasmosis is a disease entity caused by the protozoan parasite *Toxoplasma gondii. T. gondii* was first reported simultaneously in 1908 by Nicolle and Manceaux in a North African rodent, *Ctenodactylus gondii*, and by Spendore in Sáo Paulo in the rabbit[20 40 56]. The name of the parasite is derived from the Greek *toxon*, meaning bow or arc, alluding to their lunate shape and from the North African rodent. *T. gondii* has a world wide distribution and has been isolated from the tissues of many species of mammals and birds[3 4 6 7 10 13 16–18 20 23 28 30–32 44 50 51 56 57 61 67 73].

Numerous serological surveys have been carried out in various parts of the world, the findings of which confirm the high prevalence of toxoplasma antibodies in animals and man[1 5 9 23 29 32 36 39 51 52 56 57 61 64 67 70 74].

Both in animals and man, toxoplasmosis covers a wide clinical spectrum and occurs either as a congenital or an acquired infection. There is also a high incidence of asymptomatic infections in mammals and birds[8 9 18 22 24 29 32 40 45–49 52 56 58 63 66 73]. The various clinical manifestations of toxoplasmosis with its concomitant pathology can be regarded as being of interest, not only to the protozoologist and pathologist, but to the veterinary and medical professions in general.

For a better understanding of the pathogenesis and pathology of toxoplasmosis it is necessary to outline the life cycle of *T. gondii.*

LIFE CYCLE

The life cycle of *Toxoplasma* is divided into 2 separate cycles, namely an entero-epithelial cycle and an extra-intestinal (tissue) cycle. The coccidian-type entero-epithelial cycle with oocyst production occurs only in cats and other feline species[14 19 20 28 32 40 43 50]. The extra-intestinal cycle occurs in both feline and non-feline hosts and constitutes the entire cycle in non-felines[12 18 20 28 50]. The three known infective stages of *Toxoplasma* are bradyzoites (tissue cysts stage), tachyzoites (proliferative forms in tissue during acute infection) and sporozoites (in sporulated oocysts).

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Entero-epithelial cycle:

The feline host is infected by the ingestion of tissue cysts (containing bradyzoites) or proliferative forms (tachyzoites) in tissues during acute infection or by the ingestion of sporulated oocysts originating from feline faeces. Multiplication takes place in the epithelial cells of the small intestine with the eventual formation of oocysts. The oocysts are detached from the intestinal epithelium and are discharged with the faeces. The oocysts are non-infective when unsporulated, with sporulation taking 1 to 5 days[1 11 12 19 20 25 27 28 32 50 68 70 71]. Millions of oocysts may be shed in a single stool and may remain infective for a year in warm, moist climates or longer in cooler climates[20 21 29 32 40 53 75].

Extra-intestinal cycle:

Infection can occur from the ingestion of sporulated oocysts of feline origin or the ingestion of tissue cysts or the transplacental infection of the foetus with proliferative forms (tachyzoites) after ingestion of sporulated oocysts or encysted tissue organisms by the pregnant female[2 20 24 25 28 35]. A parasitaemia then occurs with cyst formation taking place as the host's immunity builds up[20 28 32 33]. Tissue cysts are characteristic of the chronic infection and may occur in any tissue[37]. The tissue cysts may persist for the life of the host[11 20 24].

PATHOGENESIS

When a mammalian or avian host is infected by the ingestion of sporulated oocysts or tissue cysts toxoplasmosis begins with the development of tachyzoites in the lamina propria of the intestine[32]. A parasitaemia then develops with the tachyzoites being disseminated to various organs (including the placenta) in macrophages, lymphocytes, granulocytes and in free forms in the circulation[20 24 25 28 33 37 59].

T. gondii, according to some authors, does not appear to show a special affinity for any particular tissue or cell type[37].

The tachyzoites localize intracellularly in various tissues and begin to proliferate. The localization may be followed by active lesions in the affected tissue or encystment of the *Toxoplasma* organisms in which form

they may remain viable for a long time[20 28 32 33 59]. From about 8 to 16 or more tachyzoites accumulate in the host cell before it disintegrates and new cells are infected.

The typical response of all tissues to proliferating organisms is necrosis[33]. In certain cases *T. gondii* appears to actively invade the vascular walls resulting in early damage to capillaries, arterioles and venules, with increased vascular permeability and perivascular oedema[37]. Plasma cells are not uncommon in the perivascular infiltrates, and some authors think that an allergic response may play a role in the pathogenesis of toxoplasmosis[20 28 32 37].

This general pattern of infection and cyst formation occurs in all species. The severity of the infection is determined by the degree of cellular necrosis, which will depend on the virulence of the strain; the organ or tissue involved; the number of proliferating organisms; the species of animals infected; its age and resistance; and the degree of hypersensitivity, if any[20 24 28 32 33].

The tissue-cysts may persist for the life of the host as a sub-clinical infection. Should, for various reasons, the host's immunity wane, cyst organisms may be released and a renewed proliferation of tachyzoites will be initiated, causing a localized or generalized relapse[20 46]. Increasing numbers of cases of toxoplasmosis are being recorded in humans undergoing therapy for malignancies, in patients on immuno-suppresive agents and in people suffering from an immuno-deficient disease[20 46 50 63].

Information concerning the pathogenesis of placental and foetal toxoplasmosis in humans and animals is scant. The infection in the dam is usually asymptomatic. It may be assumed that placental and foetal infection can occur at any time during an active infection where tachyzoites are in the general circulation[33 62]. Whether the female host chronically infected with tissue cysts can give rise to placental and foetal infection is a controversial point. Some workers, however, claim that premunition prevents infection of placenta and foetus during successive pregnancies[20 24 28 56].

It has been claimed that invasion of the placenta may occur in a chronic infection due to rupture of endometrial cysts during implantation[33 62]. It is noteworthy that occasional cases of toxoplasmosis have occurred in lambs of ewes that had high antibody levels to *Toxoplasma* at mating[24].

PATHOLOGY

In animals, such a wide diversity of pathological manifestations has been attributed to infection with *T. gondii* that it is difficult to ascribe limits to this condition. The organs and tissues most often affected are the brain, myocardium, lymph nodes, lung, intestinal muscularis, pancreas or liver[8 11 24 29 45 48 49 59 65]. A high incidence of asymptomatic infections occur.

In tissue sections, tachyzoites and bradyzoites of *Toxoplasma* may be crescent-shaped, but also occur in rounded and ovoid forms. They are most frequently found in the cytoplasm of cells but may be free. A large number of the organisms may be encountered in a single cell or may be contained by a thin membrane which is believed to be the remnant of the wall of a host cell by some who speak of the structure as a "pseudocyst". Others consider the membrane to be a product of the parasite and regard it as a true cyst[33 55 59].

Toxoplasma organisms are not always readily demonstrable but may be seen in microscopic sections of animal tissues in which the injury to the host is severe, minimal or even non-recognizable. In active toxoplasmosis the microscopic findings in a particular organ are reasonably characteristic and diagnosis is therefore not entirely dependent on the demonstration of the parasite[59].

In the *brain*, active infection is indicated by diffuse non-suppurative infiltration of the brain parenchyma; lymphocytic cells accumulate within the Robin-Virchow spaces and are scattered throughout the parenchyme. Vacuoles may occur in the white matter. *Toxoplasma* organisms may be found scattered singly or in pairs in the parenchyma. Focal areas of necrosis are seen in the grey and white matter. After glial activation has taken place nodules form. Proliferation of perivascular reticuloendothelial cells is prominent and tends to make the vessels very obvious. They contribute to the scarring of the necrotic foci so that both glial and fibrous elements are present in the nodules[33 59].

Affected *liver* often shows large sharply delimited areas of coagulation necrosis involving any part of the hepatic lobules. Small numbers of organisms may be found singly or in pairs within liver or Kupffer cells and later in cysts containing large numbers of organisms[33 59].

The *lung*, when involved, exhibits rather striking changes, particularly in the alveolar walls; the lining becomes cuboidal or columnar and very rich in cells, suggesting in this respect the appearance of foetal lung. The alveoli are filled with large mononuclear cells and leukocytes with aggregations of *Toxoplasma* organisms in the cells lining the alveoli. These lesions have a nodular distribution throughout the lung, appearing grossly as small, grey, tumour-like masses scattered throughout one or all the lobes[33 59].

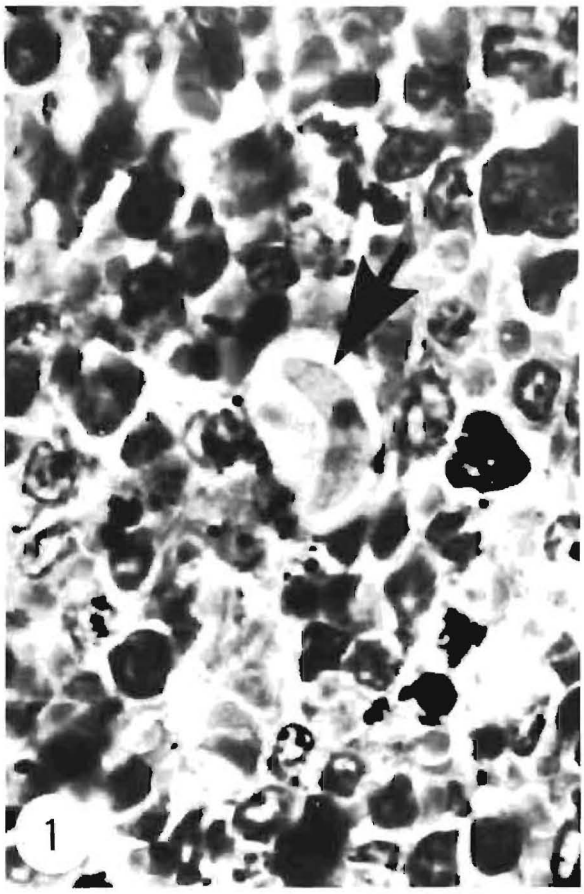
The *lymph nodes*, particularly those draining an affected organ or area, are commonly involved in active cases of toxoplasmosis. They are usually enlarged, firm in consistency and congested. Extensive coagulation necrosis is seen microscopically and organisms may be found adjacent to these necrotic areas, particularly in endothelial cells of veins, but may be with the cytoplasm of monocytic cells or free in the tissues[33 59].

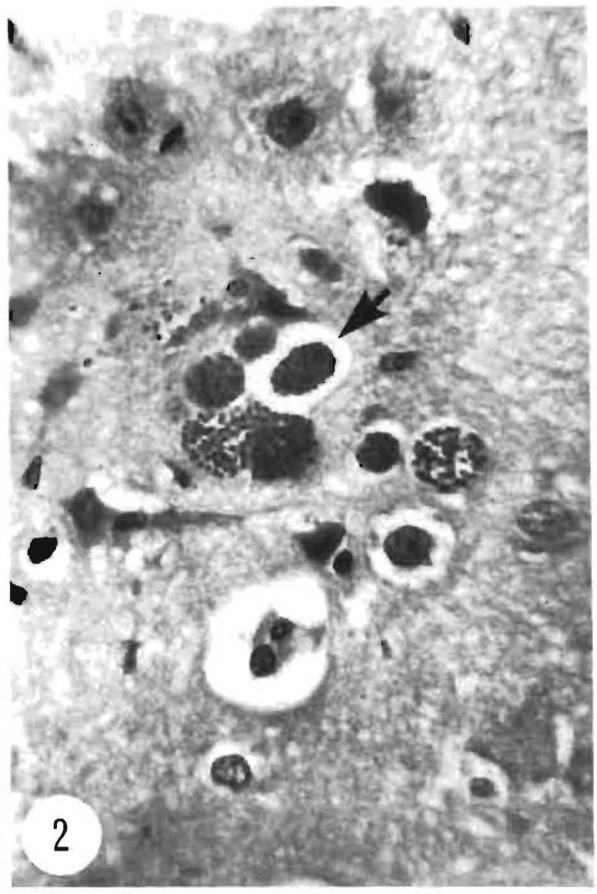
Ulcers in the *intestine*, presumably resulting from necrotic changes in sub-mucosal lymph nodules have been described. *Toxoplasma* may invade the muscularis of the intestine where a necrotizing lesion followed by production of granulation tissue results in large, grossly detectable granulomatous nodules. *Toxoplasma* organisms are noted in small or large groups in the muscularis and the granulation tissue.

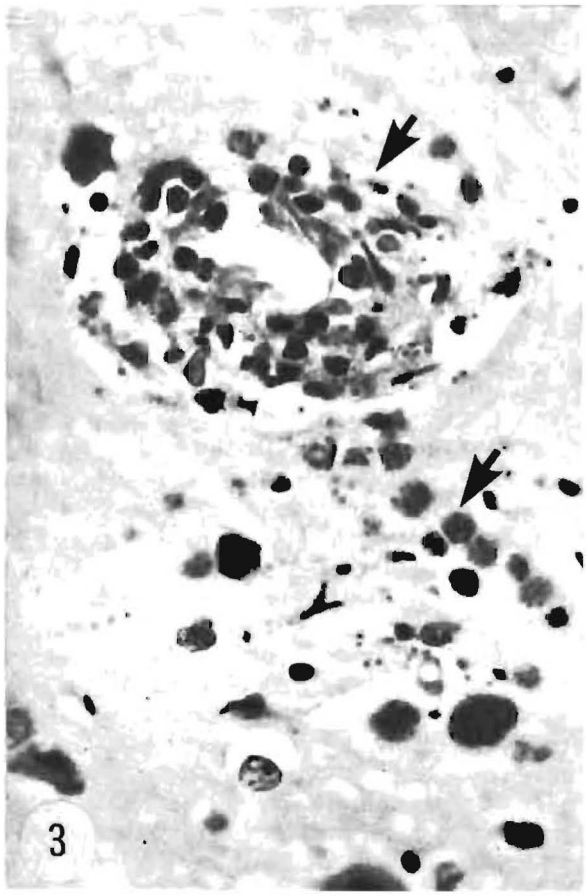
In the affected *pancreas*, acute necrosis, intense lymphocytic infiltration, oedema and swelling may be seen. Numerous organisms in both duct and acinar cells are seen[33 59].

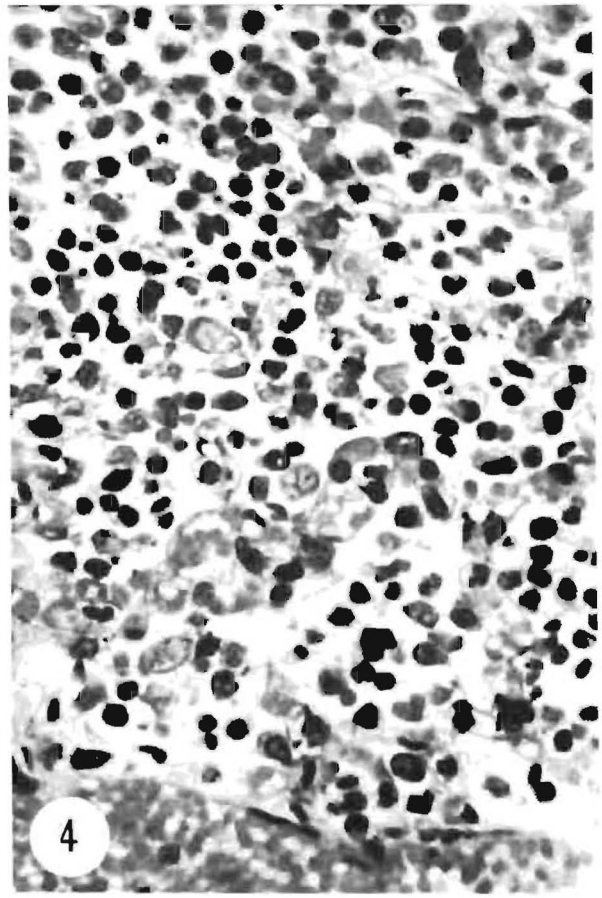
The *myocardium* is frequently invaded by *Toxoplasma* with small or large groups of organisms within the cytoplasm of the cardiac muscle cells. Focal necrosis with severe lymphocytic infiltration or very little signs of inflammation may be seen[33 59 66].

It is apparent that *Toxoplasma* infections may be manifested in a variety of forms. This is illustrated by the pathology of toxoplasmosis in domestic animals and man.









Spleen. Proliferative form of *T. gondii*.

Brain. *Toxoplasma* tissue cysts with no cellular reaction.

Fig 3 Brain. Severe vasculitis associated with *Toxoplasma* cysts in the perivascular tissue.

Fig 4 Lung. Infiltration of alveoli with macrophages and neutrophils (from confirmed case of toxoplasmosis).

Sheep:

Abortion due to toxoplasmosis is probably more important in sheep than in any of the other animals. Abortions often occur late in pregnancy.

Affected foetuses show no significant gross lesions. But histologically the parasites can be demonstrated and isolated from the myocardium, lung and brain. The foetal placenta bears what are probably characteristic lesions. The cotyledons are bright to dark red as compared with a normal deep purple colour; scattered amongst the foetal villi are numerous white flecks or small soft white nodules 1–3 mm in diameter. The villi are oedematous and there is focal necrosis and desquamation of trophoblastic epithelium. In more extensive cases there may be caseous and calcified cotyledonary nodules. The organisms are readily identified microscopically from the placental material, either free or in cysts[3 4 33 38]. The intercotyledonary placenta usually only shows oedema[33].

The neuropathology of ovine toxoplasmosis has been described in detail by Koestner[38]. Twenty-five sections at various levels of the central nervous system (CNS) were taken. The lesions in the CNS were found to be equally distributed and no predilection site was found. Foci of necrosis with numerous extracellular proliferating organisms were seen[38].

Microglial proliferation occurs with glial nodules being formed in the subacute and chronic stages. Glial nodules basically consist of microglia, activated oligodendroglia, astrocytes and monocytes. The *Toxoplasma* organisms occur intracellularly and in cysts at this stage. A striking feature is the mineralization of the vascular walls of the cerebral and meningeal vessels[38].

There are very few reported cases of clinical forms of toxoplasmosis in adult sheep, other than outbreaks of abortion and perinatal mortality. McErlean reported a case of two sheep showing progressive paralysis. No macroscopic lesions were noted at post mortem examination but extensive perivascular cuffing in the spinal cord associated with several *Toxoplasma* cysts was observed[41].

Cattle:

Toxoplasmosis is exceptionally rare in cattle[38 44]. Cattle are very difficult to infect with *T. gondii* and the CNS seems to be particularly resistant[38 44]. Koestner claims that the neuropathology in cattle is the same as that described for sheep[38].

Congenital toxoplasmosis has been known to occur in calves[38 58 72]. Affected calves showed occasional CNS lesions which were accompanied by oedema and perivascular gliosis[38].

Pigs:

Although *Toxoplasma* infection is prevalent in pigs in all parts of the world, the infected host seldom shows any pathological changes. Organisms in pigs without obvious lesions are frequently isolated from striated muscle, brain, lung, stomach and large intestine[34].

Experimental evidence suggests that infection during the 3rd month of pregnancy will result in foetal death and congenital toxoplasmosis in any piglets born alive[72]. Clinical cases of toxoplasmosis have been reported in pigs, occurring mainly below the age of 9 weeks. The pathological changes in these particular cases were as follows: hydrothorax, hydropericardium, ascites, focal necrosis of liver, fibrinous peritonitis, catarrhal pneumonia and enteritis. Parasites were easily demonstrated in wet or stained preparations of the affected organs[17].

Horses:

Not much is known about toxoplasmosis as a disease entity in horses. Cusick *et al.* described toxoplasmosis in two horses that showed progressive paralysis of the hindquarters and a marked myelomalacia of the spinal cord at autopsy[8].

Dogs:

A large number of dogs are regarded as having benign asymptomatic infections[37 72].

Congenital toxoplasmosis has been reported in dogs[58].

There is evidence that both immaturity and a concurrent distemper infection increases the susceptibility to toxoplasmosis. Whether distemper lowers the resistance of the host to enable primary toxoplasmosis to become established or whether it encourages reactivation of a latent infection is not known[72].

There is a considerable variation in the severity of toxoplasmosis when seen in dogs[33 37 58].

The following pathological lesions have been recorded in dogs:

Lungs – are usually affected and often manifest the most pronounced lesions. The lesions vary from small irregular areas which are grey in colour and firm in consistency, to interstitial pneumonia or focal fibrinous pneumonia. The acute interstitial pneumonia which frequently occurs strongly suggests the diagnosis. There is a adenomatoid hyperplasia of the alveolar cells, many of which contain organisms[33 37 58].

Liver – numerous areas of focal necrosis may occur[33 37 55].

Lymph nodes – these are often enlarged and are congested with focal areas of necrosis[33 58].

Spleen – enlarged, with numerous foci of necrosis in the parenchyma in which organisms can be demonstrated[33 58].

Pancreas – haemorrhages and necrosis[33].

Intestine – acute duodenal ulcers, the involved segment of intestinal wall being thickened with haemorrhages and oedema[33].

Retina – in the dog inflammatory changes have been reported as being predominantly in the retina rather than in the choroid (opposed to the classic chorioretinitis seen in man). Haemorrhages and inflammatory exudate with mononuclear cells and small foci of necrosis in the retina are seen adjacent to *Toxoplasma* organisms. Perivascular cuffing with mononuclear cells in the retina and its optic fibre and ganglion cell layers may also occur[33].

Brain and spinal cord – Koestner considers the incidence of brain and cord lesions in canine toxoplasmosis to be high and of great diagnostic significance. *Toxoplasma* cysts may also occur in the CNS with no signs of any tissue response[37]. Young dogs appear to be more susceptible and congenital infections result in the most severe and extensive lesions. Koestner describes the neuropathology of canine toxoplasmosis in great detail. Lesions are found in all parts of the CNS. Acute cases

are characterised by vascular damage and focal necrosis; by glial nodules and repair in chronic cases. Glial nodules contain microglia, oligodendroglia, astrocytes and monocytes[37].

Toxoplasma organisms appear to actively invade the vascular walls the Virchow-Robin spaces and the adjacent nervous tissue. The parasitic invasion results in necrosis[37]. At the margin of the lesion numerous cysts, ruptured cysts and extracellular proliferating organisms can be seen. The meninges may show a slight infiltration of lymphocytes and plasma cells[37].

Cats:

Apart from playing a role as the source of *Toxoplasma* oocysts, the cat can also develop the extra-intestinal form of the infection and show signs of acute toxoplasmosis as well as chronic and asymptomatic infections[47 72].

The lungs are the most frequently and most severely affected organs and at post-mortem examination these show nodular necrotic lesions, interstitial pneumonia and oedema[15 473 72]. The CNS may also be affected[15 72]. The liver, myocardium and lymph nodes show focal areas of necrosis[47]. In young kittens the infection may be fatal[15]. Ocular lesions have also been described in cats[65].

Impression smears from affected lungs may in some instances reveal organisms[47].

Chinchillas:

In an outbreak of toxoplasmosis in chinchillas in South Africa with a 17% mortality the following lesions were recorded: focal necrosis of liver and myocardium, interstitial pneumonia and, in the brain, parasitic cysts associated with focal gliosis and perivascular infiltration by plasma cells[16].

Man:

Both congenital and acquired forms of human toxoplasmosis occur. The *acquired form* covers a wide clinical spectrum. *Lymphadenopathy* is a frequent manifestation and toxoplasmosis is accepted by many as the most frequent cause of lymphadenopathy in which the Paul-Bunnell test for glandular fever is negative[9 22 24 40 46 56 60 72]. Ocular lesions (especially chorioretinitis), myocarditis, pneumonia, pericarditis and polymyositis have all been associated with the acquired form of human toxoplasmosis[9 18 22 40 46 54 56 60 66 73]. There is also a high incidence of asymptomatic infections in man[18 22 24 52 56 61 73].

In human medicine the *congenital form* of toxoplasmosis is of the utmost importance. The various clinical and pathological manifestations include the following: encephalitis, chorioretinitis, hydrocephalus, pancreatitis, myositis, necrosis of lymph nodes and various osseous changes[19 22 32 40 42 59 60 73]. Calcification, particularly in the walls of the lateral ventricles of the brain, may cause obstructive hydrocephalus[72]. Radiologic examination of affected bones reveals abnormalities in the process of enchondral ossification. It appears as if, in these particular cases, the inflammatory cells interfere with the blood vessel invasion of the spaces between the calcified cartilage cores, and the remodelling by osteoclasts is impeded.

CONCLUSION

Although toxoplasmosis is a clinical disease that occurs rather sporadically, the clinical and pathological manifestations can be severe in the congenital and acquired forms. In domestic animals the disease is of the greatest economic importance in sheep.

From the foregoing it is apparent that both medical and veterinary clinicians and pathologists should include toxoplasmosis in their differential diagnosis where an immediate diagnosis is not possible.

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EMBRYO TRANSFER IN CATTLE: AN EVALUATION OF THE CURRENT SITUATION*

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ABSTRACT: Bouters R.; Dhondt D.; Coryn M.; Vandeplassche M. Embryo transfer in cattle: An evaluation of the current situation. *Journal South African Veterinary Association* (1978) **49** No. 1, 9–12 (En) Fac. Vet. Med; State Univ., Ghent, Belgium.

A technique for the transcervical recovery of ova in cattle is described. The donors and recipient pool consisted of 122 cows of different ages and 4 different breeds. Of these 102 cows were superovulated. For recovery of ova an apparatus consisting of a metal catheter glued to the inside of a 3-way Foley catheter was used. The technique used for embryo transfer was basically the same as for collecting the embryos.

The 102 donors yielded at slaughter a total of 956 *corpora lutea* i.e. an average of 9,4 per cow. Of the ova released 294 or 30% were recovered on transcervical flushing of the uterus. 40% of the inovulated cows conceived. The future development of inovulation in cattle is discussed on the basis of the results recorded and on those previously documented.

INTRODUCTION

The transplantation of fertilised bovine eggs has recently received considerable attention from both research workers and the commercial world. Almost 30 years has elapsed since Rowson and his group in Cambridge started their first experiment on surgical egg transfer in the bovine. Since that time several laboratories over the world have been involved in basic and applied research on this problem, the outcome of whose efforts has been extensively reviewed[7 8 9]. Nevertheless, the possible introduction of the embryo transfer technique in cattle breeding is still controversial and, moreover, it is still far from being the cheap, fast and effective method for genetic improvement in cattle that was visualised at the beginning of the first experiments.

In the last decade however, some A I organisations and veterinary practices have started to offer their services to cattle breeders on a commercial basis. A critical analysis of the costs involved and of the genetic gain achieved clearly points to the fact that these units can only survive under circumstances where very expensive "exotic" breeds are used and where the owner may more easily be persuaded to carry financial burdens to achieve more rapid multiplication of the imported animals.

Referring to our own experimental work, we anticipated from the beginning that a primary condition for the introduction of the technique in the field would be the development of a bloodless non-surgical approach both for the collection and transfer of the ova. In this paper the results of 2 year experimental study on the non-surgical method will be presented and compared with those obtained by other laboratories using similar or different techniques.

MATERIAL AND METHODS

1. Donor and recipient pool:

A total of 122 cows were used, 102 as donors, 20 as recipients. These animals were purchased from among slaughterhouse material. No details were available regarding the reason for culling or their reproductive status. The only criterion for being included in the experiment was a normal genital tract with functional ovaries. The donor pool was heterogenous and constituted animals of all ages and four different breeds (36 Holsteins, 40 of the Blue-white breed of Middle Belgium, 42 of the Red-white breed of East-Flanders and four of the Red breed from West-Flanders). The animals were kept indoors all the time at the veterinary clinic and were checked daily for oestrus. In addition blood samples were collected every second day from the coccygeal vein from each of the cows and progesterone levels determined, using a modified radio-immuno-assay technique.[14]

2. Superovulation:

To induce superovulation, a standard procedure was used. On Day 10 of the cycle (Day-O being the day of standing heat) eight donor animals received a single intramuscular injection of 2 000 units PMSG (Folligon, Organon). A further 94 donor animals received 3 000 units of the same product. The batch of PMSG was previously tested for its effectivity in immature rats and the same batch was used throughout the experiment. On Day 12 (2 d following PMSG treatment), an i/m injection of either 37,4 mg Prostin+ (Upjohn) or 1 mg of Estrumate+ (ICI) was given.

The animals were inseminated with deep-frozen semen in ministraws 8 h after the first observed signs of heat. Insemination was repeated every 12 h until no further signs of oestrus could be observed. As a rule the induced oestrus period occurred 48 h after the prostaglandin injection and lasted for 36–48 h so that most of the donors were inseminated three or four times during the oestrus period.

3. Transcervical recovery of ova:

Recovery of the ova was performed on Day 6 or Day 7 after the first insemination. The donors were placed in stocks and an epidural anaesthesia was administered with 4 ml of a 2% xylocaine solution. Air and faeces were removed from the rectum before complete relaxation of the anal sphincter had occurred. The perineal area was then thoroughly washed and disinfected with alcohol. A telescopic embryo collector was then guided under rectal control, through the cervix into one uterine horn. This apparatus consisted of a metal catheter (A, Fig. 1), length 500 mm, diameter 3,4 mm and inner diameter 2,7 mm which was glued to the inside of a 3-way Foley catheter No 20 (B, Fig. 1). Once inside

*Financial support: IWONL-CSVH, Grant No 2208A.

+The generous supply of these drugs by the Upjohn and ICI companies is gratefully acknowledged.

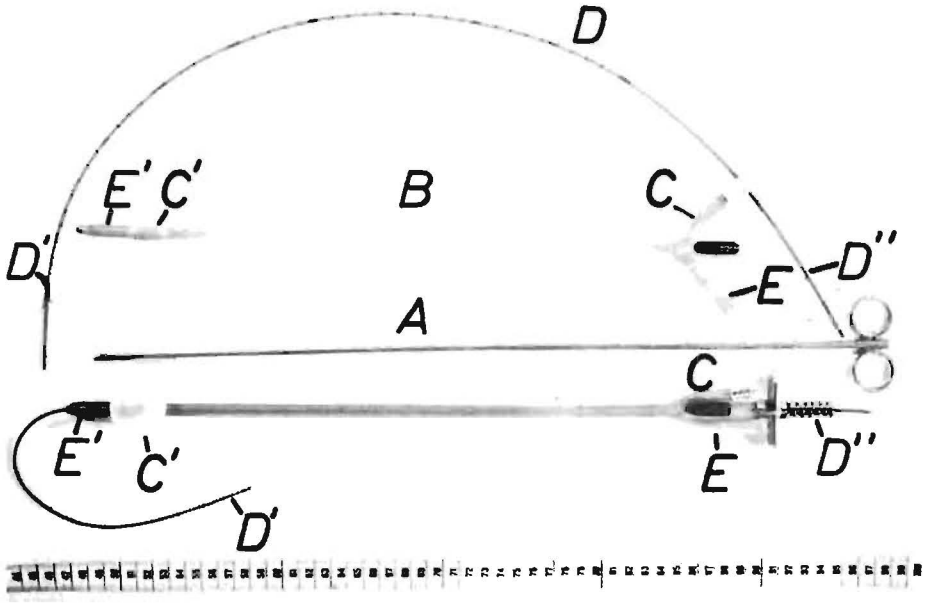

Figure 1. Apparatus used for the collection of embryos

the uterine horn, the balloon (C, Fig.1) was inflated with 10–15 ml air and a thin urinary catheter of the type generally used for male dogs (D, Fig. 1) was introduced under rectal guidance towards the utero-fallopian junction of the horn.

The assembled unit as used is illustrated in Fig. 1.

The flushing medium, consisting of Dulbecco's PBS (phosphate buffered saline), was then introduced via inlet E either as an interrupted or a continuous flow and collected via outlet D. When transplantation for specific embryos was planned, the medium was further supplemented with 20% foetal calf serum. An average of 5 x 50 ml of medium was perfused before the embryo collector was removed and guided into the other uterine horn and the same procedure repeated. After flushing, the medium was collected in conical 100 ml glass containers. The cells and debri were allowed to settle for 20 m, then 5 ml was withdrawn from the bottom and examined under a stereo microscope (Wild M8) for the presence of embryos. All donor animals were slaughtered within 24 h following collection and the number of *corpora lutea* (CL) and follicles on the ovaries counted.

4. Transfer of embryos:

Twenty embryos in the blastocyst stage were transferred to 20 recipient cows, the oestrus cycle of which had been synchronized with those of the donor animals by a single injection of 37,5 mg Prostin or 1 mg Estrumate. The luteolytic drug was given on the same day to both donor and recipient. Oestrus behaviour was found to be much less pronounced in the recipient cows than in the superovulated donors. A few cases of silent heat were seen and in these animals heat was presumed to have occurred 3 d following prostaglandin administration. Although some experiments were conducted on an *in vitro* culture of bovine embryos, only freshly collected eggs (within 4 h after flushing) were transplanted. In each recipient a single embryo was transferred into the uterine horn ipsilateral to the *corpus luteum*. The technique used for embryo transfer was basically the same as the technique described for collecting embryos, except that a thin metal catheter was used instead of the thick Foley catheter. To eight of the recipients 200 mg of a uterus relaxant (isoxsuprine lactate, Duphospasmin, Phillips Duphar) was given immediately prior to inovulation while 12 recipients were left untreated.

RESULTS

1. Superovulation:

At slaughter the 102 donors yielded a total of 956 *corpora lutea* with an average of 9,4 per individual cow. There was great variation amongst individual cows, the number of *corpora lutea* ranging between 0 and 42. In 17 or 16% of the 102 donors, no ovulation had taken place. The same applied to the number of stimulated follicles still present on the ovaries at Day 6-7. The donor pool had a total of 660 follicles or an average of 6,5 follicles per donor, with a variation of 0 and 40 amongst them.

No correlation was found between the number of follicles and the number of *corpora lutea* present in the same animal. In 12 cows all having 10 or more follicles, a total of 173 or an average of 10 CL per donor were recorded, whilst in 45 cows with no follicles still present on the day of flushing, a total of 448 or an average of 10 CL per animal were counted.

Neither breed, age, nor the stage of lactation of the donor had an influence upon the ovulation rate or proved to provide a practical parameter for predicting the ovulatory response. In those animals in which a low progesterone level was recorded at the moment of PMSG injection, there was a strong tendency to a poor ovulatory response or none at all. Of the 17 donors with no CL present at the time of slaughter, 11 (or 65%) had levels less than 2 ng progesterone per ml blood on Day 10, the day on which PMSG was administered. A reduction of the dose of PMSG from 3 000 to 2 000 U reduced the number of ovulations to 24 in eight animals (mean three per donor) but did not influence follicular development since 85 follicles (mean 10,5 per donor) were counted at slaughter.

Fig. 1 reproduced with permission from the *Vlaams Diergeneeskundig Tijdschrift.*

2. Recovery of ova:

From a donor population in which 956 *corpora lutea* had been induced, only 294 embryos were recovered. If every CL corresponded with an embryo entering the uterus, an overall recovery rate of 30% would have been achieved. When the results are critically analysed, however, it soon becomes evident that two groups of animals averaged figures far below this value.

The group of 14 donors with progesterone levels below 2 ng/ml at the moment of superovulation produced a total of 23 CL (mean seven per donor) of which only three could be recovered. The group of 17 donors with more than 10 follicles on the ovaries at the moment of recovery illustrates this more clearly. Although they had a good ovulatory response with a total of 183 *corpora lutea,* only 16 or 19% of the total could be flushed out.

When the results obtained in the two previously mentioned groups are deducted the remaining 71 donors totalled 275 or 37% of the number of embryos found in the uterine washings. Expressed as the number of embryos per donor, an average of 2,9 embryos per donor animals were recovered. Taking into account only animals in which at least one ovulation had occurred, the average number of embryos recovered increased to 3,5 embryos per donor.

3. Transfer of ova:

Of the group of 12 recipients not pre-treated with isoxuprine lactate, five (40%) became pregnant following a single ovum transfer. Of the seven animals returning to oestrus, all except two had normal oestrus cycles (19 and 22 days) and no inflammatory response of the endometrium had been recorded. In two animals an oestrus interval of 46 and 54 days respectively was recorded. During this time the progesterone levels remained high and the *corpora lutea* were found to be fully developed. In the second group of eight animals pre-treated with isoxsuprine lactate, only one of the blastocysts transplanted, developed into a normal pregnancy (15%). The remaining seven animals returned to oestrus after a normal oestrus cycle (18–24 d)

DISCUSSION

The outcome of these experiments, with an average of about 10 ovulations per donor, a non-surgical recovery of 3,5 embryos per ovulated donor and a pregnancy rate after non-surgical transfer of 40% in animals not pre-treated with an uterus relaxant, compares favourably with data published[1 4 6 11 13]. Both Rasbech[10] and Elsden, Hasler & Seidel[5] reported more favourable recovery rate (up to 6,9 per donor) but their material was highly selected for reproductive fitness and were not slaughtered after flushing, which makes an accurate estimation of the number of CL induced questionable. The observation that the recovery of embryos reported with surgical techniques can be as high as 75% of the number of ovulations is not surprising. Most of the surgical interventions are performed on Day 3 or Day 4 and it has been demonstrated by Sreenan and Beehan[12], that for a recovery rate of 75% at Day 3 only 64% can be recorded on Day 6, 50% on Day 7 and 46% on Day 8.[12]. Furthermore on Days 6 and 7, when most of the non-surgical recovery work was done, 8–14% of the ova were still retained within the oviduct and could only be flushed out using surgical techniques.[8] The transcervical approach has the definite advantage of being cheap, fast and can be repeated several times in the same animal.

The results recorded for our non-surgical transfer are equivocal since they are much lower than can be obtained with surgical transplantations reported in the literature but much better than the earlier results obtained with the transcervical approach. We believe that the major progress was made when the actual time of transfer had been postponed until Day 6 or even to Day 7. It was shown that in the earlier days following ovulation, the transplanted embryos were rapidly rejected from the uterine lumen into the vagina but that the endometrial activity was much lower from Days 6 onwards due to the influence of progesterone.[2] In the light of these observations, the negative influence on pregnancy of a relaxant drug such as isoxuprine lactate following non-surgical transfer was an unexpected finding and, since the trauma of a non-surgical transfer definitely has to be regarded as less traumatic upon the uterus than the surgical approach, the poorer results of the former method are difficult to evaluate. It may be that the lowest number of ova in the greater volume of flushing medium used during non-surgical recovery could have had a deleterious effect upon the viability of the embryos. When the results of the progress made hitherto with embryo transfer, are critically analised regardless of the method used, it must be admitted that the original high expectations were not achieved. An average of no more than 1,5 calves were born for each superovulated donor. The losses encountered are spread almost equally amongst superovulation, recovery and transfer procedures.

The introduction of deep-freeze techniques for the preservation of embryos will probably substantially alleviate the costs involved with the management of a recipient pool but could account for further loss of embryos. In our opinion the most critical factor in bovine embryo transfer is coupled to superovulation because of the variable and unpredictable ovulatory responses. Furthermore, the formation of antibodies against PMSG reduces the potential number of interventions per animal to two or three. We further believe that, in future, better results will be obtained with non-surgical recovery and transfer of embryos. An average recovery and transfer rate of 50% should be well within the capability of a well-trained technician. If this is achieved, the indications are that flushing single ova is probably preferable to inducing superovulation. After single ovulation, more ovulatory cycles can be induced in the donor by the injection of prostaglandin on the day of flushing. In this way one donor could be made to ovulate ±20 times a year. This would represent 10 embryos (which survive the standard deep freeze techniques) per year and entails a figure much more favourable than the results currently obtained with superovulation.

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BOOK REVIEW — BOEKRESENSIE

PRAOMYS (MASTOMYS) NATALENSIS: THE SIGNIFICANCE OF THEIR TUMOURS AND DISEASES FOR CANCER RESEARCH

EDITED BY JUN SOGA AND HURUO SATO

Daiichi Printing Co., Ltd. Niigata, Japan 950, 1977 pp XVII and 201, illustrated.

This is a first attempt at a concise publication in the form of a book on these singularly important rodents now in use in research for over 3 decades. Although *Mastomys* was a genus primarily sought after for studies in plague and bilharzia, the discovery in 1954 of spontaneous glandular stomach tumours among them by the late Dr Oettle of the SAIMR in Johannesburg was fortuitous. World attention was immediately focussed on the *Mastomys*, for before the era of the nitrosamines it was virtually impossible to find tumours of the glandular stomach in experimental animals. Recently there is again an upsurge of interest in the *Mastomys* since their implication in the deadly Lassa virus infection.

In their book Soga and Sato provide the historical background and draw attention to problems of animal husbandry in captive wild rodents. Before proceeding to cancer research applications a fairly detailed account of immunological characteristics is submitted. The chapter on tumours of the stomach deals extensively with the glandular stomach of *Mastomys*, its endocrine cell types and the fundic argyrophilic carcinoids frequently found in older *Mastomys* of both sexes. The authors have extensively quoted the pioneering works of Dr K Snell of the NIH, USA, particularly with reference to argyrophilia. Subsequent ultrastructural studies have supported the histological diagnosis of these transplantable metastasising tumours. Further chapters describe primary tumours of the liver, the thymus, the adrenals and several tissues except the mammae. Included in the book are also proceedings of the eight Japanese seminars on *Mastomys*. Easy to read author as well as subject indices bring the book to a close.

A minor fault is the generous sprinkling of awkwardly framed sentences, obviously a result of language problems. This should not distract a mature reader. Both the incorrect grammar and the overlapping of certain chapters written by different joint authors lead to considerable repetition. Chapter II has much in common with the others. The utility of the book lies in the encyclopaedic nature of its contents which should prove of enormous value to scientists involved in experiments with these rodents.

The main shortcoming of the book, as admitted in a personal communication by one of the editors, is that international expertise could not be involved to make the book up to date. Data from South Africa, where the strains originated, are a particular point at issue. In the last six years further tumour studies have been carried out in untreated and chemically treated *Mastomys* of the Y and the Z strains which are being inbred. Besides the well known tumours of *Mastomys*, spontaneous tumours of the forestomach and the mammary gland have also been found. The Japanese, who form the largest group of workers using *Mastomys* have, however, in the meanwhile decided to tell their side of the story. The result is a book, well produced and edited, with good quality illustrations. *Mastomys* most decidedly plays a role in medical research in plague, several parasitic diseases, bilharzia, osteo-arthritis, auto-immune diseases, Lassa virus infection and cancer.

J.D.R.

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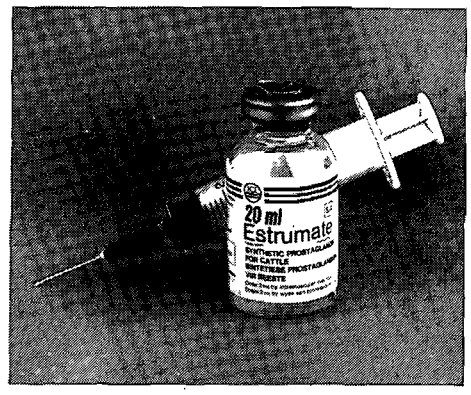

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IS BEEF PRODUCTION FROM SOUTH AFRICAN DAIRY HERDS POSSIBLE?

D R OSTERHOFF & S COUVARAS

ABSTRACT: Osterhoff D.R.; Couvaras S. Is beef production from South African dairy herds possible? *Journal South African Veterinary Association* (1978) **49** No. 1, 15–18, (En) Dept of Zootechnology, Fac. Vet. Sci. Univ. of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

The possibility of beef production from South African dairy herds was investigated. Beef production from dairy herds is closely linked to the productive life of dairy cows, i.e. the number of lactations completed and replacement by heifers entering the herd. The average number of lactations reached by South Arican dairy cows was analyzed and the following results were obtained for the year 1974/75: 29 638 Grade Friesians averaged 2,64 and 5 391 Grade Jersys averaged 2,95 lactations. If the total number of cows in a herd is to remain constant, at least 88% of all heifer calves must yearly be taken up in the herds as replacements. This includes a 25% selection on all replacement animals. Investigation shows that under current South African conditions beef production from dairy herds by cross-breeding is not possible. Various factors implying veterinary involvement, which are of cardinal importance in this type of investigation such as calving rate and calf mortality, are discussed.

INTRODUCTION

Numerous publications on this subject exist, but only relevant opinions will be cited here. Penzhorn & von La Chevallerie[9] pointed out that dairy cattle will have to play an increasingly important role in augmenting the national beef supply and that interest in the crossbreeding of dairy cows with beef bulls received special impetus with the advent of the Charolais breed in this country. Crossbred Charolais × Friesian steers were employed in determining the best rations and slaughter weights. Naude & Armstrong[7] pointed out that in Europe only a small proportion of beef is produced from beef breeds and that the production of beef from dairy cattle has received considerable attention from beef producers there. Their work on beef production from Jersey and Jersey crossbred steers and bulls proved that the productivity of the slow gaining, less efficient, purebred Jerseys could be considerably improved by crossing with either Simmentaler or Brown Swiss bulls. Mentz, Coetzer, Vermeulen & Coetzee[6] followed up the basic work of Naude & Armstrong[7] and included Afrikaners, Charolais and Hereford bulls in their crossbreeding experiments with the Jersey dam as basis. The crossbred animals were compared in respect of birth mass, occurrence of dystocia, weaning mass, post weaning growth on veld, growth in feedlots, level of feed conversion and final carcass yield. Breed influences could clearly be shown on the different performance measures.

Friesland steers were used by Reyneke[12] in an investigation on the supplementation of energy and protein to animals grazing summer veld on the Highveld. The same author[13] also showed that Friesland cows could be incorporated successfully in a weaner calf production system if managed similarly to a beef cow herd. The possibility of applying a multiple suckling system on veld was investigated and a considerable improvement in beef production per unit area was indicated with the implementation of a double-suckling system.

Cunningham and McClintock[3] studied the effects of varying cow replacement and beef crossing rates on the breeding policy appropriate for a dual-purpose cattle population where "dual-purpose" was defined as any population in which the cows are milked and the male and surplus female progeny are reared for beef. They pointed out that a high level of beef crossing is economically feasible but that more emphasis should be given to dairy traits in selecting dual-purpose bulls.

Cunningham[2] listed the advantages of maximising the extent of beef crossing in dairy herds relative to dual-purpose selection practised in most European countries as follows: "(a) genetic gain in beef and dairy cattle can be faster by selection within separate strains, (b) breeding a portion of the dairy herd to beef bulls reinforces the economic impact of the remaining dairy-to-dairy inseminations; (c) scope of dairy progeny testing can be reduced; (d) requires dairymen to exercise more selectivity in choice of dams of replacement heifers; (e) provides beef and dairy heifers that can be used as replacements in beef herds (or increase size of beef population), (f) calving problems of dairy heifers can be minimized; (g) allows exploitation of any heterosis for beef traits, which may be around 6% in beef x dairy crosses". The disadvantages of beef crossing were given as "(a) it reduces culling pressure on dairy females within the herd and (b) it may result in fluctuations in herd size, particularly in small herds".

The object of this investigation was to determine to what extent and under what conditions the South African dairy farmer could consider incorporating a similar system of beef crossing in his dairy herd in order to benefit from all the advantages cited by Cunningham[2].

PROCEDURE

Beef production from dairy herds is closely linked to the productive life of dairy cows, i.e. the number of lactations completed and the replacement of these cows by heifers which have to enter the herd. This relationship is explained in Table 1.

It is obvious that in a herd of 20 cows averaging three lactations, 33% (or seven heifers) must be ready every year to enter the herd as replacements for cows leaving the herd. If an additional 25% selection is applied, nine heifers must be ready to enter the herd. If, on the other hand, five lactations were completed as an average in the same herd, four heifers would be required as replacements without selection and six heifers with selection.

In order to obtain some indication of the average number of lactations attained by dairy herds under South African conditions, one of the main objects of this investigation was therefore to investigate the number of lactations completed by the National Dairy Herd. Data for 1974/75 from the National Milk Recording Scheme on both registered and grade Friesland

Table 1: THE INFLUENCE OF THE PRODUCTIVE LIFE OF DAIRY COWS ON REPLACEMENT

Productive life i.e. number of lactations completed	Replacement-percentage, no selection is applied to heifers calving for the first time	Replacement-percentage, if 25% selection is applied. (3 out of 4 heifers selected)
3	33	44
4	25	34
5	20	27
6	17	23
7	14	19

Table 2: THE PERCENTAGE DISTRIBUTION OF GRADE FRIESLAND COWS IN THE DIFFERENT REGIONS ACCORDING TO LACTATION NUMBER (1974/75).

REGION	I West. Prov.	II East. Prov.	III O. Free State	IV Transvaal	V Natal	Total R.S.A.
No. of cows	5 242	4 205	4 766	6 358	9 068	29 638
Number of lactations						
1	35,7	31,2	36,3	36,1	31,5	34,0
2	24,5	22,0	27,4	25,0	23,2	24,3
3	18,4	14,3	16,4	17,5	17,1	16,9
4	9,8	11,1	8,6	10,0	11,0	10,2
5	5,0	8,2	5,6	5,5	7,0	6,3
6	4,3	5,2	2,8	2,8	4,6	4,0
7	1,4	3,6	1,4	1,6	2,4	2,0
8	0,4	2,2	0,9	0,8	1,7	1,2
9	0,3	1,2	0,4	0,4	0,8	0,6
⩾10	0,2	1,0	0,2	0,8	0,7	0,5
Av. number of lact. completed	2,47	3,00	2,41	2,46	2,81	2,64

and Jersey cattle were kindly made available by the Officer in Charge, Mr P.J. Basson[1] and analyzed by the Department of Zootechnology at Onderstepoort. Since registered animals are not customarily used for crossing with beef bulls, the investigation was limited to the grade Friesland and the grade Jersey cattle poulation taking part in milk recording. In order to secure an unbiased survey of the productive life of South African herds, the analysis included not only the cows that appear in the official Annual Milk Recording Report published by the Department of Agricultural Technical Services but also those which for various reasons are excluded from the report. The latter include cows having had abnormal lactations (either too short or too long) and those whose lactation record had to be terminated either as a result of death, change of ownership, etc. From the figures obtained projections were made on the availability of dairy cows for beef production considering aspects such as calving percentages, calf mortality and the special situation of producing beef only from first calvers so as to minimize calving problems of dairy heifers.

Direct economic consequences were not considered since this would unneccesarily complicate the issue. The authors are aware of the importance of the age at first calving and also of the direct loss in the milk produced if the production of say the first or second lactation is compared to the fourth or fifth lactation. It has been shown by many workers[4] that the production of milk increases from the first to the fifth lactation by approximately 30%.

Furthermore, no efforts were made to investigate the reasons for cows leaving the dairy herds. Great differences exist between countries in this respect. In South Africa the main reason for cows leaving the herd would certainly be problems of sterility, subfertility and mastitis while in well managed herds in European countries low production would be regarded as the most important reason.

RESULTS AND DISCUSSION

Productive life of dairy cows

The productive life of the dairy population in South Africa is expressed in the number of lactations completed by our cows. The records from the Milk Recording Scheme are divided into five regions Only the two largest groups of grade cows, namely Friesland and Jersey were included since Guernseys and Ayrshires would only play a minor role in the production of beef from dairy herds. Table 2 presents the results of the survey of all Friesland cows which completed the different lactations in the five milk recording regions; the total number of cows in each region is also included.

From this table one can conclude that the Grade Frieslands completed on average slightly more than 2½ lactations in South Africa. Eastern Province presented better results, followed by Natal but as a whole, about 60 % of all cows do not complete more than two lactations. Although registered herds should not be used for crossing with beef bulls, the average productive life of the 7 309 registerd Frieslands that participated in the milk Recording Scheme during 1974/75 was calculated at 3,01. Table 3 presents the results of the survey of all Grade Jersey cows which completed the different lactations.

Table 3: THE PERCENTAGE DISTRIBUTION OF GRADE JERSEY COWS IN THE DIFFERENT REGIONS ACCORDING TO LACTATION NUMBER (1974/75).

REGION	I West. Prov.	II East. Prov.	III O. Free State	IV Transvaal	V Natal	Total R.S.A.
No. of cows	1 175	1 560	573	512	1 566	5 391
Number of lactations						
1	26,7	34,5	44,2	32,9	32,0	32,9
2	21,9	16,7	19,9	23,2	24,1	20,9
3	19,1	16,3	10,3	14,3	14,0	15,4
4	13,7	9,0	9,0	11,2	10,7	10,8
5	6,6	6,2	5,2	7,5	6,6	6,4
6	5,4	6,0	6,3	3,7	5,0	5,4
7	3,6	3,7	3,3	3,5	3,4	3,5
8	1,0	3,3	1,0	1,5	1,9	2,0
9	0,9	2,2	0,4	1,2	1,1	1,3
⩾10	1,1	2,1	0,2	1,0	1,2	1,4
Av. number of lact. completed	3,01	3,15	2,52	2,85	2,90	2,95

Table 4: THE INFLUENCE OF CALVING AND CALF MORTALITIES ON THE PERCENTAGE OF COWS REQUIRED FOR THE PRODUCTION OF REPLACEMENT CALVES.

Productive life i.e. lactations completed	Replacement % allowing for 25% selection	Percentage of cows required for the production of dairy replacement calves — Calving rate 100 — Calf mortality 0	5	10	Calving rate 90 — Calf mortality 0	5	10	Calving rate 80 — Calf mortality 0	5	10	Calving rate 70 — Calf mortality 0	5	10
3	44	88	93	98	98	—	—	—	—	—	—	—	—
4	34	68	72	76	76	80	84	85	89	94	97	—	—
5	27	54	57	60	60	63	67	68	72	76	77	81	86
6	23	46	49	55	55	58	61	58	61	64	66	69	73
7	19	38	40	45	45	47	50	48	51	53	54	57	60

Table 5: THE PERCENTAGE OF DAIRY COWS AVAILABLE FOR BEEF PRODUCTION IN HERDS USING ALL AVAILABLE HEIFERS FOR BEEF PRODUCTION AND THEN ALLOWING FOR A 25% SELECTION OF THESE HEIFERS AS REPLACEMENT ANIMALS.

Productive life i.e. lactations completed	Percentage of cows available for beef production — Calving rate 100 — Calf mortality 0	5	10	Calving rate 90 — Calf mortality 0	5	10	Calving rate 80 — Calf mortality 0	5	10	Calving rate 70 — Calf mortality 0	5	10
3	—	—	—	—	—	—	—	—	—	—	—	—
4	3,5	1,7	0,5	0,5	—	—	—	—	—	—	—	—
5	10	8,5	7	7	5,5	4	4	2,5	1,6	1,0	—	—
6	14	12,5	11	11	09,6	8,2	7,9	6,6	5,4	4,7	3,6	2,6
7	18	16,6	15	15	13,5	12	11,8	10,5	9,2	8,5	7,4	6,3

The conclusion here is that the Grade Jersey in South Africa completed, on average, almost three lactations. The average productive life of the 5 462 registered Jerseys that participated in the Milk Recording Scheme during 1975/74 was calculated at 3,54. Compared to the average figures of other countries of agricultural importance, the South African figures are low:

Of 2¾ million dairy cattle in the U.S.A. the average lactation figure is 3,68; in the United Kingdom the figure is 3,9 [10]; in Germany it is 4,2[14] and in Sweden 4,0 and 5,2 lactations for high and low producing herds respectively.[17]

Availablility of dairy cows for beef production

The low average productive life of the South African dairy herds necessitates a higher replacement rate which is further influenced by the calving rate of all cows in the herd and also the calf mortality. Exact figures for calving rate and calf mortality are not available but the problem of low fertility, sterility and calf losses have repeatedly been discussed[5 11 15 16]. For the purpose of this investigation we have assumed that the calving rate in dairy herds varies between 70 and 100% and that the calf mortality varies between 0 and 10%. In Table 4 the percentage of cows which must be set

aside for the production of replacement calves are presented for various combinations of calving rate, calf mortality, lactation number and selection pressure of 25% on replacements.

From Table 4 it is evident that in South African Friesland and Jersey grade herds with an average of 2,64 and 2,95 completed lactations, no cows are available for crossing with beef bulls if a 25% selection of heifers entering the herd is applied. Only in the best herds with a high calving rate and low calf mortality would animals be available for crossing with beef bulls. For example: in herds completing on average four lactations, having a calving rate of 90% and a calf mortality of 5%, 80% of the cows are needed for the production of replacement heifers, leaving only 20% for crossing with beef bulls.

"Specialists" very often recommend mating all heifers (first calvers) to beef bulls in order to minimize calving problems. The results of the calculations on the availability of dairy cows for beef production when all first calvers are mated to beef bulls are presented in Table 5.

From Table 5 it is obvious that beef production according to this scheme of mating all heifers with beef bulls is only possible in the very best herds with an average productive life of five or more lactations.

Such a system for South African conditions is therefor totally unjustified and has no scientific backing. Such a farmer would get into serious trouble by not having sufficient dairy calves available for replacement, especially if one keeps in mind that for genetic improvement a 25% selection pressure is generally recommended.

In Fig. 1 the availability of dairy cows for beef production is depicted. For ease of representation only herds with an average productive life of three and five lactations are given. Calving percentages of 60% and higher and calf mortalities from zero to 10% are considered. All first calving heifers are used for dairy calf production.

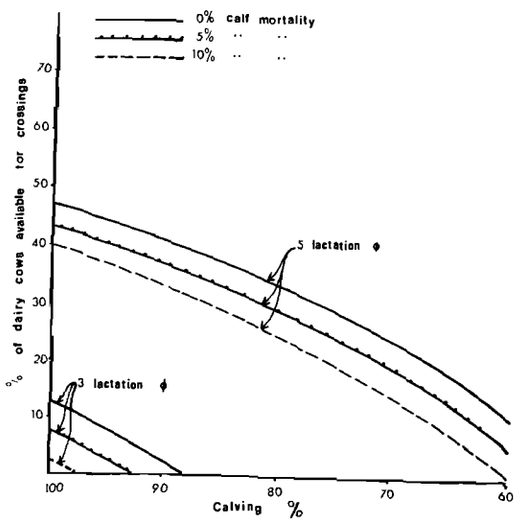

Fig. 1: Availability of dairy cows for beef production

From the figure it can clearly be seen that for the average farmer whose dairy herd has a productive life of three lactations, no cows can be set aside for crossing with beef bulls. This applies to almost all South African dairy farmers (see Table 2 and 3). It must be borne in mind that half the number of calves are male and that no practical techniques are as yet available to separate the X- from the Y-bearing sperm for possible prediction of the sex of the calf. Thus provision has to be made for twice the number of calves, half of them being the females considered as replacement heifers. Bull calves can obviously be fed in feedlots and used for beef[18] but the fact remains that under current South African conditions almost all cows must be used for the production of dairy replacements and therefore cannot be mated with beef type bulls. Only in the very best herds can beef at present be produced from dairy cows by cross breeding. The veterinary implications of this investigation are clear. If through veterinary involvement, the number of cows available for beef production could be increased and beef production by the average dairy farmer from his dairy animals would then be possible.

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KIKUYU GRASS POISONING OF CATTLE IN NATAL

R.W. BRYSON and S.J. NEWSHOLME

ABSTRACT: Bryson R.W.; Newsholme S.J. Kikuyu grass poisoning of cattle in Natal. *Journal South African Veterinary Association* (1978) **49** No. 1, 19–21 (En) Reg. Vet. Lab. Private Bag X9005 Pietermaritzburg. The clinical findings in six natural and two experimental cases of Kikuyu grass poisoning in Natal, South Africa, are described and compared with findings in cases of toxicity reported elsewhere. The toxic factor has not been identified but a mycotoxin is suspected.

INTRODUCTION

Kikuyu poisoning was first recorded in New Zealand in 1962[1 2] and has continued to cause sporadic losses up till 1973. Mortality rates of up to 65% of animals at risk have been recorded. In Western Australia, two outbreaks occurred in 1974[3]. In both mortality occurred on lush Kikuyu grazing undamaged by any pest and as far as army worms are concerned, neither *Spodoptera exempta* nor *Pseudaletia separata* have been recorded in this area.

They make two postulations on the condition:

(a) that it is a physiological disorder related to ruminal engorgement[7].

(b) that the disease is caused by toxin-producing strains of *Myrothecium* sp.[4]

The species of army worm found in South Africa is *Spodoptera exempta* whereas that found in New Zealand is *Pseudaletia separata*.

The condition has been observed in various parts of Southern Africa by Kellerman in Bulawayo[5] and Kitching in Natal 1973[6]. The toxic factor has not yet been identified in any of the outbreaks in South Africa, New Zealand or Australia. A mycotoxin is suspected and experimental work by Di Menna, Mortimer, Smith and Tulloch[4] showed that large doses of a mammalian toxin from three species of *Myrothecium* produced pathological changes similar to Kikuyu poisoning. Kitching[6] isolated two unidentified fungi from army worm faeces but no further progress was made.

GEOGRAPHICAL, SEASONAL AND PASTURE FACTORS

The paddock affected was 0,6 ha in extent and vegetation consisted only of Kikuyu grass, *Pennisetum clandestinum*. It faced North-East on a latitude of 02905 S and sloped gently, being protected on one side by a line of mature shade trees.

The paddock had been invaded by army worm (*Spodoptera exempta*) on 28.2.77. The grazing was decimated and the insect was eventually brought under control by the application of insecticide on 3.3.77. As little grazing remained, the paddock was rested and grass was growing plentifully when cattle were re-introduced on 24.3.77. The temperature and rainfall for this period is shown in Table 1.

During this rest period factors conductive to toxicity were as described in New Zealand i.e. a period of drought followed by patchy rainfall and a humid atmosphere[1 2].

Neighbouring paddocks were less heavily infested and for this reason cattle were allowed to graze these 2 d after insecticidal treatment. No sickness was observed.

Table 1: METEOROLOGICAL DATA RELATING TO OCCURRENCE OF KIKUYU POISONING

Day	Rainfall mm	Min. Temp. °C
–10 (18.2.77)	0	8,6
–9	0,4	10,0
–9	1,2	12,5
–7	0	11,2
–6	1,4	10,7
–5	1,5	14,4
–4	18,0	13,7
–3	3,0	12,2
–2	0	12,2
–1	0	13,5
0 (28.2.77)	0	10,7

CLINICAL SIGNS OF DISEASE

The first batch of cattle to enter the paddock, consisted of twenty four mature Friesland cows, all dry and the majority pregnant. Grazing commenced on 24.3.77 and the first illness was noticed on 27.3.77 (a Sunday). The cattle appeared reluctant to walk the short distance out of the paddock for the daily concentrate ration. No treatment was given and observation was inhibited by the day being Sunday with only a skeleton staff operating.

On the morning of Monday 28.3.77 three cows were found dead, two were dying and several others sick.

Ruminal overload with acidosis was suspected. Treatment with oral doses of sodium bicarbonate was given and all the cattle removed from the paddock and deprived of food and water. During the afternoon of the same day, the two badly affected cows died. One regurgitated liquid ruminal contents and expired shortly after drenching. A further cow died during the night. Kikuyu poisoning was not suspected at that stage and no post mortem examinations were carried out.

Two of the surviving cows received symptomatic treatment, one for mild colic and one for tympany. Poloxalene was used for tympany and chlorodyne for colic. Both recovered rapidly. Food and water was offered the next day in limited quantities with a generous supplement of molasses. Grazing was resumed twenty four hours later, no further sickness being observed. Two of this group aborted some weeks later but examination of the foetus did not reveal the cause.

A second batch of "tracer" animals entered the paddock on 1.4.77. Five young Sussex steers with an average mass of 175 kg were used and were accompanied by five adult Merino sheep. All took readily to the still plentiful grazing. On 5.4.77, two of the steers, Nos 100 and 123 were obviously sick and the following clinical symptoms were noted:

Standing close to the water trough, the lips and

tongue were dipped into the water and the head moved from side to side with no apparent swallowing, aptly described by New Zealand workers as "sham drinking"[1,2]. Drooling of large quantities of watery saliva occurred at the same time. Detailed examination showed normal temperatures, dehydration and excessive fluid in the rumen with mild tympany. No lesions in the mouth or pharynx were seen and the tongue was fully mobile and normal in every way. The same clinical signs as observed in the previous group were seen. Blood analysis at this stage disclosed the findings summarized in Tables 2 and 3.

Table 2: BLOOD CHEMISTRY

No.	Mg. mg/100mℓ	B.U.N. mg/100 mℓ	Cholesterol mg/100 mℓ	P.C.V. %
100	18,7	25	260	49
123	204	45	144	53
Normal	26	10–20	100–200	30–35

Table 3: HAEMATOLOGY
Differential Leucocyte Count:

Steer No.	100	123	Normal
Neutrophils	36%	39%	50–70%
Lymphocytes	47%	32%	20–30%
Monocytes	17%	27%	3–6%
Eosinophils	—	1%	1–3%
Basophils	—	1%	0,2%

The group was allowed to remain on the toxic paddock. During the subsequent 12 h, the two sick steers became progressively worse. Colic was severe, looking at and kicking the left flank. Hyperaesthesia was obvious in both; one showed inco-ordination and a "high-stepping" gait reminiscent of heartwater.

At this stage all steers were removed to a small pen without food and water, the sick steers recovering spontaneously. A third batch of five steers was introduced 4 d later. After 4 d grazing one steer again became affected as before. It recovered after removal with the others from the paddock.

A fourth batch of much heavier steers was introduced the next day and showed no evidence of disease intoxication up to the time of removal 3 weeks later when the paddock was grazed out.

The sheep remained unaffected throughout the trials.

A detailed investigation of pasture, army worm and faeces thereof was conducted during the outbreak and the following fungi were isolated:

1) *Mucor* sp
2) *Fusarium* sp
3) *Cladosporium* sp
4) *Geotrichum* sp
5) *Myrothecium* sp

No *Aspergillus* sp were isolated.

Transmission trials to identify the causative factor were not possible during the present outbreak and are also inhibited by the fact that army worm does not breed naturally in South Africa, the adult moth invading from territories to the North on a sporadic basis and laying eggs which hatch into the larval "army worm" stage. Investigation is being made into the artificial hatching of pupae during the coming summer.

DISCUSSION

The only common implicating factor is Kikuyu grass. Whilst this outbreak was being studied reports of poisoning were received from widely distributed areas in South Africa. In one outbreak fifty cattle died. In all cases Kikuyu grass was being grazed after army worm damage. Grazing of adjoining pastures of rye, star and veld grasses damaged by the worms produced no deleterious effects on the cattle.

Predisposing factors in the present outbreak closely followed the pattern in New Zealand. These are recorded in Table 1 and are of critical importance. This combination closely follows that extant in the paddock in New Zealand where poisoning occurred, described in New Zealand as "toxic"[1,2].

Both in South Africa and New Zealand a "prepatent period" when the infested pasture was not grazed, appeared necessary for the toxicity to develop. This varied in New Zealand but was usually up to 10 d[1,2]. This period could not be accurately assessed in the present outbreak as the pasture was rested for 3 w and at any stage, toxicity could have developed. It was known to be toxic at 28 d, persist for 40 d.

In all outbreaks a period of 24 to 96 h elapsed between commencing of grazing and the advent of signs of disease.

New Zealand observations[1] showed that the mucosae of the fore-stomachs were damaged in half the cases with patchy or diffuse reddening of the epithelium which could easily be stripped off. The fore-stomachs, the abomasum and the small intestine, were overfull with coarse ingesta.

Linear erosions up to 30 mm long and 3 mm across were noted along the ridges at the free margins of the abomasal folds. In a few cases, circular mucosal ulcerations up to 5 mm in diameter were seen. Other findings were subepicardial haemorrhages in the vicinity of the coronary and longitudinal grooves of the heart.

CONCLUSIONS

Although the causal factor has not been determined, certain precautions can be recommended:

1) If the paddock has to be rested due to army worm damage, allow only a few trial animals to graze first for up to 96 h to check for toxicity. Alternately leave vacant for at least 40 d or more. (Three weeks rest period is insufficient.)
2) Present experience indicates that grazing paddocks immediately after they were damaged by army worms, regardless of insecticidal treatment, did not result in toxicity presumably because the causal factor had been disturbed. If the grazing is urgently required, trial animals should be introduced within a maximum of 2 to 3 days.

ACKNOWLEDGEMENTS

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INFORMATION — INLIGTING

LIVESTOCK GROWTH STIMULANT ISOLATED

A plant extract, acid-resistant hemicellulose, or ARH, has been found by West Virginia University scientists to be an excellent growth stimulant, which could revolutionise animal nutrition and feeding. Its use could save large quantities of grain and a great deal of money currently expended on feed.

Maize cobs were the source of this extract in the research, but other sources are being sought. Using the laboratory procedures which they have developed, the West Virginia University team believes the growth stimulant can be manufactured from most fibrous and woody plants or their by-products, including some sawdusts, maize stover and many low-quality roughages and grasses.

The research followed an animal feeding experiment with lambs fed urea as a replacement for some protein in the ration.

In this experiment, one group of lambs was fed a ration containing wheat straw as the sole source of roughage and another group a ration with ground maize cobs as the roughage. Both diets contained urea as the dietary source of nitrogen.

After making adjustments for the nutrient differences between the two diets, it appeared that the lambs on the maize cob ration were obtaining significantly more protein, and utilising more of the available nitrogen from the urea than were the group on the wheat straw ration.

Assuming that the maize component was responsible for this difference, the scientists sought the reason, believing, at first, that it was having an effect on the rumen microorganisms.

Laboratory experiments were therefore conducted, employing a fractionation process for breaking down the maize cobs. Each of the resultant extracts was tested on rumen microorganisms taken from fistulated animals and placed in an artificial rumen, in the laboratory.

Only the acid-resistant hemicellulose was found to stimulate growth and utilisation of urea by the rumen microorganisms.

Other tests were conducted with lambs and rats.

In the tests with rats, laboratory-produced ARH added to the rats' diet resulted in a 15 per cent weight gain over that of the controls.

Adding ARH to the ration of lambs resulted in increased urea utilisation.

Rumen microorganisms normally cannot be cultured without the presence of rumen liquor or certain substances which it contains. In 1976, however, the West Virginia University researchers demonstrated that these microorganisms could grow without rumen liquor, if ARH was substituted.

Additional tests with fistulated animals were made, in which the animals were fed ground maize cobs and urea, and food samples subsequently taken from their rumens. These samples contained lignin-hemicellulose, which was found to be very similar, chemically, to the acid-resistant hemicellulose produced in the laboratory by the researchers.

This fact and the observation that this substance stimulated the growth of rumen microorganisms in the simulated rumen, supported the researchers' belief that the growth stimulant could be produced from hemicelluloses by microorganisms in the rumen. Hemicelluloses, previously considered to be non-essential in the nutrition of animals, may henceforth be considered as essential, in that they yield ARH through the action of rumen microorganisms.

These findings may bring about extensive changes in animal-feeding practices, the ultimate outcome of which would be the feeding of low-quality fibrous roughages, together with urea as a source of nitrogen.

In non-ruminant animals such as rats, chickens, hogs and others, the researchers believe that bacteria in the intestines might digest hemicellulose and yield products that are growth stimulators for other microorganisms in the body. The net result of this would be increased growth, as was found to be the case in the experimental rats fed ARH.

The researchers are continuing their study and are attempting to determine how much of the stimulant can be used in the diet, and the effect of various amounts of ARH on certain animals.

They estimate that by adding the stimulant to chicken feed, for example, poultrymen could cut their feed use by at least eight per cent. This would represent a substantial saving, when one considers that more than three billion broilers are produced in the United States each year.

The finding has far reaching possibilities, particularly for poor and/or developing countries, where meat could be produced inexpensively by raising large herds on urea and readily available low-quality grasses.

(American Society of Animal Science, 69th. Annual Meeting, University of Wisconsin, Madison, Wisconsin, July 24–30, 1977).

GEELDIKKOP: PRESERVATION OF TOXIC MATERIAL

G.F. BATH*, E.M. VAN TONDER* AND P.A. BASSON**

ABSTRACT: Bath G.F.; Van Tonder E.M.; Basson P.A.; Geeldikkop: Preservation of toxic material. *Journal South African Veterinary Association* (1978) **49** No. 1, 23–25 (En) Reg. Vet. Lab. P. Bag X528,5900 Middelburg, Rep. of South Africa.

Wilted *Tribulus terrestris* plants were harvested from a camp in which geeldikkop had just previously broken out. The plants were treated in various ways in an attempt to preserve their toxicity.

The only successful method of preservation found was rapid freezing of harvested material. Three kg of plants were kept frozen for six weeks and then dosed via rumen fistula to a sheep which subsequently developed characteristic symptoms and lesions of geeldikkop.

INTRODUCTION

Research into geeldikkop has been hampered because it is difficult to reproduce the disease under controlled experimental conditions[2-5 8-13]. To date this has been done in two ways: either sheep have been allowed to graze under controlled conditions on pastures where the disease had previously occurred; or sheep have been fed the plant *Tribulus terrestris* L. (Zygophyllaceae) which was freshly harvested from such pastures[9 10 13 14]. These findings indicate that a labile toxin is probably involved since material kept for more than a few days at ambient temperature is not capable of producing the disease.

Further research on geeldikkop depends in part on finding a way of preserving the toxicity of material for later investigation and this problem has thus been given high priority in attempts to identify the cause of the disease. The following report records this aspect of an investigation of geeldikkop.

MATERIALS AND METHODS

Features of the natural outbreak of the disease.

Geeldikkop occurred on the farm de Rust in the Aberdeen district of the Cape Province during December 1972. On 3/11/72 ca. 60 mm rain fell and subsequently the weather was fine and hot. On 5/12/72 in one camp (ca. 150 ha) containing 166 two-tooth Merino ewes, 68 sheep were showing clinical signs of geeldikkop and seven were dead. Examination of dead sheep revealed characteristic lesions of geeldikkop both on gross and microscopic pathology[14].

The veld type in the affected camp was False Karroid Broken Veld[1]. The veld was eroded, denuded in patches and invaded by poor quality pioneer bushes, presumably due to previous poor grazing practices.

In two main areas within the camp there were very large numbers of *T. terrestris,* which grew mainly on bare sandy patches where the soil had been disturbed. The plants were extremely wilted, greyish-green to brownish-green and very hairy. Few flowers or fruits were present. Stems were about 10 to 50 mm long, and were raised from the ground, standing nearly upright. Leaflets were folded along the rachis. This was in sharp contrast to the usual procumbent habit of the plant[6].

Collection of toxic material

Wilted *T. terrestris* plants as described above were harvested from areas where it grew abundantly and where there were signs of previous grazing. All material was harvested between the second and ninth days after the outbreak when weather conditions were much the same as before the outbreak.

Due to the extremely small size of individual plants, picking by hand was impractical and a method of harvesting was evolved to suit the conditions. If necessary, loose debris and sand were removed with a yard broom. As *Tribulus* plants have long taproots they were not affected by this sweeping. A sharp, flat spade was then scraped forward along the ground at an acute angle, so cutting off the plants at ground level. The plants were then swept together and thrown onto a very coarse sieve, made of bird netting, which allowed sand and fine matter to fall through. The remaining plants were then handsorted to ensure that only *T. terestris* remained.

Preservation of Toxic Material

Collected material was treated in various ways in an attempt to preserve its presumed toxicity. Since all previous attempts at preserving toxic plants by drying had failed[4 10 13], this method was not used. Those methods which were used were aimed at stabilising the apparently labile toxin present.

Method 1:

A sample of 600g of freshly harvested material was immediately placed in ether which was allowed to evaporate. The material was then kept in a dry state at room temperature until dosed 68 days later at the Regional Veterinary Laboratory, Middelburg, Cape Province.

Method 2:

As the plants were harvested, they were weighed in batches at short intervals and then immediately placed in a 20% aqueous solution of household vinegar kept at ambient temperature. A total of 3 kg was harvested and stored in this way over 4 days. On 13/ 12/72 as much liquid as possible was expressed from the plants, and after transfer to the Regional Laboratory at Middelburg, both liquid and plant material were stored at 4°C until dosed. The vinegar extract was kept for 43 d and the plant residue for 53 d before dosing.

Method 3:

Plants were harvested and placed in small plastic packets. These were weighed and then immediately placed in a polystyrene insulated box containing ice cubes. The interval between cutting the plant and putting it in the coolbox was never more than 10 minutes. Every half hour the contents of the box were transferred to a freezer and fresh ice was added to the coolbox if neces-

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sary. The plants were kept frozen at −15 °C until dosed 41 d later at Middelburg. A total of 3 100g was preserved in this way.

Experimental Animals

All sheep used in the experiments originated from the Grootfontein Agricultural College, on which property no outbreaks of geeldikkop occurred either before, during or after the experiments in that summer season. They had all been fistulated for direct intraruminal dosing and had in the interim been kept in open pens at the Regional Veterinary Laboratory. The animal receiving the ether treated material (Method 1) and the two sheep which received vinegar extract or plant residue (Method 2) were all 10 month old Merino sheep, while the sheep receiving frozen material (Method 3) was an adult Merino ewe.

Control sheep were also from the Grootfontein Agricultural College and were in all respects treated in the same way as the four experimental animals, except that they were not fistulated and were not dosed any material.

All sheep were placed in open pens exposed to prevailing sunlight for most of the day and were allowed water and milled lucerne *ad libitum*.

Dosing Experiments

Tribulus plants which had been treated with ether were pulverised in an iron pestle and mortar and then dosed as a single dose via a ruminal fistula.

Plants placed in vinegar were dosed in three equal doses over three consecutive days. Material was first pulped using an iron pestle and mortar and a little rumen content was removed each day to facilitate dosing. The vinegar extract was dosed by stomach tube in three equal doses over three consecutive days.

Plants preserved by freezing were dosed in four equal doses over four consecutive days. Immediately before dosing the frozen material was pulped using an iron pestle and mortar and a little rumen content was removed to facilitate dosing.

Examination and collection of specimens

All sheep were examined clinically daily and were bled at intervals for blood analysis. Standard methods were used to determine the haematocrit, bilirubin, blood urea nitrogen, glutamic oxalacetic transaminase, plasma proteins and plasma copper. After slaughter at the end of the experiments all the sheep used were examined for gross pathology. Organs were fixed in 10% buffered formalin and later sectioned and stained by standard methods for microscopic examination.

Field Experiment

To ensure that the agent causing geeldikkop in the camp was still present during the investigation, six 9 month old Merino sheep from the Grootfontein Agricultural college were released in the camp and allowed to graze freely. Two were released on 7/12/72 and four on 14/12/72. Between these dates they were examined clinically daily and subsequently the owner of the farm was asked to observe them as often as possible for signs of geeldikkop. On 18/12/72 those animals which had become affected were removed and the remainder were removed on 4/1/73 to Middelburg. All sheep were slaughtered at the end of the experiment and where abnormalities were found, specimens were taken for histological examination.

RESULTS

Clinical and chemical pathological findings

No abnormalities were seen in the control animals. None of the sheep which received doses of vinegar extract, the plant residue or the plants treated with ether showed any clinical symptoms of geeldikkop or any deviations in blood chemistry. No significant lesions were found on post mortem examination.

The sheep which received frozen plants first showed clinical lesions seven days after the first dose. Moderate oedema of the ears and muzzle was accompanied by slight hyperaemia of the affected parts and the animal sought shade. The body temperature was elevated and a mild icterus was visible in the mucous membranes. No lesions were seen on the coronets. The haematocrit, urea nitrogen, transaminase and bilirubin levels were above normal.

The next day hyperaemia and oedema of the facial area as well as the icterus had become more intense. Subsequently the oedema and hyperaemia, followed by the icterus, gradually subsided. On Day 13 the oedema had virtually disappeared and bilirubin levels had also fallen markedly. The haematocrit decreased gradually from the first day of symptoms but both transaminase and urea nitrogen levels increased for two days before also subsiding. Hyperaemia of the coronets was first seen on the tenth day. After this the superficial layers of the skin of the ears and muzzle became hard and began to slough.

Pathology

The sheep was slaughtered for post mortem examination on Day 13. Pathological changes included the following: slight subcutaneous oedema of the muzzle and ears; necrosis and sloughing of the superficial epidermis around the eyes, the dorsal muzzle and the ears; moderate coronitis confined to the abaxial aspect of the hooves only. Marked icterus was present; the liver was swollen, friable, yellowish-brown and the lobuli were prominent. The whole liver was more or less evenly involved. Moderate nephrosis, mild tumor splenis, mild dehydration, gastro-intestinal stasis and adrenocortical hyperplasia were also present.

Histopathological examination of the organs showed typical and diagnostic lesions of geeldikkop in the liver[14], which was studded with many enlarged and proliferated groups of Kupffer cells containing cholesterol – like clefts. The groups appeared more frequent in the periportal areas. Cholesterol-like clefts were also present in bile ducts. Bile ducts were mildly proliferated and more prominent in some sections. There were a few Kupffer cells containing a brownish pigment (probably lipofuchsin), very mild megalocytosis and mild hepatic degeneration. A few portal foci of mild mononuclear cell infiltration were present.

The kidney showed mild nephrosis with prominent cystic changes in the tubuli. There were also foci of mild interstitial round cell reaction and there was evidence of tubular regeneration. The facial skin revealed mild hyperkeratotic dermatitis.

Field cases

Four of the six sheep which had been released into the camp for free grazing also developed typical signs of

geeldikkop. On 18/12/72 the two sheep which had first been released and one other sheep were showing symptoms. By 27/12/72 a further sheep had become affected and one of the original sheep had died. Gross and histological examination of affected sheep after slaughter showed typical and diagnostic lesions of geeldikkop[14].

DISCUSSION

In all essential features the disease which occurred in the natural outbreak was identical to that which occurred in four of the six experimental sheep allowed to graze freely in the affected camp as well as the sheep dosed with *T. terrestris* plants harvested from the camp and kept frozen until dosed. In all three cases diagnostic microscopic lesions of geeldikkop were found.

Since the sheep which was dosed frozen plants was kept in a bare pen amongst control sheep which showed no sign of the disease at any time, it is evident that the classical geeldikkop produced was due to the dosing of plant material. It is important to note that the experimental animals were kept in a pen more than 100 km from the field outbreak, and had been fed only milled lucerne for some months previous to the experiment.

If any infectious agent was involved it must have been in or on the plant or occurred throughout the region and required ingestion of the plant to trigger clinical symptoms. This hypothesis however seems unlikely and is discounted by several previous negative findings [5 8 13 14]. A specific hepatotoxic substance is the most likely aetiological agent.

The plants used were extremely wilted. This lends support to previous evidence that the toxic factor is associated with wilted plants[6 9 10 13 14]. In addition, free grazing sheep were observed to show a marked preference for grazing wilted *T. terrestris*. This has been noted by several previous workers[3 9 13 14].

A practical method of harvesting large quantities of small *T. terrestris* plants was developed. Of those methods of preservation attempted, only freezing was found successful in preserving the toxicity of the material. Far less plant material was treated with ether, and a valid comparison with the other methods is impossible. Almost the same quantity of *T. terrestris* was frozen as was treated with vinegar, and although it may be argued that the toxic element may have been partly extracted into the vinegar solution, a comparison of the methods is justifiable. Roughly the same amount of plant material was harvested in the same way from similar areas and over the same period of time when weather conditions did not change. In addition, the vinegar extract and expressed plants were chilled for most of the period of storage. If the toxic principle had been partly extracted intact into the vinegar solution, some sign of clinical or subclinical disease should have been seen in either the sheep dosed the vinegar extract or the plant residue. Since this did not occur it appears that the toxic element was largely lost in the vinegar-treated material and in the vinegar extract.

This finding lends considerable support to the contention that a labile toxic element is responsible[6 9 10 12 14]. Taking the findings of previous workers into account it seems that, after harvesting, the toxin is destroyed by desiccation, temperatures above 0°C, weak acid solutions or possibly all three factors.

The plants were derived from veld which had caused a severe outbreak of geeldikkop with a high morbidity and considerable mortality. Subsequently no similarly severe cases of geeldikkop have been reported in the area, probably due to the very good rains which have fallen in later seasons. Such cases as did occur, were of limited extent, and often only young lambs, which are known to be highly susceptible, were involved, while adult sheep in the same camp were not clinically affected. As a result it has not been possible to date to confirm the results of this paper. Highly toxic material which has caused an extensive outbreak in adult sheep and which is harvested under conditions identical to those prior to the outbreak, seems necessary for success.

The finding that 3 kg of known toxic *T. terrestris* may be preserved without serious loss of toxicity for at least 6 weeks provided it is immediately frozen and kept frozen, nevertheless represents a potentially valuable finding which may lead to the identification of the elusive toxic principle.

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AN OUTBREAK OF "KIKUYU POISONING" IN WESTERN TRANSVAAL

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ABSTRACT: Van Heerden J.; Williams M.C.; Van Rensburg I.B.J. and Ipland F.F. An outbreak of "Kikuyu poisoning" in Western Transvaal. *Journal of the South African Veterinary Association* (1978) **49** No. 1, 27–30 (En) Dept. Medicine, Fac. Veterinary Science, Univ. of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

An outbreak of mortality in cattle grazing kikuyu *(Pennisetum clandestinum)* pastures on a farm in the western Transvaal is described. These pastures had been heavily infested with army worm *(Spodoptera exempta)* two weeks preceding the onset of deaths. The main symptoms were excessive salivation, paresis of the tongue and pharynx, localized fine muscular tremors, ruminal tympany and stasis and congested or cyanotic mucous membranes. Significant clinical pathological findings were leukocytosis, elevated blood sugar levels and lowered serum magnesium and sodium levels. Autopsy findings of interest were patchy or diffuse hyperaemia of the rumen and abomasum and small haemorrhages in especially the lymph nodes, heart and tracheal mucosa. The most important histopathological finding was superficial or deep necrosis of the ruminal epithelium with marked neutrophile infiltration. All forms of treatment, including intravenous administration of magnesium sulphate, were of no avail. The condition closely resembles so-called "Kikuyu poisoning" in New Zealand.

INTRODUCTION

Outbreaks of mortality in cattle following the grazing of kikuyu grass *(Pennisetum clandestinum)*[1 2 7] in northern New Zealand have been described, the condition being known as "Kikuyu poisoning"[7]. Poisoning by kikuyu pastures has also occurred in Western Australia[3] and is suspected to have occurred in Rhodesia and South Africa[4].

Sheep have also been affected[6]. Although the exact aetiology has yet to be established, mortality has been ascribed to a physiological disorder related to ruminal engorgement[2] or to mycotoxin producing strains of *Myrothecium sp*[5]. Excepting the outbreak in Australia, mortalities were always associated with army worm *(Pseudaletia separata)* infested kikuyu pastures[1 2 8]. This report deals with an outbreak of mortality in cattle on a farm in the Koster district of the Transvaal. Deaths occurred in animals grazing on army worm *(Spodoptera exempta)* infested kikuyu pastures.

EPIZOOTIOLOGICAL ASPECTS

The property on which deaths occurred is situated in a summer rainfall area. The average monthly precipitation for the 3 m preceding the outbreak was 131,3 mm. The average rainfall for the same months over the preceding 27 y was 94,9 mm. The maximum and minimum diurnal temperatures for the preceding 2 m were 31,6°C and 11,0°C, respectively. Maximum diurnal temperatures ranged from 25,5°C to 31,6°C in February and from 19,5°C to 31,6°C in March.

Grazing consisted of separate artificially established kikuyu and buffalo grass *(Cenchrus ciliaris)* paddocks. These camps were regularly irrigated and were fertilized in early August with an N:P:K mixture followed by the application of 150 kg of urea per hectare every 6 weeks. They varied from almost flat to well sloped in contour. A few large trees in each camp provided small shaded areas. Although they had been in use for 1 to 7 y, no infestation with army worm or unexplained deaths had occurred during this period.

On visiting the farm during the outbreak at the end of March 1977 the cenchrus pastures were found to be heavily infested with army worm while the kikuyu pastures were only very lightly infested. According to the farm manager, however, the kikuyu paddocks had been heavily invaded by army worm 2 w previous to our visit.

All mortalities during the outbreak could be traced to cattle that had grazed on army worm infested kikuyu and/or cenchrus pastures. Deaths occurred in 3 groups of animals (A, B and C) but not in a further 2 groups (D and E). All animals had free access to an urea-free commercial lick.[+]

Group A consisted of Hereford females which grazed on cenchrus pastures by day and on kikuyu pastures at night. Of this group only a single animal became ill and died.

Group B also consisted of Hereford females. They were pastured alternately in cenchrus and kikuyu paddocks for 2 to 4 d. The heaviest mortality occurred in this group (Fig. 1).

Group C consisted of four Friesland cows pastured continuously in kikuyu paddocks. One animal became ill and died.

Group D was represented by Hereford bulls in kikuyu camps. These animals received supplementary feeding in the form of cenchrus hay and a commercial feed ration.[++] None of these bulls were affected.

Group E consisted of a small number of Brahman cattle which grazed exclusively on kikuyu pastures. None of these animals became ill.

With the exception of the Friesland cow all other affected animals were Hereford females. The majority of the latter were pregnant heifers.

CLINICAL FINDINGS

In the five groups of cattle discussed above 17 out of 125 animals became ill; 76,4% of these subsequently died. In fatal cases death occurred within 12 to 48 h of symptoms being shown, the animals becoming recumbent terminally. Non-fatal cases remained standing, only lying down intermittently for varying periods. Four animals made a complete recovery within 4 to 7 d. No further cases were seen after approximately 72 h following removal of the cattle from the pastures.

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[+]A lick specially formulated by Epol Stock Feeds, Box 175, Silverton, Pretoria.
[++]Epol Bull Ration. Epol Stock Feeds, Box 175, Silverton, Pretoria.

The outstanding clinical symptoms were: excessive salivation, partial paralysis of the tongue, localized fine muscular tremors, ruminal tympany and stasis and congested or cyanotic mucous membranes.

Affected animals appeared depressed and showed inco-ordinated movements. On lying down the position most often assumed was lateral recumbency with the head extended, together with slight opisthotonus. Animals in lateral recumbency showed periodic convulsions with the head thrown backwards and all four limbs extended. Occasionally a position of sternal recumbency with the head and neck turned towards the flank, was assumed. One recumbent heifer showed marked muscular tremors of the tongue. Some individuals were observed to intermittently stand up and lie down. Muscular fremitus, especially over the shoulder and flank regions, was seen in affected animals.

In some animals the tongue protruded slightly. On pulling the tongue the tonus was found to be reduced and withdrawal was weak. Attempts to clean the nostrils with the tongue were unsuccessful. Profuse salivation was observed. Standing animals repeatedly approached drinking troughs, putting their muzzles near or into the water but appeared unable to swallow. Although the majority of individuals made unsuccessful attempts to ingest food some were seen to feed between periods of lying down and were observed to ruminate. The latter cases seemed remarkably normal between periods of recumbency. In all affected cattle tympany occurred, being most marked in recumbent individuals. On auscultation of the abdomen of non-ruminating affected animals, complete ruminal stasis was evident. Occasional grunting, grinding of the teeth and repeated turning of the head towards the flank was seen. The mucous membranes were markedly congested in all individuals and cyanotic in prostrate animals.

Some animals showed fast, shallow respirations. In recumbent individuals the heart rate varied from 120 to 140 with a weak pulse. Clinical dehydration was evident in only one case. Three affected animals showed a fever (Because of the danger of tickborne diseases on the farm the temperatures of all cattle are measured twice daily).

CLINICAL PATHOLOGY

Blood samples were taken from seven affected and five unaffected animals which were randomly selected for this purpose during a visit to the farm. Results of the clinical pathology tests of them are given in table 1.

Five of the affected cattle died within 1 to 10 h of the blood sample being taken. There was no evidence of haemoconcentration. Five of the affected animals showed definite leukocytosis. Three affected individuals showed markedly elevated blood sugar levels. Generally, affected animals had lower serum magnesium levels than the randomly sampled normal cattle. The serum sodium level was decreased in five of the affected individuals.

NECROPSY FINDINGS

Necropsies were performed on three 2½ y old Hereford heifers that had died during the outbreak. All were about 4 m pregnant. A composite description of the autopsy findings is given below.

The animals were in good condition but two were mildly dehydrated as evidenced by slightly sunken eyeballs and dark red, viscous blood. Two heifers showed fairly marked abdominal distention. All the carcasses showed moderate general congestion and cyanosis. The lungs, lymph nodes and brains of all three animals were markedly congested.

Table 1: RESULTS OF CLINICAL PATHOLOGY TESTS

	Affected cattle		Unaffected cattle	
Determinations	Range	Mean	Range	Mean
Hb (g/1)	133 – 167	146	122 – 150	127
RCC (10^{12}/1)	5,95 – 11,00	7,81	4,41 – 7,92	6,15
Ht	0,34 – 0,415	0,388	0,305 – 0,425	0,363
WCC (10^9/1)	8,6 – 41,8	22,17	8,5 – 16,5	12,9
Ca (m mol/1)	1,72 – 2,75	2,04	1,5 – 2,47	2,13
Mg (m mol/1)	0,42 – 0,81	0,52	0,75 – 1,5	1,18
B. sugar	2,66 – 11,9	6,7	2,8 – 4,2	3,2
Na (m mol/1)	104,8 – 144,0	123,4		
Rumen pH (3 cows)	6 – 7			

The most significant lesions were found in the gastrointestinal tract. In all three heifers the rumen was very well filled with bright green, sloppy, well chewed ingesta. In one case a few patches of mild ruminal hyperaemia about 50 mm in diameter were seen. In another heifer the abomasal mucosa was diffusely hyperaemic while in the other the mucosa showed multiple disseminate foci of hyperaemia. No macroscopic lesions were seen in the small intestines and the contents appeared normal. In one the contents of the large intestine was drier than normal and decreased in quantity. In two of the animals, ruminal ingesta was found in the oesophagus and pharynx.

Haemorrhages, varying from petechiae to ecchymoses, were found in many organs but appeared consistently in the lymph nodes, heart (particularly subendocardially) and tracheal mucosa. The lungs, besides being markedly congested, also showed moderate oedema. A mild splenomegaly due to congestion of the red pulp was seen in all three cases.

HISTOPATHOLOGICAL FINDINGS

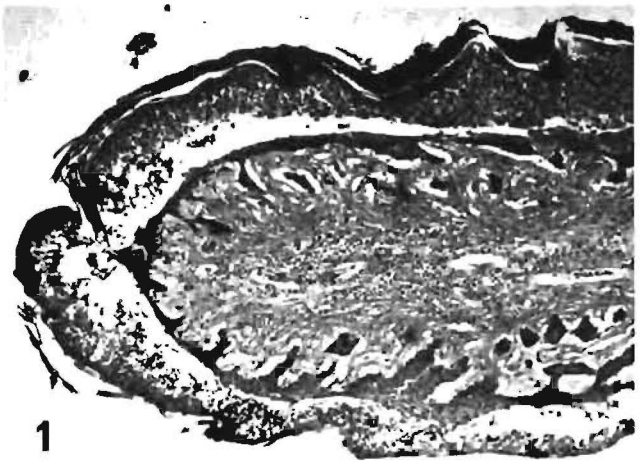
Specimens of various organs from two of the cases autopsied were examined. The following results were obtained:

Rumen Wall

The most significant lesions observed were found in the mucosa of the rumen. The superficial layers of the mucosa were completely necrotic, but the depth of the necrosis varied in different areas. In some instances it went as deep as the *stratum basale* while in others it involved only the *stratum spinosum* and the superficial layers. The necrotic layers sloughed easily and were intensely infiltrated by neutrophiles. The muscular layers were not affected, although hyperaemia and some haemorrhage was apparent. (Figures 1, 2 and 3).

Liver

In the cases showing least post mortem changes the hepatocytes stained more eosinophilically than normal,



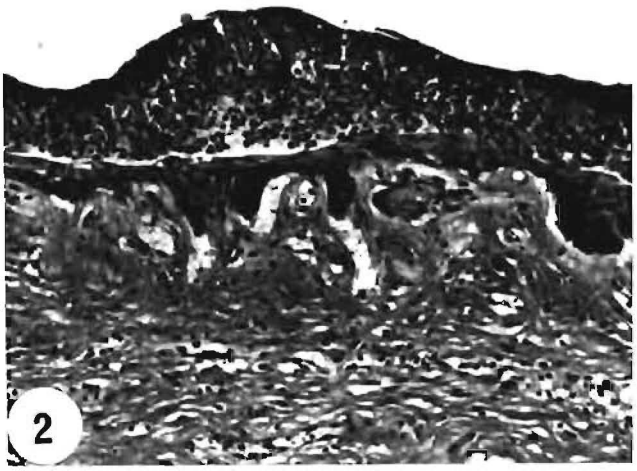


Fig. 1 Rumunal papilla showing necrosis of epithelium with neutrophil infiltration. H & E 40 X.
Fig. 2 & 3 Rumunal wall illustrating epithelial necrosis and neutrophil infiltration. H & E 100 X & 400 X respectively.

some having an almost hyaline appearance. Under high magnification (1000 ×) small eosinophilic streaks or droplets criss-crossed the cytoplasm. This was interpreted as being caused by dilation of the smooth endoplasmic reticulum which contained an eosinophilic staining material. The nuclei were unaffected. A few small areas of neutrophilic accumulation were noticed in both cases, which is similar to the observations of Martinovitch and Smith[7]. There was an increased cellularity of the portal areas and although some neutrophiles and round cells were present, not all the cells present were leukocytes. It seemed as if pericytes from blood vessels as well as fibroblasts had proliferated to some extent.

The epithelium of the majority of bile ducts was swollen, which resulted in narrowing the lumen. The epithelial cells in some of the larger bile ducts contained fine basophilic granules in their cytoplasm, the nature of which could not be ascertained.

Lung

The organ was severely congested in both cases. Focal haemorrhage had taken place while the alveoli were filled with a protein-rich oedematous fluid. In one of the cases the muscular coat in the medium-sized arteries showed increased eosinophilia.

Kidney

In both cases there were focal areas of round cell intistitial nephritis. In one of these this was localized to the corticomedullary junction in the vicinity of the larger blood vessels. The cytoplasm of the tubular epithelium had a granular appearance but the cells were not swollen. In one case the cytoplasm contained fine eosinophilic droplets in some areas. In this animal the tubular lumens contained a dense pink-staining proteinaceous material which was so concentrated that no sharp distinction between cell cytoplasm and lumen content could be seen. The glomeruli revealed increased cellularity due to mesangial proliferation.

Spleen

This organ was examined in only one case. It was very congested while the germinal centres had a "washed out" appearance. In some of these karyorrhexis of the lymphocytes was encountered.

Brain

There was congestion and some microscopic haemorrhages. A few small foci of paraventricular gliosis were observed. Around a few blood vessels in one case only, small eosinophilic droplets occurred in the Virchow-Robin spaces. The submeningeal space contained a pink-staining fibrillar material giving the impression that the cerebro-spinal fluid contained more protein than usual.

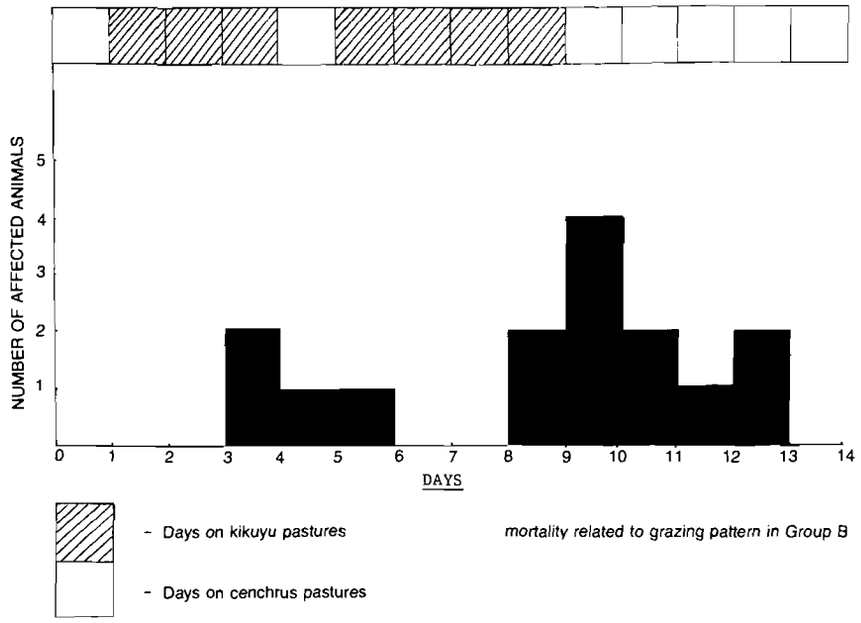
Myocardium

This was examined in one animal only. It was congested and the subendocardial connective tissue as well as around the larger blood vessels seemed oedematous and resembled mucoid degeneration. A slight leukocytic infiltration was present in these areas. The muscle fibres revealed no significant lesions.

TREATMENT

The finding of low serum magnesium values prompted initial treatment of affected animals with magnesium sulphate*. Piracetam** was administered intravenously. Antibiotic therapy*** was instituted in some cases. All these forms of treatment were ineffective.

*350 cc of a 20% magnesium sulphate solution, subcutaneously.
**"Nootropil", U.C.B. S.A., De Korte Street 93, Braamfontein, Transvaal. 30 mg/kg intravenously.
***Penicillin or tetracyclines.



DISCUSSION

The condition described shows a marked similarity to kikuyu poisoning as reported by other workers[1 2 3 7]. To a greater or lesser degree there are resemblances in the clinical, pathological and epizootiological aspects.

Although most of the animals which became ill were grazing on both kikuyu and cenchrus pastures, manifestations of the condition and mortalities in every case could be related to grazing of kikuyu grass. Brahmans (Group E) which were grazing on kikuyu grass heavily infested with army worm showed no mortalities. Deaths in Group B peaked following their grazing in kikuyu paddocks and dropped once they were withdrawn from these pastures. (Fig. 4).

Marked dehydration, as reported by Martinowich & Smith[7], was not observed (Table 1) in any affected animals and could perhaps be ascribed to the fact that death in the majority of cases occurred fairly rapidly.

The excessive salivation observed in most cases is considered to have been due to inability of the animals to swallow. Paresis or paralysis of the pharynx was evidenced by sluggish tongue movements and inability to ingest food or water. Ephemeral fever, botulism and diplodiosis, all conditions causing a similar bulbar paralysis, were excluded as possible causes of mortality in this outbreak. Inability of the animal to swallow saliva, of high sodium content, may explain the lowered sodium-serum values. Additionally the necrosis in the rumen epithelium may have resulted in reduced uptake of sodium ions.

The observed leukocytosis could perhaps be related to the lesions described in the forestomachs and/or stress-induced cortisol release[8].

Hypomagnesaemia is a relatively unimportant clinical entity in the Republic of South Africa and perhaps only of importance in certain areas. However, since grazing conditions on this particular farm could give rise to such a syndrome, serum-magnesium values were immediately determined. Although low blood levels of magnesium were found, administration of magnesium sulphate had no beneficial effects. The only explanation for the low magnesium levels that can be offered is that they are secondary to reduced uptake from the digestive tract.

The high blood-glucose levels were all obtained from animals in the terminal stages of the disease and could therefore be ascribed to the effects of severe stress.

In conclusion we would advise that during seasons when army worm infestation of pastures reaches epidemic proportions, cattle be removed from affected kikuyu pastures for at least 6 weeks. Since the period for which the kikuyu remains toxic is not known with any certainty, a few less valuable cattle should be used to test the pasture for "toxicity".

ACKNOWLEDGEMENTS

The authors wish to express their gratitude towards Drs J H Williams, A Immelman and the technical staff of the departments of Medicine and Pathology for their assistance; Messrs C Friedman, D Robinson and D Howarth for their cooperation and hospitality; and Profs R C Tustin and K van der Walt for reading the manuscript.

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LETTER TO EDITOR — BRIEF AAN REDAKSIE

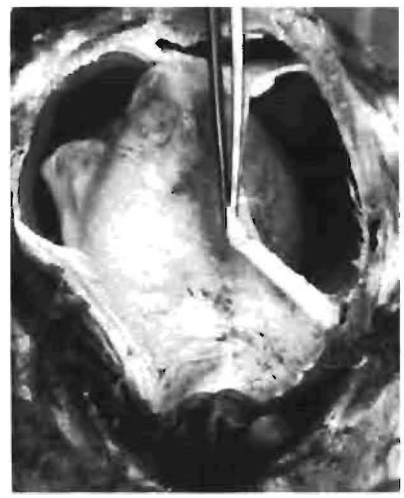
HYDRANENCEPHALY IN CALVES IN THE NATAL REGION

Sir

Further to Dr Barnard's letter in October 1977's edition of the Journal, our observations in Natal may be of interest to colleagues.

During the late summer of 1977, isolated cases of this condition came to the notice of this laboratory. Eventually cases were recorded from all over the province with no definite foci of infection being pin-pointed.

All breeds are apparently affected, with the condition being seen in Frieslands, Charolais, Sussex, Aberdeen Angus, Herefords, and various crosses. Males and female calves are affected although more males than females were submitted for examination. The condition is more readily spotted in dairy calves, less so in beef cattle running with dams. Calves examined varied in age from 2 days to a month or more. No doubt many calves have, in the past, been destroyed as weaklings, etc.



Symptoms observed:

Inco-ordination, blindness and deafness occur. On handling some affected calves go into convulsions which are followed by ataxia and typical opisthotonus. Most calves cannot drink unaided and are hand reared on the assumption that they are weaklings and will eventually recover.

Skeletal and muscle development is normal and superficially it is difficult to detect that the calves are abnormal. Hand rearing often results in secondary colisepticaemia.

Post mortem lesions are confined to the brain, except where secondary infection has occurred. The cerebral hemispheres are partially or completely replaced by a straw coloured watery fluid, which is occasionally blood tinged. The amount varies according to brain size and the severity of the condition and ranges from 150–250 ml. In some cases a thin layer of cerebral cortex remains. The fluid is not under pressure nor is the bone structure of the cranium affected, as in some forms of hydrocephalus.

The ventral portions of the brain, the cerebellum and medulla are not visibly affected. The meningeal membranes are thickened and in advanced cases are very thick indeed, if the calf has survived a month or more. In early cases, the cerebral hemispheres show a brownish-black discolouration of the frontal lobes of the cerebrum similar to necrosis. Controvery exists as to whether the virus produces its effect by cerebral destruction or by interference in cerebral development in the embryo.

In discussion with colleagues at Onderstepoort, it was mentioned that the condition could be due to intrauterine infection of the foetus with one of several viruses namely Bluetongue, Wesselsbron, Rift Valley fever or Akabane. The latter has been diagnosed in Japan, Australia and Israel and causes hydranencephaly, arthrogryposis and abortion. It has been confirmed serologically in South Africa in the Transvaal region. Sera from dams of affected calves and calves themselves, before and after suckling have been submitted to Onderstepoort for examination. In addition, fresh brain tissue, cerebrospinal fluid and other tissues have been sumbitted for virus isolation.

To date, antibodies to bluetongue and Akabane viruses have predominated while there has been a smaller incidence of antibodies to Wesselsbron and Rift Valley fever viruses. The serological survey has now been extended to all known infected farms, and those farms adjoining. In addition farms on which cases of undiagnosed abortions have occurred are being checked. On several of these farms, clinical cases were confirmed which would not have been reported otherwise, and in addition several farmers have mentioned that other calves have been destroyed which had shown similar symptoms.

The condition is probably much more widely distributed than at first thought and if abortion is part of the effect of Akabane virus, as well as abnormal calves, it assumed an economic importance. Inoculation of non-pregnant females with blue tongue vaccine is a matter for consideration. Wesselsbron (ovine) and Rift Valley fever vaccines are of course available, but so far, no commercial Akabane virus vaccine has been produced, although recent reports indicate that a vaccine for Akabane virus has been used on a trial basis by Japanese workers. The latter virus is associated with arthrogryposis as well as hydranencephaly and abortion, but arthrogryposis was never seen in cases investigated by members of this laboratory. Infection with Akabane virus confers lasting immunity.

Investigations are still in progress and results will be published at a later date. Comments from colleagues would be welcome.

Yours faithfully

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FIELD OBSERVATIONS ON THE OCCURRENCE OF RABIES IN CATTLE IN THE MAGISTERIAL DISTRICTS OF SOUTPANSBERG AND MESSINA

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ABSTRACT: Brückner G.K., Hurter L.R., Boshoff J.N. Field observations on the occurrence of rabies in cattle in the magisterial districts of Soutpansberg and Messina.

Journal of the South African Veterinary Association (1977) **49** No. 1, 33–36 (En) Private Bag X2408 Louis Trichardt 0920, Republic of South Africa.

The field manifestation of rabies in cattle is discussed. The observations cover the period 1974–1977 with emphasis on the geographical spread, vectors, symptoms and immunization of cattle with the Flury H.E.P. (high egg passage) rabies vaccine as prepared by the Onderstepoort Veterinary Research Institute.

INTRODUCTION

Observations under field conditions on the occurrence and spread of rabies in cattle were made over a four year period (1974–1977) in the magisterial districts of Soutpansberg and Messina. Sporadic cases of rabies were encountered within the area before 1974[1] [2] mostly in jackal *(Canis mesomelas)*, to a lesser extent in cattle and only ocasionally in dogs and cats.[3] Since 1974 the occurrence of the disease in cattle has been alarming, so that probably for the first time the farming community regarded the disease as of economical importance apart from being a zoonotic hazard.

The area is a rabies proclaimed area where a permit system is enforced for the movement of dogs and cats. All dogs are officially vaccinated annually, free of charge, with the Onderstepoort LEP rabies vaccine. Cats are vaccinated within a 25 km radius of a confirmed focus.

SPREAD OF THE DISEASE

Reports were received during the latter part of 1973 that rabies was apparently causing concern in the bordering states north of the Limpopo River.[7]

During March, April and May 1974 three cases were diagnosed in the district of Messina on farms adjoining the southern bank of the Limpopo. All three cases were in the Black-backed jackal *(Canis mesomelas)*. An average period of 32 d elapsed between each positive case.

Two months later a bovine was found positive on a farm 30 km west from where the last of the 3 jackals was found to be positive for rabies.

The spread of the disease took on a south-westerly course towards Alldays, went further south to by-pass the western buttress of the Soutpansberg mountain range and then east towards Louis Trichardt. A total number of 121 cattle on 40 different farms contracted the disease until August 1977. See Fig. 1 showing the spread of rabies and tables 1 and 2 for a summary of positive cases.

VECTOR

It is assumed that the cattle were bitten by rabid jackals, but this could not be proved beyond doubt. Definite bite marks could be seen in only 2% of all post mortems performed. Not knowing what the actual incubation period in each case was, it is possible that the presumed bite mark or wound inflicted by a rabid jackal would have healed by the time the disease was diagnosed.

The mode of transmission to cattle in most cases could not be determined without doubt, but it can be reasonably assumed that one or other member of the species *Canis mesomelas* was responsible. In 60% of cases where rabies was confirmed in cattle, rabid jackals or jackals with an abnormal behaviour pattern were seen nearby 3 weeks or more prior to the onset of symptoms in cattle. These jackals were observed by farmers and farm labourers, usually only by chance, which accounts for the 13 jackals submitted for diagnosis and of which all were subsequently found positive. Two of these were found drowned in a cattle drinking trough. It is estimated that a fairly concentrated population of jackals is present north and north-west of the

Table 1: OCCURRENCE OF RABIES IN CATTLE: 1974–1977. DISTRICTS OF SOUTPANSBERG AND MESSINA.

Year	Number of different farms	Number of positive cattle
1974	2	2
1975	14	57
1976	17	47
1977	7	15
Total	40	121

Table 2: OCCURRENCE OF RABIES IN SPECIES OTHER THAN CATTLE: 1974–1977.

Year	Species	Number of different farms	Number Positive
1974	Canis mesomelas	4	4
1974	Canis domesticus	1	2
1975	Canis mesomelas	3	4
	Canis domesticus	2	2
	Felis domesticus	1	1
	Procavia capensis	1	1
1976	Canis mesomelas	3	4
	Mellivora capensis	1	1
1977	Canis mesomelas	1	1
	Civettis civetta	1	1
Total		18	21

Total number of jackals positive: 13.

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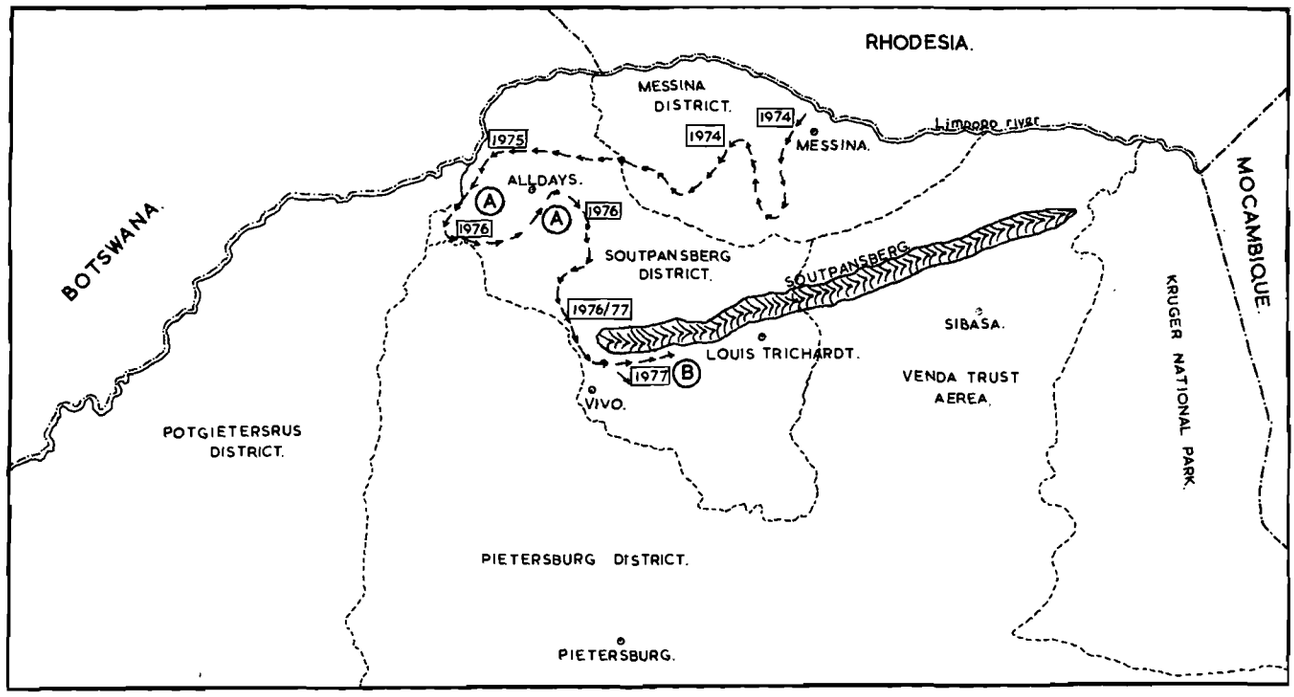

Soutpansberg. On one farm more than 100 jackals were poisoned in one night in an area not larger than 40 km².

The disease seemed to be most prevalent during the months of July to November which coincides with the mating and whelping season of this species.[8]

Bueler[8] states that the mating of *Canis mesomelas* in South Africa takes place from May through July. Since the gestation period is 60 to 65 days, whelping occurs from July through September. During this period the jackals are not confined to their normal defined hunting areas.[8]

A female jackal seeking a mate attracts a following of several males[6 8].

The prevalance of rabies during this period thus seems to coincide with the increased migratory behaviour of jackals during this period especially as far as the male member of the species is concerned. This is also the time when natural food resources eg. small rodents, ground birds, wild berries and fruit are less abundant[68]. Roberts[6] states that: "The Black-backed jackal is a hardy and resourceful animal, wandering over great expanses of country when in search of food".

Bueler[8] estimates that they normally cover 8 to 10 km per hour and prefer to follow paths, roads or natural features of the terrain that allow easy travelling. It can keep up a steady pace for hours and may cover 30 to 40 km in a single night if food is scarce.[6 8]

The chances that a healthy jackal would then came into contact with a rabid member of the species are therefore presumably enhanced.

It is then reasonable to deduce that jackals appear to be the most important vectors of rabies in this area. No evidence could be found that the disease can be transmitted from one bovine to another.[3 4]

SYMPTOMS IN CATTLE

It appears that those cattle highest in the ranking order of a herd, are the most likely victims of a rabid jackal. Of the positive reactors, 60% were from animals which occupied the top rank in a herd. They would usually be the first to confront or attack a foreign intruder to the herd. The following symptoms were observed:

1. Attenuation

This rather than emaciation was a common finding and coincides with the early onset of pharyngeal paralysis.

2. Paralysis

Which most often starts in the hindquarters – very often only in one leg with a typical "knuckling" at the fetlock joint.

Swaying of the hindquarters, almost identical to the symptoms shown by an animal with lumbar trauma or an epidural abscess, was seen. This usually progressed rapidly to a total paralysis in all the extremities within 4 to 6 d after the onset of symptoms.

3. Salivation

Was observed in 30% of cases – mostly only a slight dribbling of saliva rather than profuse salivation.

4. Hydrophilia

This was observed in contrast to the expected hydrophobia. In one instance a heifer with obvious pharyngeal paralysis tried to drink unsuccessfully 25 times over a 30 minute period. This craving for water probably also occurs, as mentioned earlier, where rabid jackals were found drowned in a drinking trough.

5. Aggressiveness

Cattle turned aggressive only on provocation. Where total paralysis of the limbs has set in this symptom was seldom or ever seen, even with extreme provocation.

They would usually attempt to charge a person when provoked, but in no instance was the charge continued with the aim of eventual attack. They would stop within a few yards and stagger away aimlessly. Although other cattle were seen to shy away from a rabid member in the herd, no instance was recorded where rabid cattle tried to bite or attack other cattle within the herd although butting was observed.

6. Straining

This symptom was constantly observed. Repeated efforts were made either to defecate or urinate.

In 4 instances farmers confused this symptom with the typical straining often seen during parturition.

7. Pseudo-oestrus

Observed in both pregnant and non-pregnant heifers and cows. Continuous attempts were made to mount other cattle. A mucoid, vaginal discharge ("bull-string") was observed in a cow 7 m pregnant.

8. Bellowing

Not a common symptom during the early stages of the disease occuring mostly in the terminal phase. The sound differs from the normal in that the bellow of a rabid animal is more hoarse to highpitched. Continuous bellowing for variable periods of time and also attempts to bellow, without producing any sound were observed.

PREVENTATIVE MEASURES

Apart from the compulsory annual vaccination of all dogs in the area, the following regime was followed:

1. Eradication of Jackals

Farmer's meetings were held and the rabies threat evaluated. It was then decided to embark on an eradication campaign with the main aim of protecting valuable livestock and by using strychnine baited meat pellets.

The expected results were not achieved despite a fairly concentrated jackal population and because not all the farmers cooperated. The chances were small that a rabid jackal would still be able to swallow poisoned bait if pharyngeal paralysis had already set in and it would still be able to transmit the disease.

It did help in that the numbers of healthy jackals, being potential transmitters of the disease, were reduced on certain farms. The fact that a rabid jackal would most probably travel long distances, once it develops the mad form of the disease[4] lessened the chance that it would be eliminated by bait put out in a confined area and by a limited number of farmers.

Bueler[8] states that one reason for the plague of jackals in South Africa, may be that inbreeding, which limits fertility, has been discouraged by attempts at control. Under natural conditions jackals often find mates in their own litters, but, if persecuted, they tend to scatter, thus mixing gene pools. The system of inbreeding between litter mates might be a mechanism for keeping the jackal population stable and not allowing it to exceed its food supply. However it is doubtful whether this natural process of self limitation is sufficient to completely abandon the practice of controlled reduction in numbers by poisoning.

2. Vaccination

The farming community, realising the economic importance of the disease in cattle, were in urgent need of a suitable vaccine. Two vaccines were available:

a) The Flury H.E.P. vaccine as prepared by the Veterinary Research Institute, Onderstepoort.

b) A commercial live virus of porcine tissue culture origin (ERA strain)[9]

The Flury H.E.P. vaccine is only used officially for the vaccination of domestic cats. The vaccination of cattle is not compulsory in the Republic of South Africa and is not carried out under official supervision as in dogs and cats. Farmers may vaccinate cattle at their own expense and risk. The commercial vaccine was said to establish an immune status lasting as long as 48 months after a single vaccination[9] but the vast majority of farmers used the Flury H.E.P. vaccine because it was more readily available.

Immunisation required two intramuscular injections within a 30 d interval.

It was not possible to assess the effectivity of the Flury H.E.P. vaccine in cattle by trial infection – The only assessment being based on field observations. (Table C).

RESULTS AND DISCUSSION

As mentioned the chances are limited for one bovine to infect another. The number of cattle that could contract the disease on only one farm is probably determined by:

a) The immune status of the cattle.
b) The number of non-domestic animals contracting the disease.
c) The number of cattle attacked by a rabid vector. (An average number of 2 positive cattle per farm was recorded, with a maximum of 10 on one farm.)

It is most unlikely that an infected jackal would remain long enough on the same farm to transmit the disease to more than one or two cattle. This theory is supported by the manner in which the disease "jumped" geographically. Successive positive cases often being diagnosed 30 to 40 km apart.

As a result vaccination of cattle in a wider area than just on farms adjoining the infection became necessary. Vaccination in area A on the map commenced during June–July 1976 with most of the infected and surrounding farms fully vaccinated by June 1977.

The 4 farms on which positive cases were diagnosed two months post vaccination are within area A. No further positive cases occurred on the other 13 farms post vaccination.

Table 3: OCCURRENCE OF RABIES IN CATTLE BEFORE AND AFTER VACCINATION ON 17 FARMS: AREA A: 1974–1977.

Before vaccination		After vaccination	
Farms	Cattle positive	Farms	Cattle positive
17	61	4	12

1. On one farm, all 380 cattle except one control heifer were vaccinated twice with a 30 day interval. The heifer contracted rabies one month after the last vacci-

nation. A suspected case of rabies in a jackal was confirmed on this farm only 24 d prior to the onset of symptoms in the heifer. The rabid heifer was left amongst the other vaccinated cattle and was shot on the seventh day after the commencement of symptoms. None of the other animals contracted rabies.

2. Six cattle died of rabies on a farm completely surrounded by farms on which all cattle were vaccinated (Area A) and where no vaccinations were done. Evidence suggested that the rabid jackal had travelled through the vaccinated farms without transmitting the disease.

3. Most of the outbreaks occurred in the Alldays area and an attempt was made to have all the cattle vaccinated. Two farmers refused to vaccinate until rabies was confirmed on farms 2 months after the cattle on the surrounding farms were fully vaccinated. The disease spread to area B during 1977 and the same phenomenom occurred where one farm was left unvaccinated with two cattle on this unvaccinated farm contracting the disease 40 days post vaccination.

Vaccine reactions

No adverse reactions from either vaccine can be attributed to the attenuated virus itself. Slight allergic reactions were observed in cattle previously immunized against Chlamydioses with a vaccine consisting of formalin inactivated *Chlamydia psittaci* organisms cultured and propagated in embryonated eggs and emulsified in mineral oil. Some cattle have been vaccinated 3 times with no reports of anaphylaxis; contrary to the findings in South America[5] where continued use of the Flury H.E.P. vaccine has resulted in several cases of anaphylaxis.

On one farm 360 head of cattle were accidentally vaccinated twice with the Flury L.E.P. vaccine with no untoward symptoms. Their immune status could however only be ascertained by viral challenge.

Conclusion

It would appear that there was a definite decrease in the number of positive cases in cattle after vaccination.

South American workers have found that cattle inoculated with H.E.P. vaccine were resistant to challenge 22 months later.[5] Cattle vaccinated with the ERA strain resisted challenge 48 months post vaccination.[9] No trials of this nature have as yet been attempted in the Republic of South Africa and annual vaccination is therefore recommended. Rabies is a definite threat to the health of livestock in the northern most districts of the Republic of South Africa and to protect them, preventative vaccination seems to be the most reliable precautionary measure.

ACKNOWLEDGEMENTS

The Director of Veterinary Services is thanked for permission to publish this article and Mr. E. Louw for preparing the map of the geographical spread of rabies.

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INFORMATION — INLIGTING

DOUBLE HORMONE TREATMENT FOR OVARIAN CYSTS IN COWS BRING THEM INTO HEAT QUICKLY

Two years ago, scientists from the University of Missouri reported that intramuscular injection of gonadotropin releasing hormone (GnRH) was a very effective treatment for ovarian cysts.

Their studies had shown that 80 per cent of cystic cows responded to GnRH treatment and came into heat in 18 to 21 days.

These scientists now believe that they can treat cystic cows with GnRH plus another hormone, and breed them without having to check whether or not they are in heat.

The scientists have found that cystic cows treated with GnRH and then with prostaglandin F-2 alpha nine days later, grew another follicle and came into heat in about three days, according to H. Allen Garverick, dairy scientist at the University.

This treatment will save dairymen eight days in bringing a cow back into production.

While there is no current research data available, the scientists speculate that it may be possible to breed the cows three days after treatment with prostaglandin without having to observe them for heat.

Dairy economists estimate that producers lose R1,30 for each day's delay in bringing a cow back into production. Says Dr Garverick, "With over one million cystic cows in the U.S. to-day, that would amount to about R10,5 million per year".

("New Treatment for Ovarian Cysts in Cows": News from University of Missouri-Columbia, Extension Information: 69th. Annual Meeting, American Society of Animal Science, University of Wisconsin, Madison, Wisconsin, July 24–30, 1977).

INFESTATION OF SPRINGBOK "BILTONG"* WITH BOOKLICE *LIPOSCELIS BOSTRYCHOPHILUS* BADONNEL 1931 (PSOCOPTERA: LIPOSCELIDAE)

H.N. VAN DER MADE and E.M. NEVILL

ABSTRACT: Van der Made H.N.; Nevill E.M.; Infestation of springbok "biltong" with booklice *Liposcelis bostrychophilus* Badonnel 1931 (Psocoptera: Liposcelidae). *Journal of the South African Veterinary Association* **49** No. 1, 37, (En) Vet. Res. Institute, 0110 Onderstepoort, Rep. of South Africa.

The finding of booklice on stored salted air-dried springbok venison is reported.

INTRODUCTION

The Psocoptera is an order of about 1700 species of small louse-like insects with large heads and rather long antennae. According to Smithers[2] they may be ... "found on the foliage or branches of trees and shrubs, on or under bark, on fences and walls in leaf litter, under stones, on rocks, in caves, in human habitations and in stored products".

Many families of this order have been reported infesting cereals and processed foods, particularly if these have been stored for long periods. One of the Psoquillidae, *Psoquilla marginepunctata* Hagen 1865, has been recorded infesting salami sausage in Brisbane, Australia[1].

REPORT

This report concerns the finding in March 1975 of *Liposcelis bostrychophilus* Badonnel 1931 on dry springbok biltong which had been stored for several months in a closed container. The specimen was forwarded from Windhoek in South West Africa.

Signs of spoilage of the biltong were visible to the naked eye. It was covered with white powdery material and salt crystals and there were holes and tunnels into which the insects retreated. Several comparatively large eggs were scattered singly on the surface. Fig. 1 shows the typical appearance of the insect.

DISCUSSION

Members of the family Liposcelidae are characterized by enlarged hind femora. Smithers[2] states that "species of *Liposcelis* sometimes occur in large numbers in stored products, houses, ships and merchandise stores; many species are of world-wide occurrence, the traditional 'booklouse' usually being one or other species of this genus. They also cause damage to insect collections and may occur in houses in sufficient numbers to be a nuisance. Members of this family are commonly found on or under bark". In Queensland, Australia *L. bostrychophilus* has been found associated with powdered potato, mixed grain residue in a farm silo, wet and mouldy wheat residue, bagged nut-in-shell peanuts, sorghum and wheat[1].

*salted air-dried raw venison of *Antidorcas marsupialis*

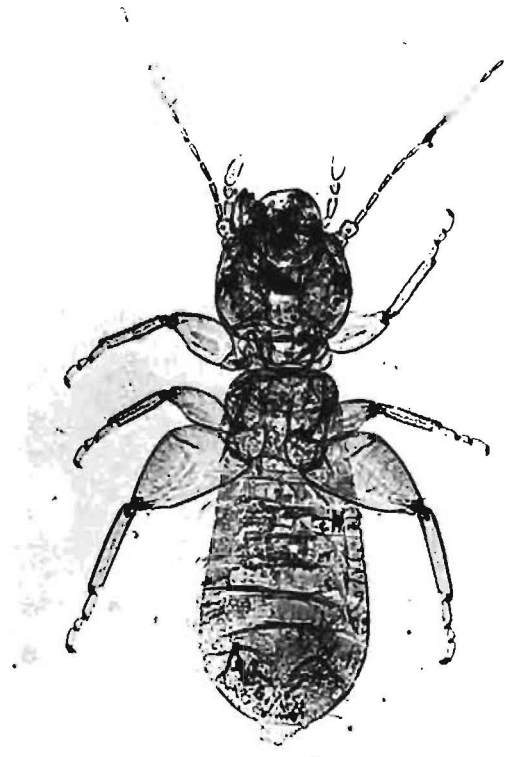

Fig. 1 *Liposcelis bostrychophilus* Badonnel 1931 Length (excluding antennae) 1,1 mm

ACKNOWLEDGEMENTS

Dr. E.K. Hartwig of the National Insect Collection, Plant Protection Research Institute, Pretoria is thanked for indentifying this insect and Mr. A.M. du Bruyn of the Photography Section, Onderstepoort for the photograph.

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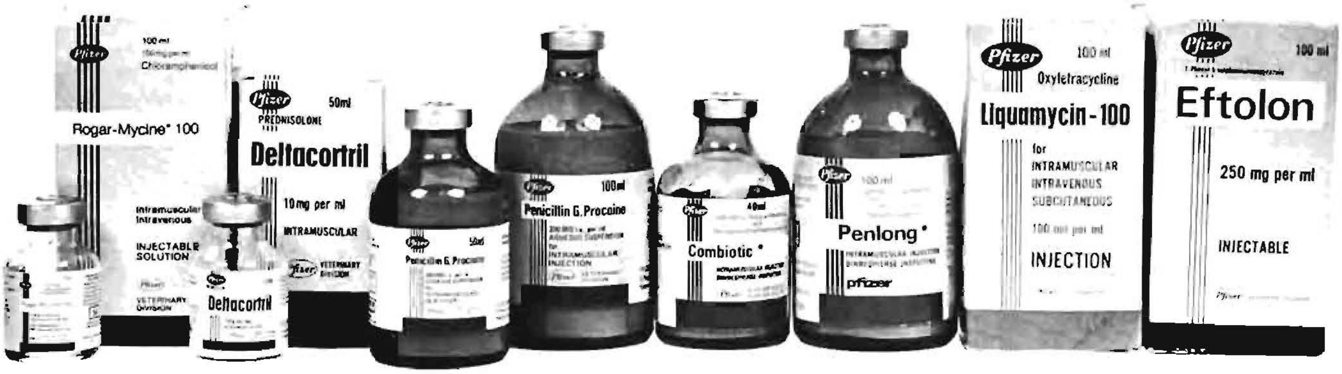

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PORCINE ARTHRITIS AND MEAT HYGIENE IN SOUTH AFRICA*

G V S TURNER**

ABSTRACT: Turner G.V.S. Porcine arthritis and meat hygiene in South Africa. *Journal of the South African Veterinary Association* (1977) **49** No. 1, 40–44 (En).

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The incidence, economic implications, aetiology and possible public health significance of porcine arthritis in South African slaughter pigs is discussed. An investigation into the aetiology and pathology of porcine arthritis as found at a South African abattoir revealed that *Erysipelothrix rhusiopathiae* was present in 48% of the affected joints and *Streptococcus* spp., *Corynebacterium* spp. and *Staphylococcus* spp. in 20%, 4% and 2% respectively. The joints showed various degrees of subacute to chronic proliferative sero-fibrinous to fibrinopurulent arthritis regardless of whether bacteria were isolated. It is essential that the *Lnn. axillares primae costae* which drain the forelimb joints also be examined routinely during meat inspection.

INTRODUCTION

For many years arthritis was consistently the second most common cause for the total condemnation of pig carcasses at the Newtown Municipal abattoir in Johannesburg (Table 1). During the period from July 1973 to June 1974 this condemnation rate of arthritic pig carcasses reached a peak and 748 pig carcasses (16,9% of the total number condemned) were condemned for arthritis at an estimated wholesale market value of R21 123,00 (Table 4).

This represented a total loss to the producer as there is no insurance cover on the total condemnation of pig carcasses in the controlled areas in South Africa. It was at this stage that the pig industry in South Africa also became alarmed at the relatively high incidence of arthritis in slaughter pigs and its concomitant economic implications. With the Newtown abattoir being the largest abattoir in South Africa and slaughtering 24 to 32% of the total number of pigs slaughtered in the controlled areas (Table 3), this abattoir was regarded as a model for the types of conditions and diseases encountered in slaughter pigs in South Africa.

For comparison the slaughter and condemnation figures of a private export abattoir were reviewed. For the year May 1976 to April 1977 arthritis was the fourth most important cause for the total condemnation of pig carcasses at this abattoir (Table 2). Furthermore, a small percentage of carcasses underwent partial condemnations for localised chronic arthritis which reflects additional economic loss to the producer.

In overseas countries the occurrence, importance and aetiology of porcine arthritis is well documented[2 4 6 8 11 13]. In many countries *Erysipelothrix rhusiopathiae* is regarded as the major aetiological agent responsible for polyarthritis in slaughter pigs[2 8 11].

E. rhusiopathiae is the cause of swine erysipelas in pigs and erysipeloid in man. In South Africa swine erysipelas is a scheduled and notifiable disease in terms of the Animal Diseases and Parasites Act (Act No 13 of 1956). Swine erysipelas manifests itself in three distinct forms: the peracute to acute septicaemic form, the subacute cutaneous or diamond skin form and the chronic form with arthritis and vegetative endocarditis[9 11 12]. Erysipeloid in man is a well documented zoonosis and is regarded chiefly as an occupational disease in many countries[9 11 12]. *Erysipelothrix* infection in man usually consists of an acute local cutaneous lesion invariably introduced through minor skin wounds[22]. The infection can become generalized with an acute septicaemia together with serious complications such as endocarditis, polyarthritis, and central nervous system involvement. The outcome may be fatal[9 12].

Because only animals which are normal and apparently healthy are sent for slaughter, the acute and subacute forms of swine erysipelas are seldom seen at the abattoir. The pathognomonic rhomboid urticarious lesions of the subacute diamond skin form are occasionally seen during meat inspection. The chronic arthritic form appears to be difficult to ascertain in the live ani-

Table 1: CONDITIONS REQUIRING TOTAL CONDEMNATION OF PIG CARCASSES AT THE NEWTOWN ABATTOIR EXPRESSED AS PERCENTAGE OF ANNUAL CONDEMNATIONS

Year (19—)	70/71	71/72	72/73	73/74	74/75	75/76
Slaughtered	208628	212716	222787	217994	188748	160676
Condemned	5652	5174	4427	4415	3323	2329
Cysticercosis	35,6	40,5	25,5	20,9	42,5	47,6
Arthritis	11,8	13,0	15,5	16,9	11,4	5,3
Gangrene	10,2	8,3	8,6	12,0	9,2	10,6
Nephritis	6,4	—	9,3	6,9	1,2	0,6
Pneumonia	—	4,8	6,8	6,6	7,6	10,6
Scrotal sepsis	5,5	4,7	5,6	7,9	2,6	3,3
Pyaemia	—	—	4,9	5,5	5,6	6,4
Miscellaneous	30,3	28,5	23,6	19,6	15,6	

Table 2: CONDITIONS REQUIRING TOTAL CONDEMNATION OF PIG CARCASSES AT THE ESKORT BACON CO-OP ABATTOIR EXPRESSED AS PERCENTAGE OF ANNUAL CONDEMNATIONS

Year	1975/76	1976/77
Slaughtered	75 827	86 646
Condemned	614	983
Pneumonia	36,8	37,5
Tuberculosis	22,6	9,9
Cysticercosis	5,3	17,3
Pyaemia	11,4	7,7
Arthritis	4,2	8,9
gangrene	1,6	3,9
Miscellaneous	17,9	14,4

*Based on a paper presented at the Public Health Group Session of the Biennial Scientific Congress of the South African Veterinary Association, Grahamstown, 29 August 1977.

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Table 3: ANNUAL NUMBER OF PIGS SLAUGHTERED AT SOUTH AFRICAN ABATTOIRS

Year	1970/71	1971/72	1972/73	1973/74	1974/75	1975/76
Total A	1 288 264	1 238 746	1 413 440	1 613 302	1 465 185	1 444 104
Controlled Areas – B % of A	51,0	53,0	51,0	48,0	48,0	46,0
Newtown Abattoir % of B	32,0	32,6	31,0	28,0	26,6	24,3

(Data by courtesy of the Livestock and Meat Industries Control board.)

Table 4: VALUATION OF ARTHRITIC PIG CARCASSES CONDEMNED AT NEWTOWN ABATTOIR

Year	1970/71	1971/72	1972/73	1973/74	1974/75	1975/76
Condemned	5 652	5 174	4 427	4 415	3 323	2 329
Estimated total mass condemned (kg)	298 425	276 809	230 646	226 931	166 150	110 161
Estimated total value (Rand)	125 926	138 404	115 854	124 679	124 978	93 229
Carcasses with arthritis-A	665	672	685	748	378	123
Estimated value of A (Rand)	14 816	17 976	17 926	21 123	14 216	4 923

(Data by courtesy of The Livestock and Meat Industries Control Board.)

mal but is the most common form observed at the abattoir during post mortem inspection of the carcass.

The genus *Streptococcus* contains organisms that are important pathogens of man and animals. Streptococci have been isolated from a wide variety of tissues in pigs of all ages[13]. Streptococcal infections in swine can give rise to various clinical manifestations such as arthritis, endocarditis, meningo-encephalitis, abscesses, mastitis, reproductive tract infections and various other disease processes[4 6 13]. *Streptococcus* spp. are regarded as the second most common cause of arthritis in pigs[6 8 13]. Streptococci in man are capable of infecting most body tissues and causing a variety of suppurative diseases. The most serious form of human streptococcosis is caused by Lancefield's group A streptococci although members of most serologic groups have been isolated from man[3]. The actual status of streptococcosis as a zoonosis is poorly defined at present. Current knowledge, however, suggests there may be several hazardous situations in which animals can contribute to human streptococcosis[3]. For example, ten cases of septicaemia and purulent meningitis caused by porcine streptococci have been reported in patients who had close contact with live or slaughtered pigs[14]. With the high incidence of streptococcal infections in man and the wide range of Lancefield's groups causing arthritis in swine, the danger of arthritic pig carcasses transmitting pathogenic streptococci to man always remains.

In the United Kingdom salmonellae, staphylococci and *Cl. welchii* accounted for approximately 76% of the recorded incidents of foodborne illness in 1969[10]. It has been estimated that about two million persons in the United States are infected annually with salmonellae[10]. Arthritic joints infected with salmonellae or staphylococci are a potential source of infection to man. The pathogenic serotypes of *Escherichia coli* in animals as a source of infection to man is gaining importance. In theory, pathogenic *E. coli* from the arthritic pig carcass could lead to infection in man.

With swine erysipelas being a notifiable disease and a zoonosis and *E. rhusiopathiae* being the main arthritogenic agent encountered in slaughter pigs, it is difficult to understand why the high incidence of arthritis in pig carcasses and the economic implications thereof did not prompt an earlier investigation into the aetiology of the problem in South Africa. With no such work having been carried out, it became apparent that an investigation into the problem was necessary in order to ascertain the aetiology, the extent of the lesions and the possible public health significance of arthritis in slaughter pigs as found at the abattoir.

MICROBIOLOGICAL AND PATHOLOGICAL INVESTIGATION

Fore- and hindlegs with unopened intact joints were obtained from freshly slaughtered pig carcasses which had been condemned for polyarthritis. In order to ensure satisfactory results the fresh material was processed in the laboratory on the day of slaughter.

The microbiological examination procedure catered for the isolation of the following micro-organisms: *Erysipelothrix rhusiopathiae; Streptococcus* spp.; *Corynebacterium pyogenes; Staphylococcus aureus; Escherichia coli; Haemophilus influenzae suis; Mycoplasma* spp.; *Salmonella* spp.; *Chlamydia* and viruses. Samples of the synovial fluid and synovial membranes of affected joints were aseptically obtained and inoculated onto specific media known to support the growth of the above mentioned micro-organisms. The gross pathology of each of the arthritic joints was also determined.

The results of the microbiological investigation are summarized in Table 5. *E. rhusiopathiae* was the most common cause of arthritis in the pig carcasses examined and *Streptococcus* spp. were the second most common.

All the joints showed a progressive subacute to chronic serofibrinous to fibrino-purulent arthritis with

Table 5: RESULTS OF MICROBIOLOGICAL EXAMINATION OF ARTHRITIC PORCINE JOINTS

Isolations	Number Carcasses Affected	%
Erysipelothrix rhusiopathiae	24	48
Streptococcus spp.	10	20
Corynebacterium pyogenes	2	4
Staphylococcus aureus	1	2
Negative	13	26
Total	40	100

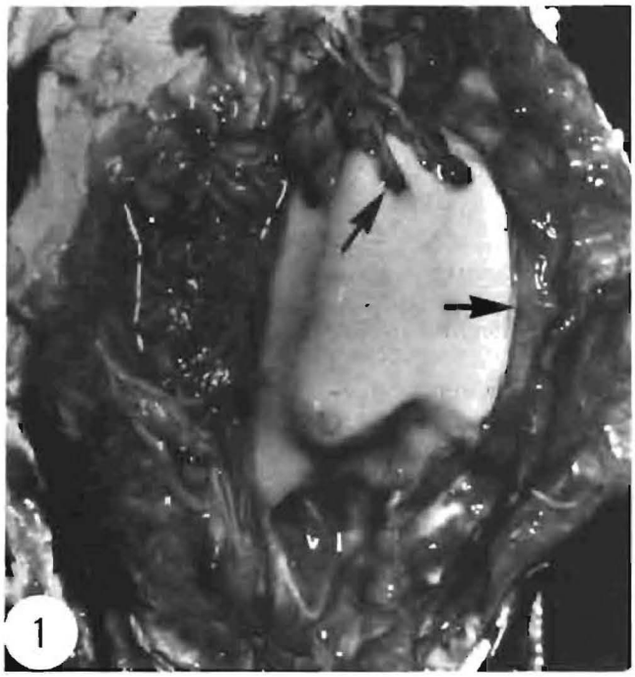

Fig. 1 Hypertrophy of the synovial villi and thickening of the joint capsule.

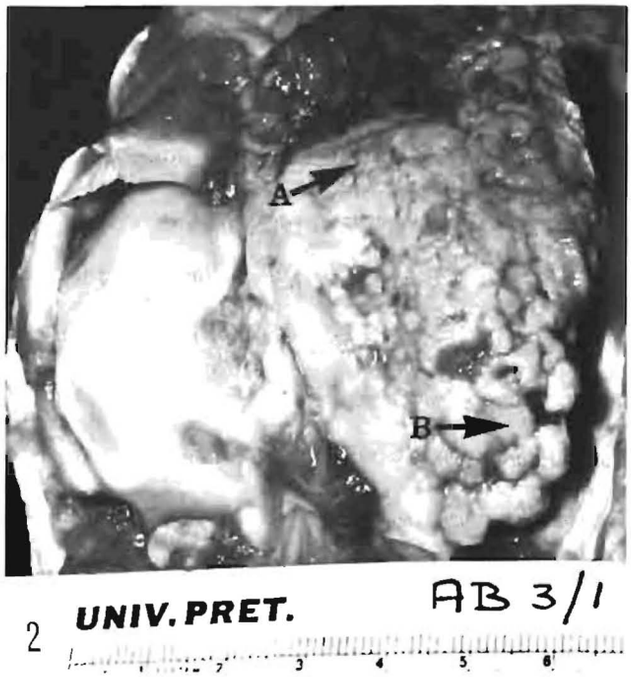

Fig. 2 Severe chronic arthritis with erosions (A) and osteophytes (B) of the articular cartilage.

marked hypertrophy of the synovial villi and fibrous thickening of the joint capsule (Fig 1). The amount of exudate varied from small amounts to 15 ml in one joint. In some cases erosions of the articular cartilage were observed (Fig 2). No correlation was found between the causative organism involved and the pathological changes in the arthritic joint.

INSPECTION OF THE PORCINE CARCASS

In the Republic of South Africa the Animal Slaughter, Meat and Animal Products Hygiene Act (Act No 87 of 1967) and the Standing Regulations thereof, provide for the maintenance of proper standards of hygiene in the slaughtering of animals and the handling of meat and for the prevention of the transmission of diseases to humans and animals by diseased or infected animals, meat or animal products[7]. The Act is applicable in toto to all the major abattoirs in South Africa. *Inter alia* the Act specifies that all animals intended for slaughter at an abattoir and all meat and animal products derived from any animal slaughtered at the abattoir shall be inspected. Part VII and Schedule 2 of the Standing Regulations of Act 87 of 1967 specifies the procedures for the routine primary post mortem inspection of slaughter animals. With regard to establishing whether a pig carcass is arthritic or not the following aspects are considered during the primary inspection: any swelling or enlargement of the joints; the *Lnn. iliaci mediales et laterales* on both sides are examined for any abnormalities which would indicate hindleg involvement. Once arthritis has been diagnosed, Schedule 4 of the Standing Regulations specifies that arthritis in a carcass can either command total condemnation or partial condemnation with conditional passing of the rest of the carcass[7]. The secondary examination and evaluation of an arthritic carcass is based upon the severity, acuteness, generalisation, extent and systemic effects of the arthritis.

A noticeable omission is that the Standing Regulations of Act No 87 of 1967 do not stipulate that the lymph nodes draining the joints of the forelimb be examined at routine primary or secondary inspection.

DISCUSSION

Act 87 of 1967 requires that a monthly return of all the condemnations at an abattoir be submitted to the Chief Meat Hygiene Officer in the form of Schedule 8 of the Standing Regulations[7]. It was by means of this Schedule 8 that the high incidence of porcine arthritis at the Newtown abattoir first became apparent. This substantiates the importance of making accurate diagnoses and keeping strict records at an abattoir. A large abattoir can be regarded as the "post-mortem hall" of a country's livestock population and Schedule 8 can serve as a useful barometer with regard to the diseases and conditions that occur frequently in slaughter animals. It also gives an indication of those diseases and conditions which may be of particular economic and public health significance.

From personal observation it appears that porcine carcasses have been condemned for arthritis at abattoirs on the basis of an incorrect diagnosis. Arthritis is the inflammation of the intra-articular components of a joint and should not be confused with unrelated conditions such as spirochaetal granulomas situated on or near joints or with agonal haemorrhage in the hip joint. The latter occurs when a stunned pig is shackled and hoisted by a hindleg, and the ligament of the head of the femur is torn with the concomitant rupture of associated blood vessels and haemorrhage into the joint cavity.

At both the Newtown and Heidelberg (Transvaal) abattoirs only a small percentage of pigs were observed or suspected at ante-mortem inspection to be suffering from arthritis. The majority of cases were noted or sus-

pected at the routine primary post-mortem inspection and were diagnosed and evaluated by a veterinarian at the secondary post-mortem inspection. All the cases in the above survey were first noted at routine primary post-mortem inspection due to the enlargement and inflammation of the *Lnn. iliaci mediales et laterales* together with visibly swollen joints on some occasions. On further examination pig carcasses were sometimes found to have polyarthritis, the forelegs also being involved. At no stage during this survey did the spinal column of an affected pig carcass show any signs of arthritis. As the lymph nodes draining the joints of the porcine forelimb are not routinely examined it can be assumed that where only the joints of the forelimb were arthritic, these particular carcasses passed the primary inspection and were considered as being fit for human consumption. The importance of lymph nodes in meat inspection and the knowledge of their drainage areas is well illustrated in the cases where the iliac lymph nodes indicated involvement without the presence of any noticeable external changes in their areas of drainage. In the majority of cases a more detailed examination revealed arthritis in one or more of the relevant joints. It is interesting to note that during the investigation carried out at the Eskort Bacon Co-op. abattoir, the incidence of arthritis increased from that of the previous year (Table 2). This was attributed to the survey bringing about an increased awareness of the iliac lymph nodes and their drainage areas during primary and secondary post-mortem inspection.

The joints of the porcine forelimb are drained by the *Lnn. axillares primae costae*. They are intimately associated with the cranial edge of the first rib on either side where the ribs articulate with the sternum[1,5]. In pig carcasses these lymph nodes can be easily observed and inspected after the sternum has been sawn through the midline if the dressing technique is correctly performed. During slaughter and dressing the sternum must be accurately sawn down the midline, the sticking wound should be in the midline and the excess blood should be washed from the carcass before inspection is carried out.

Arthritis in slaughter pigs is usually manifested as a chronic condition localised to the joint cavity and associated synovial membranes of the affected joint[12]. Only one or two joints may be arthritic or a polyarthritis may be present. In chronic cases the arthritogenic agents are only found in the synovial fluid and synovial membranes of the arthritic joints.

In addition to the economic implications of porcine arthritis a new dimension has now been reached with *E. rhusiopathiae* being established as the main arthritogenic agent in slaughter pigs in South Africa. The public health significance of this potential hazard is a reality and is aggravated by the present legislation governing meat inspection whereby the *Lnn. axillares primae costae* are not examined as a routine during the inspection of the pig carcass in South African abattoirs.

CONCLUSIONS

It is imperative that both the *Lnn. iliaci mediales et laterales* and the *Lnn. axillares primae costae* (Figs 3 and 4) be examined during the routine primary meat inspection of the porcine carcass so as to detect any arthritic joints which may otherwise go unnoticed. Whereas the iliac lymph nodes are routinely examined in South African abattoirs it is considered essential that the *Lnn. axillares primae costae* be included in the

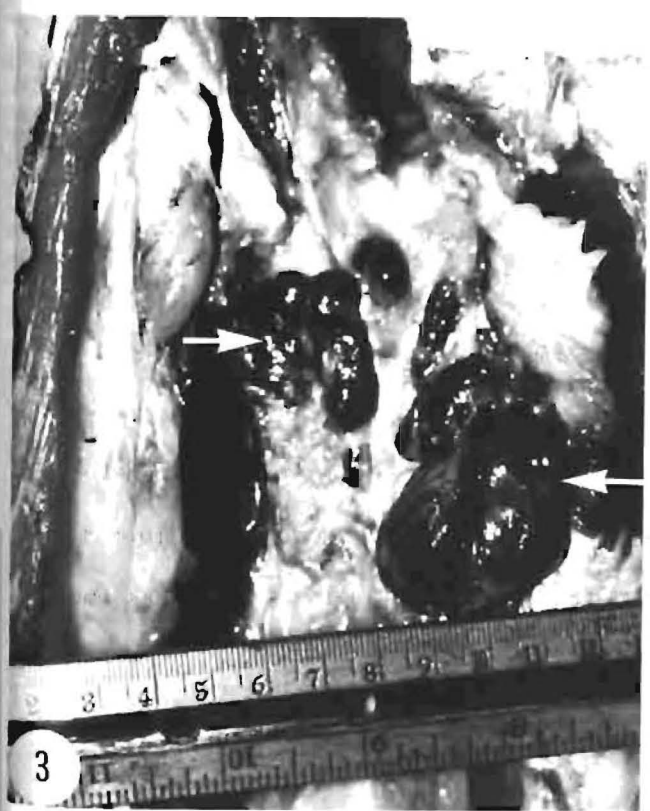

Fig. 3 Normal and swollen iliac lymph nodes.

Fig. 4 The right *Lnn. axillaris primae costae* (a) cranial to the first rib where it articulates with the sternum (B).

routine inspection procedures. Should the above mentioned lymph nodes show any sign of enlargement and inflammation with no obvious external changes in their drainage areas, then arthritis in one or more joints of the relevant limb commands a high suspicion index.

In order to establish whether arthritis is present the joint cavities should be opened in such a way as to prevent contamination of the carcass with any exudate. At secondary post-mortem inspection the diagnosis of arthritis and evaluation of the affected carcass should follow this proposed procedure: the carcass should be longitudinally sawn in half through the vertebral column which should be examined for any signs of spondylitis. The limb or limbs related to the affected lymph node or lymph nodes should be detached from the carcass without opening the hip or shoulder joints and thus prevent any possible contamination of the carcass. The limb or limbs should then be placed horizontally on a clean table. Starting at the distal end, the joints of the limb in question should be opened. When arthritic joints are located a pathological anatomical diagnosis should be made so as to ascertain the chronicity of the lesions.

The carcass with one or two localised chronic arthritic joints and no systemic involvement should undergo a partial condemnation. The affected limb should be condemned and the remainder of the carcass should be passed as fit for human consumption[11].

Polyarthritis, including spondylitis, would require total condemnation due to the generalised nature of the lesions unless partial condemnation involving deboning can be effected.

Should the abattoir be attached to a deboning establishment under optimal veterinary supervision, a suitable carcass with polyarthritis could undergo partial condemnation of the affected joints. The arthritic joints together with some surrounding normal tissue should be removed and condemned. The remainder of the limbs and the carcass, after bacteriological clearance, can be admitted to the deboning area. Should the bacteriological results be unsatisfactory or should there be any doubt as to the safety of the apparently normal tissue, then the material in question could be passed conditional to heat sterilisation.

As is applicable to the secondary post-mortem inspection of any detained carcass, the accurate diagnosis and correct evaluation of an arthritic porcine carcass is essential if a high standard of meat inspection and meat hygiene is to be attained. This can only be performed by a suitably qualified veterinarian. It is evident that careful recording and analysis of Schedule 8 returns should continue to be made so as to utilize the information available to the best advantage of meat hygiene and the livestock industry in South Africa.

ACKNOWLEDGEMENTS

I wish to record my sincere appreciation to Dr B.G. Horton for his assistance in acquiring material for this investigation. My sincere thanks to Prof L.W. van den Heever for his guidance and to Mrs M. Erasmus and Miss L. Halland for their technical assistance.

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BOOK REVIEW **BOEKRESENSIE**

VETERINÄRMEDIZINISCHE PARASITOLOGIE

J.BOCH & R. SUPPERER, 2nd edition.

Verlag Paul Parey, Berlin, Hamburg 1977. pp. xi. 517. Fig. 160. Tabs. 20.

This excellent text book has been extensively revised. The illustrations are of the same high quality and many of the publications referred to are as recent as 1976. There is a general introduction which also deals with techniques used in Protozoology, Helminthology and Entomology. The parasites of ruminants, equines, swine, carnivores, birds and laboratory animals are dealt with in separate sections which are subdivided into Protozoa, Helminths, Pentastomids and Arthropods. Further subdivisions dealing with parasites or groups of parasites are followed by a complete list of references; in some instances, e.g. the coccidia of dogs and cats, 94 publications are listed.

I have no hesitation in recommending this book to both students and practicing parasitologists.

A.V.

EVALUATION OF NEUROLOGICAL EXAMINATION OF SHEEP

C.F.B. HOFMEYR

ABSTRACT: Hofmeyr C.F.B. The neurological examination of the sheep. *Journal South African Veterinary Association* (1978) **49** No. 1, 45–48 (En) Faculty of Veterinary Science, University of Pretoria, Box 12580, Onderstepoort 0110, Republic of South Africa.

One hundred clinically negative Merino sheep were examined neurologically in an attempt to establish normal responses. Dependable results were obtained regarding eye muscle integrity, corneal sensitivity, the optic papilla and righting reflexes and to some extent the knee jerk reflex. The elicitation of other reflexes was inconsistent and unreliable except the placing reflex as shown by raising and lowering the forequarters.

INTRODUCTION

During the course of a research investigation it became necessary to establish the normal responses of sheep to neurological examination. When this present study was undertaken, no evaluation of routine neurological procedures applied to sheep could be found.

MATERIALS AND METHODS

One hundred Merino ewes and wethers two to six tooth and all clinically negative were used. The various neurological techniques employed were as follows.

Cranial nerves:

N. olfactorius – no particular strongly smelling substances were used. A hand that handled other sheep was held in front of the nose to determine response.

N. opticus – sight of individual eyes was not tested. The response of the sheep to the approach of a handler was noted. An opthalmoscopic examination was then carried out.

N. oculomotorius – N. trochlearis – N. abducens – these three nerves were tested simultaneously by inducing eye movements through raising and lowering the head and rotating it clockwise and anti-clockwise (Figs. 1, 2 & 3).

N. trigeminus – function was established by assessing the sensitivity of the cornea and upper lip reflex on either side, as well as through touch and integrity of the masseter muscles (Fig. 4).

N. facialis – ear movement was observed as well as symmetry of the face.

N. vestibulocochlearis – assessment of hearing was inconclusive. The sense of equilibrium was tested when the head was rotated in various positions (Fig. 2) and having the sheep lie on its side and watching its ability to rise without difficulty.

N. glossopharyngeus – the pharynx was palpated and the swallowing reflex elicited. Sensitivity of the posterior part of the tongue was tested by touch after drawing the tongue out of the mouth.

N. vagus – this was assessed by pharyngeal palpation and absence of dysphagia as well as by pulse rate. This tends to rise in vagus malfunction.

N. accessorius – the muscles of the neck were examined for tone and the neck for its ability to move in a normal manner.

Dean: Faculty of Veterinary Science of the University of Pretoria PO Box 12580 Onderstepoort 0110 Republic of South Africa.

N. hypoglossus – as the motor nerve to the tongue, this was tested by holding the tongue to determine its ability to retract.

Reflexes:

Flexor reflex (Fig. 5). The area dorsal between the claws was pinched and the vigor of withdrawal of the limb noted.

Patellar reflex (Fig. 6). The sheep was held in a position where the hind limb was free. By means of a plexor the patellar ligament was tapped and the degree of response assessed.

Extensor thrust reflex (Fig. 7). The sheep was held upright with its back against the body of the helper and the thrust reflex of the hind limbs was tested by sudden pressure by the hand of the examiner alternatively against the toe and the heel of the foot. The forelimbs were tested similarly when the elbow was extended by one hand of the operator.

Crossed extensor reflex (Fig. 8). By palpating the muscle mass of each limb the examiner established the muscle tone with the head of the sheep in the following positions:

Raised head
Flexed head
Head rotated clockwise

Righting reflex (Fig. 9). The sheep was held up by the pelvis as high as the assistant's chest and the position of the head and fore feet observed. The sheep was placed in turn on its left and right sides and its ability noted to return to a sternal position.

Placing reactions:

Extensor postural thrust (also see Fig. 7). The animal was held by the axillae and lowered until its hindlegs touched the floor or other solid object to see whether the limbs extended on contact.

Forequarters raised and lowered (Fig. 11). The sheep was lifted by the head and suddenly lowered to note the extension of the forelegs on contact with the ground.

Hopping reactions (Fig. 12). With the sheep held in a horizontal position three legs were kept flexed while the sheep was lowered suddenly on the remaining leg, to determine whether it would extend. This was done with each leg in turn.

Placing reflex (Fig. 10). (Head up, feet in contact with the table). In this examination the sheep was held horizontally and moved forward so that its fore feet touched the edge of a table in order to establish whether they would support the body.

RESULTS

The results are summarised by giving the various findings as percentages of the total number of sheep reacting in a particular manner to a certain stimulus. To denote the extent of a response the following symbols are used:

No response	– 0
Slight response	– X
Medium response	– XX
Very marked response	– XXX

The response of the knee jerk was assessed by estimation.

N. olifactorius – results indefinite

N. opticus.
Ophthalmoscopic examination of the optic disc.

Shape of optic disc*	Percentage	
	Left eye	Right eye
Kidney shaped	65	61
Oval	28	30
Irregular	2	3
Round	1	1
Halfmoon	4	3
Sickle shaped	2	2
	100	100

*All were well defined.

N. oculomotorius. N. trochlearis. N. abducens.
Assesment of the integrity of the innervation of the eye muscles.

Position of head	Position of eyes turned	Percentage
Head raised (Fig 1)	Eyes turned ventrally	100
Head flexed (Fig 2)	Eyes turned dorsally	100
Head rotated clockwise (Fig 3)	Left eye turned dorsally	100
	Right eye turned ventrally	100
Head rotated anti-clockwise	Left eye turned ventrally	100
	Right eye turned dorsally	100

Reflex		Percentage			
(Fig 4) Upper lip response *N. trigeminus* Sensitivity of the cornea		Left half 0 – 1 X – 30 XX – 54 XXX – 15 XXX – 100	Right half 0 – 1 X – 30 XX – 54 XXX – 15 XXX – 100		
Fig 5) Flexor reflex		Left foreleg 0 – 87 X – 7 XX – 4 XXX – 1	Right foreleg 0 – 76 X – 14 XX – 5 XXX – 4	Left hindleg 0 – 80 X – 9 XX – 2 XXX – 9	Right hindleg 0 – 79 X – 10 XX – 3 XXX – 8
(Fig 6) Knee jerk Degree of deviation from resting state	0° 1° 2° 3° 4° 5° 6° 7° 8° 9° 10°			0 1 17 19 20 19 16 5 0 1 2	0 2 19 17 17 19 18 5 0 1 2
(Fig 7) Extensor thrust		Heel 0 – 88 X – 9 XX – 2 XXX – 1 Toe 0 – 36 X – 28 XX – 22 XXX – 14	Heel 0 – 89 X – 8 XX – 2 XXX – 1 Toe 0 – 35 X – 28 XX – 23 XXX – 14	Heel 0 – 2 X – 7 XX – 14 XXX – 77 Toe 0 – 0 X – 2 XX – 18 XXX – 80	Heel 0 – 2 X – 8 XX – 13 XXX – 77 Toe 0 – 0 X – 2 XX – 17 XXX – 80

Fig. 1 Head raised
Fig. 2 Head rotated
Fig. 3 Head flexed
Fig. 4 Sensitivity upper lip
Fig. 5 Flexor reflex
Fig. 6 Patellar reflex
Fig. 7 Extensor thrust reflex
Fig. 8 Crossed extensor reflex
Fig. 9 Righting reflex
Fig. 10 Placing reflex
Fig. 11 Forequarters raised and lowered
Fig. 12 Hopping reactions

Reflex	Percentage			
(Fig 8)	Left foreleg	Right foreleg	Left hindleg	Right hindleg
Crossed extensor (Raised head)	0 – 24	0 – 24	0 – 34	0 – 34
	X – 50	X – 50	X – 62	X – 62
	XX – 22	XX – 22	XX – 64	XX – 64
	XXX – 3	XXX – 3	XXX – 0	XXX – 0
Extensor thrust (Flexed head)	0 – 80	0 – 80	0 – 81	0 – 80
	X – 19	X – 19	X – 18	X – 19
	XX – 1	XX – 1	XX – 1	XX – 1
	XXX – 0	XXX – 0	XXX – 0	XXX – 0
Extensor thrust (head rotated anti-clockwise)	0 – 7	0 – 7	0 – 100	0 – 100
	X – 47	X – 76		
	XX – 40	XX – 16		
	XXX – 7	XXX – 1		
Crossed extensor (Head rotated clockwise)	0 – 7	0 – 7	0 – 100	0 – 100
	X – 76	X – 46		
	XX – 16	XX – 39		
	XXX – 1	XXX – 8		
			Sheep placed	
(Fig 9)		upside down	on left side	on right side
Righting reflexes*		XXX – 100	XXX – 100	XXX – 100
(Fig 10)				
Placing reactions				
Placing reflex (Head up, feet contacting table)	Inconclusive 100			
(Fig 11)				
Forequarters raised and lowered	XXX – 100			
(Fig 12)	Left foreleg	Right foreleg	Left hindleg	Right hindleg
Hopping reactions	0 – 0	0 – 0	0 – 0	0 – 0
	X – 2	X – 2	X – 2	X – 2
	XX – 8	XX – 8	XX – 8	XX – 8
	XXX – 90	XXX – 90	XXX – 90	XXX – 90

*All were well defined.

DISCUSSION

It is apparent that the Merino sheep in general is a very disappointing subject for neurological examination by the usual methods available to the clinician.

1 The optic papillae, although variable in shape, were all well-defined, therefore a choked disc would be easily recognisable.
2 The rotation of the eyes after changing the position of the head was likewise constant in normal sheep and indicated the integrity of *N. oculomotorius*, *N. trochlearis* and *N. abducens*.
3 The upper lip response was too unreliable to recognise deviations from normal. The sensitivity of the cornea was however always present as a gauge of the sensory complement of *N. trigeminus*.
4 The flexor reflex was only intermittently present.
5 The knee jerk was present in all cases examined but varied tremendously in degree of response. The absence of the knee jerk will therefore most likely have clinical significance but the degree probably has no meaning.
6 The extensor thrust showed a great variety of responses and proved to be clinically unreliable. The same applies to the crossed extensor reflex in all its stages of examination.
7 The righting reflexes proved to be one hundred percent dependable as were the hopping reactions. The placing reflex as shown by raising and lowering the forequarters was strongly present in all cases but this reflex was virtually absent when the sheep was held in the longitudinal position and its forefeet were brought into contact with a table.

In conclusion it can be stated that with the clinically normal Merino sheep, presenting such unsatisfactory results at ordinary neurological examination, the clinician would be very severely hampered in making a proper diagnosis in many kinds of pathology of the central nervous system in sheep.

BIOCHEMICAL MECHANISMS CAUSING TICK RESISTANCE

R.G. WILSON

ABSTRACT: Wilson R.G. Biochemical mechanisms causing tick resistance. *Journal of the South African Veterinary Association* Vol **49** No. 1, 49–51 (En) Wellcome Research Labs., Berkhamsted Hill, Berkhamsted, Hertfordshire, England.

Three general biochemical mechanisms can cause ticks to be resistant to acaricides: an alteration in the properties of the site of action, a change in the rate of transportation and a change in the rate of metabolism. The first mechanism enables increased concentrations of acaricide at the site of action to be tolerated, while the last two mechanisms result in a decreased concentration and/or persistence of the acaricide at the site of action. These mechanisms are discussed in detail with reference to a range of acaricides, particular emphasis being given to blue tick strains occurring in Southern Africa.

HOW RESISTANCE OCCURS

Most forms of life show a remarkable ability to adapt to a changing environment in such a way as to ensure the survival of their species. The cattle or blue tick is no exception. A single engorged female *Boophilus* tick can lay about 2 000 eggs. This overproduction of offspring leads to competition among individuals for survival. Exposure to an ixodicide applies a selection pressure to the ticks. Those whose genes determine that they can withstand the ixodicide will survive and reproduce, those that cannot will perish. After a number of generations one or more resistance-conferring genes which existed in the original susceptible population only at a very low frequency, will be apparent in the majority of individuals in a population. This is the process of the development of resistance – a visible example of microevolution.

BIOCHEMICAL MECHANISMS OF RESISTANCE

Resistance to ixodicides may be caused by many different biochemical mechanisms which can, however, be divided into three broad categories: an alteration in the properties of the site of action of the ixodicide, a change in its rate of transportation and a change in its rate of metabolism. The first mechanism enables increased concentrations of ixodicide at the site of action to be tolerated, while the latter two mechanisms result in a decreased concentration and/or persistence of the ixodicide at the site of action. The mechanisms are outlined in Figure 1. A change in the rate of transportation of ixodicide has not yet been observed in resistant ticks although the number of cases investigated is small. Small changes in transportation rate, particularly the penetration rate through the cuticle, can greatly enhance resistance factors when combined with a degradative mechanism. This has been shown in houseflies[10]. The mechanisms of altered rate of metabolism or altered site of action have been observed with a variety of ixodicides and these will be considered in turn. The literature covering tick resistance has been comprehensively reviewed recently[3].

Paper read at the Biennial Congress of the SAVA, Grahamstown, August 1977.

Arsenicals

In South Africa *Boophilus decoloratus* did not develop resistance to arsenical dips until after nearly 40 y use in the field. The arsenicals act by combining with sulphydryl groups which form part of the catalytic centre of many enzymes. Whitehead[12] showed that the levels of free sulphydryl groups were greater in a resistant strain than a susceptible strain. These surplus nonessential sulphydryl groups are able to "mop-up" the arsenic, rendering it ineffective and thus reducing the concentration of acaricide available at the site of activity.

Chlorinated Hydrocarbons

Included in this class of ixodicide are DDT, BHC and the cyclodienes. Although they are all regarded as neurotoxicants, virtually nothing is known about their biochemical mode of action. Hence evidence on the mechanisms of resistance to these compounds is mainly negative.

Resistance to BHC and the cyclodienes occurred within the very short time of 18 m after their introduction to control arsenic resistant ticks in South Africa and Australia. Resistance to BHC is conferred by a single dominant gene and this accounts for its rapid development. There is cross resistance between BHC and all the cyclodienes. Resistance to dieldrin and BHC is not due to a change in their rate of penetration or metabolism but is generally thought to be due to an alteration in the site of action, allowing increased concentrations of the ixodicide to be tolerated.

Resistance to DDT developed more slowly, occurring in *B. decoloratus* in South Africa and *B. microplus* in Australia, some 5 y after field use started. This slower development corresponds with the different genetic mechanism involved, a single incompletely recessive gene. Experiments using radiolabelled DDT have shown that penetration and metabolism rates are similar for both resistant and sensitive strains of ticks. The implications are that resistance is caused by an altered site of action and this is supported by the fact that there is cross resistance to pyrethrum[11] and the synthetic pyrethroids[9].

Organophosphates and carbamates

In contrast to the organochlorines, the biochemical mode of action and the mechanisms of resistance to the

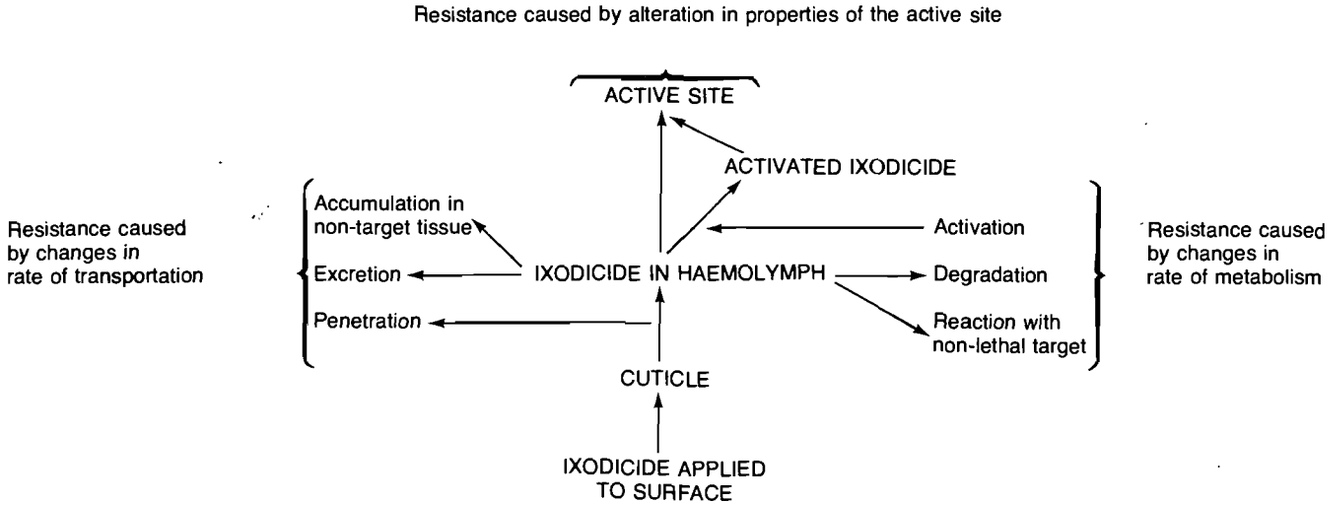

Fig. 1 Biochemical Mechanisms causing tick resistance.

organophosphates and carbamates have been studied in detail. These compounds act by inhibiting acetylcholinesterase. This enzyme is responsible for the breakdown of acetylcholine, the chemical transmitter involved at a large number of nerve junctions in both vertebrate and invertebrate nervous systems. Thus a nerve impulse on passing along a nerve axon to the axon terminal which makes a junction, or synapse, with another nerve cell, causes the release of acetylcholine which then combines with receptor sites on the post synaptic membrane causing the receptor cell to initiate an impulse. Acetylcholinesterase destroys the acetylcholine by hydrolysis so that stimulation of the receptor ceases and the synapse is able to transmit a new impulse again. In the presence of an organophosphate the acetylcholinesterase is inhibited and is unable to destroy the acetylcholine which consequently stimulates the receptor cell, resulting in death of the organism. Workers at The Wellcome Research Laboratories were the first to show that the mechanism of resistance of *B. microplus* in Australia[7] and *B. decoloratus* in South Africa[5] and Rhodesia[8] was due to a decrease in the sensitivity of the acetylcholinesterase to inhibition by organophosphates. Thus a higher concentration of acaricide at the site of action could be tolerated.

Certain organophosphates kill resistant ticks more efficiently than susceptible ticks and work is currently in progress at our laboratories to determine if this negative correlation extends to their inhibition of acetylcholinesterase from resistant and susceptible strains. More recently other mechanisms of resistance have appeared. In the Mackay strain of *B. microplus,* organophosphate compounds are degraded more rapidly than in susceptible strains; this results in a decreased concentration and persistence of the ixodicide at the site of activity. In the Mt. Alford and Grassmere strains of *B. microplus* both types of mechanism, occur together.

Most organophosphates require *in vivo* conversion to the corresponding oxygen analogue to be effective as inhibitors of acetylcholinesterase. Selection pressure with Dursban in Australian laboratories has produced a resistant strain of *B. microplus* which carries out this conversion to the oxygen analogue much more slowly than susceptible ticks.

The resistance mechanisms towards the carbamates parallel those of the organophosphates.

Pyrethrum and Synthetic Pyrethroids

Although pyrethrum and the new synthetic pyrethroids have not been used widely in the field, resistance to pyrethrum has been found both in South Africa and in Australia and more recently, resistance to the new synthetic pyrethroids has been reported from Australia[9]. In both cases this resistance was associated with a cross resistance to DDT and is suggestive of a mechanism involving an altered site of action enabling higher concentrations of the pyrethrins to be tolerated at the site of action.

At our laboratories we are particularly interested in the new synthetic pyrethroids because of their good pesticidal properties, their extremely low mammalian toxicity and their excellent stability towards photodegradation and oxidation, coupled with a rapid biodegradability. We have found that although ticks can degrade one synthetic pyrethroid, permethrin, quite rapidly, its persistence can be substantially prolonged and this could make it a viable ixodicide.

Other Ixodicides

Chlordimeform, a formamidine derivative, amitraz, a triazapentadiene[4] and clenpyrin, a cyclic amidine[2], are recently intoduced ixodicides. The first two compounds are neurotoxins but the detailed mechanism of action of all of them is unknown; it certainly differs from that of the ixodicides previously mentioned. It will be interesting to see when, where and how resistance occurs.

THE FUTURE

We have seen that ticks have developed resistance to almost all the major classes of ixodicides. There seems no reason to suppose this trend will not continue. The compounds to which resistance is not apparent probably provide only a temporary solution. It is important that the research effort to produce new ixodicides is

maintained. The mechanism of action of existing ixodicides and the mechanism of resistance to them must continue to be studied so that the best use of the ixodicides can be determined on a rational basis.

Knowledge of the biochemical mechanisms of ticks is gaining impetus and a rational search for new ixodicides for dealing with specific resistant ticks is proving worthwhile. Drugs used in the medical field are being tested for activity against ticks and in some cases are providing interesting "leads". For example it has been found that acetylcholine agonists such as oxotremorine and its analogues are excellent ixodicides[1]. Such "leads", active at, until now, unexploited sites of action, may provide a starting point for the rational design of a new ixodicide. At our laboratories it has recently been found that the saliva of *B. microplus* contains high concentrations of Prostaglandin E^2 which may be responsible for the formation of the initial lesion in the skin of the cattle host[6]. A compound which would interfere with salivation or the contents of the saliva could well provide a useful ixodicide. There is a considerable medical interest in the above classes of drugs suggesting that many more resources could be combined and made available for the control of cattle ticks.

CONCLUSIONS

Ixodicides have acted as strong forces which select those genes responsible for increased tolerance whilst eliminating those genes controlling susceptibility of the tick. This has resulted in resistance to all the major classes of ixodicides. The detailed biochemical mechanisms observed so far are very varied and the possibilities seem almost limitless. To minimise the problems of resistance combined field, biochemical and genetic studies must be made so that by the application of rational principles the best possible use can be made of existing ixodicides and the new ixodicides as they arise.

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INFORMATION

INLIGTING

STRAIGHTENING CROOKED CALVES

A program that requires that cows between the 40th and 70th days of pregnancy be prevented from grazing either young lupines or lupine in the mature seed stage can all but eliminate crooked calf disease.

Crooked calf disease is a serious economic problem for ranchers in the eleven western States. An average of two percent of all calves born each year where lupine is a problem suffer from the disease. Last year nearly 10 000 calves were born deformed.

The disease is characterised by twisted or bowed limbs or spine, twisted neck, cleft palate or a combination of any of these deformities. It occurs when cows eat certain species of lupine during the critical period of pregnancy.

Agricultural Research Service chemist Richard F. Keeler and ARS animal scientist Lynn F. James, with co-operation from Utah State University veterinarians James L. Shupe and Kent R. Van Kampen, all at Logan, Utah, Poisonous Plant Research Laboratory (1159 E. 14th N., Logan, Utah 84322), have identified the chemical compound in lupine that produces the deformities.

In studying the compound, which is the alkaloid, anagyrine, the researchers learned that whether or not a calf suffers damage depends on *how much* anagyrine the cow consumes and *when* she consumes it.

By controlling when pregnant cows graze lupine, the cases of crooked calf disease can be minimised. Other practices to further help the situation include regulating cattle breeding periods and reducing the abundance of lupine on the range.

Many ranchers have tried mineral supplementation to reduce incidences of crooked calf disease. The ARS study found no evidence to support this practice.

("Straightening Crooked Calves"; Agricultural Research, September 1977, Agricultural Research Service, U.S. Department of Agriculture, Washington D.C. 20250).

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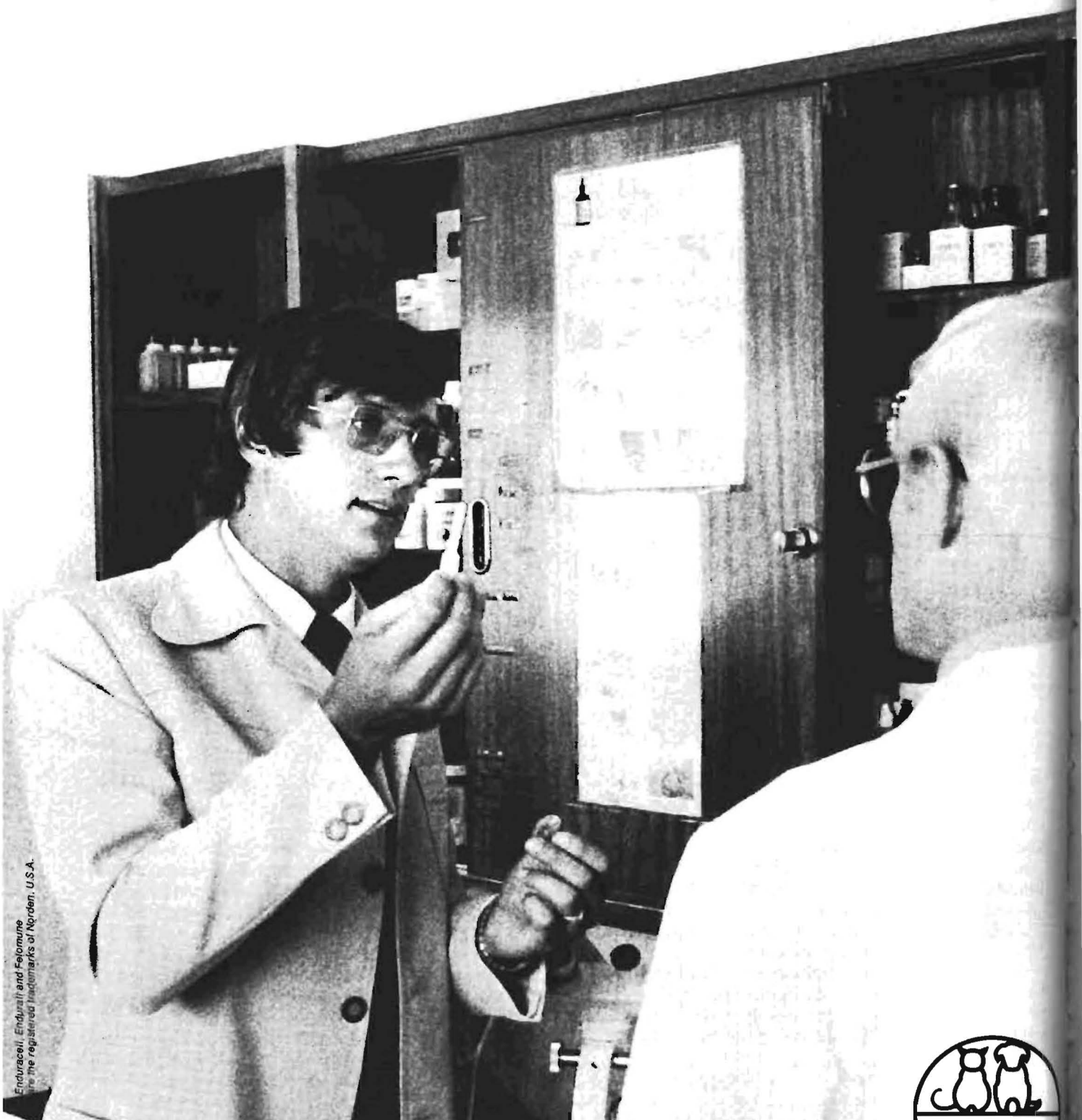

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CONGRESS PAPER **KONGRES REFERAAT**

VIRUS DIAREE IN KALWERS IN SUID-AFRIKA*

L. PROZESKY & A. THEODORIDIS

ABSTRACT: Prozesky L. & Theodoridis A. *Virus diarrhoea of calves in South Africa.* Journal of the South African Veterinary Association (1977) **49** No. 1, 53–56 (Afr) Section Pathology & Virology, Vet. Res. Inst., 0110 Onderstepoort, Rep. of South Africa.

The importance of diarrhoea in calves is briefly discussed. The role of viruses as primary invaders of the intestinal epitheium is stressed. The pathogenesis and problems encountered in diagnosing virus associated diarrhoea as well as the morphoogy of rota and coranavirus are discussed. Possible methods of prevention and treatment are briefly mentioned.

INLEIDING

Diaree was nog altyd en bly steeds die grootste probleem geassosieer met die grootmaak van kalwers onder intensiewe en in 'n mindere mate onder ekstensiewe toestande[19]. Syfers in 1972 in die V.S.A. vrygestel, toon dat ten spyte van goeie bestuurspraktyk 10% van alle jong kalwers 'n diaree ontwikkel. Mortaliteit a.g.v. diaree was 18% onder melkraskalwers en 8% onder kalwers van vleisrasse. Die berekende verlies was 250 miljoen dollar[13]. 'n Faktor wat telkens buite rekening gelaat word by die bepaling van verliese geassosieer met diaree is die verlies a.g.v. swak groei en sekondêre infeksies gedurende die herstelfase.

Diaree in kalwers moet as 'n komplekse sindroom gesien word en klinies waarneembare diaree is slegs die resultaat van die tussenwerking van verskeie faktore, alhoewel 'n groot aantal etiologiese faktore (infeksieus sowel as nie-infeksieus) diaree in kalwers kan veroorsaak, is die kliniese beeld in meeste gevalle dieselfde[7]. 'n Etiologiese diagnose vanaf die kliniese beeld alleen is in die meeste gevalle nie moontlik nie en kan alleen deur laboratoriumtegnieke vasgestel word.

Dit is nie ongewoon om meer as een erkende patogene organisme van dieselfde kalf te isoleer nie. Soms is dit nodig om meer as een kalf in 'n kudde te ondersoek voordat 'n etiologiese diagnose gemaak kan word.

Etiologie van diaree in kalwers

Diaree in kalwers kan van infeksieuse of nie-infeksieuse oorsprong wees[7]. Slegs infeksieuse toestande sal hier bespreek word met die klem op die rol wat virusse hierin speel. Rota- en coronavirusse word as die belangrikste oorsaak van virus diaree in kalwers beskou[19], maar parvo-[17], herpes-[16] en adenovirusse[1] word ook soms vanuit die dermkanaal geïsoleer, terwyl *Salmonella* en *E. coli* die belangrikste bakterieë is wat alleen of saam met virusse kan voorkom[19]. Coccidiose is die belangrikste protozoön wat diaree veroorsaak[7], terwyl sekere stamme van chlamydia as 'n primêre oorsaak van diaree beskou word[3]. Swamme, alhoewel sekondêr, word vanaf 'n klein aantal kalwers met diaree geïsoleer, veral waar hoë dosisse antibiotika oor 'n lang periode toegedien was[6].

*Referaat gelewer tydens jaarkongres van die Pretoria-tak SAVV 1977.

Rota (reo-agtige) virus

Weens die morfologiese verwantskap van rotavirus met reovirus was die term reo-agtige "reo-like" virus aanvanklik gebruik[15]. By nadere morfologiese en serologiese ondersoeke is egter vasgestel dat die betrokke virusse verskillend is en word die term rotavirus nou algemeen aanvaar[4].

Kenmerkend van rotavirusse is dat 2 morfologiese vorms in negatief gekleurde preparate waarneembaar is, nl. dubbelomhulde partikels, ± 73 nm. groot, en enkelomhulde partikels, ± 63 nm. groot (Fig. 1), met spykeragtige uitsteeksels op die buitenste oppervalkte. In albei vorms is 'n seskantige binneste struktuur soms waarneembaar[20].

Coronavirusse

Hulle wissel in grootte van 107–160 nm.[2] en word gekenmerk deur buisvormige uitsteeksels van die omhulsel wat elk 'n blaasvormige struktuur aan die punt het (Fig. 2). Hierdie blaasvormige struktuur dien om coronavirus te onderskei van morfologiese verwante virusse, soos myxo- en arenavirus[2].

PATOGENESE

Verspreiding

Tot dusver is materiaal afkomstig van slegs 10 kuddes waar diaree teenwoordig was, ondersoek. In 6 hiervan is rota- of coronavirus geïsoleer (in 2 kuddes was albei virusse teenwoordig). Distrikte waar 1 of albei van die virusse geïsoleer is, sluit in Pretoria, Standerton, Randburg, Vaalwater, Potgietersrus en Johannesburg.

Rotavirus kom hoofsaaklik voor in kalwers 1–3 d oud terwyl coronavirus meer algemeen voorkom in kalwers 5–8 d oud. Alhoewel jong kalwers meer vatbaar is vir die betrokke virusse en 'n hoër mortaliteit toon, is dit nie ongewoon om corona- of rotavirus uit kalwers wat reeds 'n paar maande oud is te isoleer nie.

Die verskillende bronne van besmetting is nog nie finaal vasgestel nie, maar die volgende mag 'n belangrike rol speel:

1) Kalwers met klinies waarneembare diaree[8].
2) Kalwers wat herstel het maar tog nog virusse vrystel[8 19].
3) Volwasse diere met of sonder 'n diaree[19].
4) Ander spesies soos skape en varke (nog nie finaal bewys nie) [19].

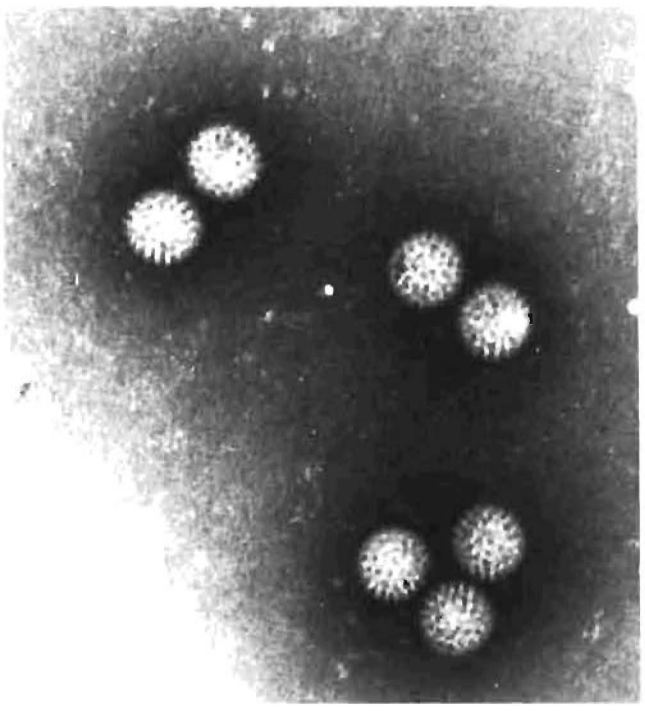

Fig. 1 'n Groep enkelomhulde virus partikels. x 210 000

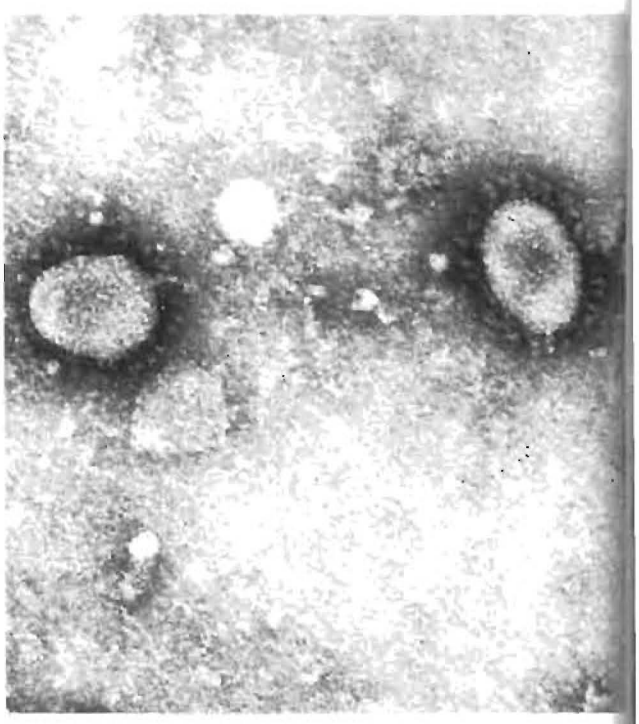

Fig. 2 Coronavirusse met die kenmerkende buisvormige uitsteeksels van die omhulsel. x 180 000

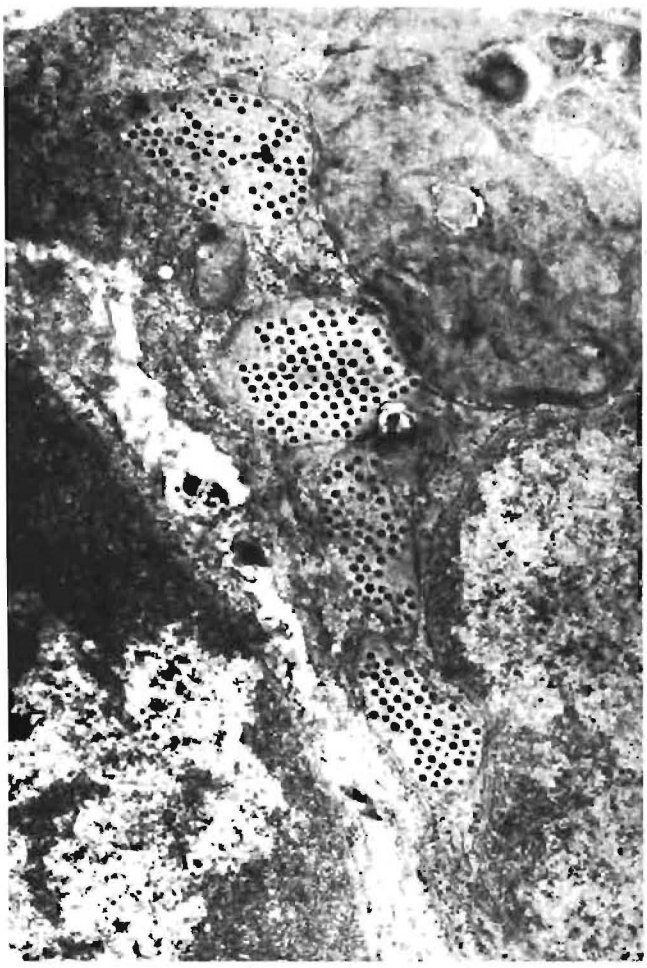

Fig. 3 Rotavirusse in die endoplasmiese retikulum van dunderm absorpsie-epiteel. x 18 000

5) Aangesien rota- en coronavirusse 'n redelike hoë weerstand teen omgewingstoestande toon, kan 'n area waar besmette diere gehuisves was vir 'n geruime tyd as 'n bron van besmetting dien[19].

Besmetting vind *per os* plaas en die virusse dring die absorpsie epiteel van die duodenum binne en vermenigvuldig daar[9] (Fig. 3). Hiervandaan versprei rotavirus na die res van die dunderm en coronavirus selfs verder na die kolon. Rotavirus veroorsaak die afstoting van besmette selle in die lumen van die dermkanaal, terwyl besmetting van coronavirus tot lise van die epiteel lei. Hierdie beskadiging van die epiteel vergemaklik bakteriële indringing en gevolglike sekondêre besmetting (Fig. 4). Veral *E. coli* speel hier 'n belangrike rol. Regenerasie van die epiteel vind plaas vanaf die basis van

Fig. 4 Dunderm villi van 'n kalf afgeneem met 'n aftaselektronmroskoop. Let op beskadiging veroorsaak deur rotavirus aan die puvan die villus.

die villi en aangesien weefselbeskadiging hier vinniger verloop as regenerasie word die fisiologiese-aktiewe silinderepiteel deur kubiese tot plat epiteel vervang. Sulke atrofiese villi met verhoogde sellulêre infiltrasie in die stroma is histopatologies waargeneem in kalwers 48–96 u na aanvang van die diaree[11].

Die gevolge van die beskadiging van die dermkanaalepiteel kan as volg opgesom word:

1. Die verlies van silinderepiteel lei tot minder effektiewe vertering en die aansameling van onverteerde voedingsreste in die dermkanaal.
2. Hierdie voedingsreste dien as kweekbodem vir bakterieë (veral *E. coli)* wat die moontlikheid van sekondêre besmetting deur die beskadigde epiteel verder verhoog.
3. Die voedingsreste en afgestote epiteel vorm 'n hipertoniese oplossing met gevolglike aansameling van groot hoeveelhede vloeistof in die dermkanaal. Saam met die toksienes deur die bakterieë vrygestel, lei dit tot verhoogde peristaltiese bewegings en 'n klinies waarneembare diaree.
4. Verminderde voedselinname, skok a.g.v. vloeistofverlies en sekondêre infeksie is almal faktore wat bydra tot die hoë mortaliteit in kalwers.

DIAGNOSE

Diagnose van virus diaree in kalwers berus op een of meer van die volgende metodes:

1. Elektronmikroskopiese ondersoek van mismonsters[5].
2. Immunofluoresensie van dermweefsel of mis in die geval van rotavirus. As gevolg van lise van selle besmet met coronavirus kan immunofluoresensie van mismateriaal nie as 'n diagnostiese metode gebruik word nie[11].
3. Kweking van virus[18].
4. Serologiese toetse op kuddebasis[19].

Voordat 'n diagnose van virus diaree gemaak kan word, moet 'n paar belangrike aspekte in gedagte gehou word. Slegs mismonsters van kalwers in die vroeë stadium van diaree is geskik vir ondersoek, aangesien die betrokke virusse 'n kort periode na besmetting vanaf die dermkanaal vrygestel word. Aangesien dit nie altyd moontlik is om te bepaal wanneer 'n kalf 'n diaree begin ontwikkel nie, moet monsters van ten minste 3 kalwers vir ondersoek gestuur word. Hoewel die virusse redelik bestand is teen ongunstige omgewingstoestande[19], moet maatreëls getref word om die monsters koel te hou. Omdat dit so algemeen is om meer as een patogene organisme uit 'n kalf met diaree te isoleer, is dit essensieël dat 'n volledige bakteriële ondersoek gedoen word en hiervoor is 'n lewendige, onbehandelde kalf in die vroeë stadium van diaree nodig.

Die isolasie van chlamydia uit die dermkanaal van kalwers met diaree skep probleme. Dit is bekend dat kalwers draers van chlamydia kan wees en dat die dermkanaal 'n belangrike storingsorgaan van die organismes is[3 14]. Ook is bekend dat sekere stamme van chlamydia 'n diaree in kalwers kan veroorsaak. Indien chlamydia en rota- of coronavirus vanaf die dermkanaal van 'n kalf met diaree geïsoleer word, is dit moeilik om die rol deur elk gespeel te bepaal.

BEHEER

'n Geattenueerde entstof wat beide rota- en coronavirus bevat, is oorsee beskikbaar[19]. Die betrokke entstof word direk na geboorte *per os* toegedien. Blootstelling van die dermepiteel aan geattenueerde virusse is skynbaar van meer belang vir die beskerming van die kalf as sirkulerende teenliggame. Kolostrum speel egter ook 'n belangrike rol in die beskerming van die kalf. Daar is gevind dat kalwers van volwasse koeie minder vatbaar is vir die betrokke virusse as kalwers van verse wat die eerste keer kalf[19]. Die moontlikheid om dragtige koeie te immuniseer word tans ondersoek[19].

BEHANDELING

Aangesien onverteerde voedselreste as 'n belangrike kweekbodem vir bakteriële vermenigvuldiging dien, is dit noodsaaklik om geen melk aan 'n kalf met diaree te gee nie. Weens die groot vloeistofverlies is dit noodsaaklik dat genoegsame vloeistof ten alle tye beskikbaar moet wees. die asidose wat ontstaan a.g.v. die verlies aan bikarbonaat-ione. die aansameling van melksuur in die spiere weens die bikarbonaat tekort en verminderde suurstofvoorsiening aan die spiere a.g.v. verlaagde bloeddruk en periferele sametrekking van die bloedvate moet geneutraliseer word.

Die toediening van binne-aarsevloeistowwe, veral in gevorderde stadia van ontwatering, lewer die beste resultate. Die oormatige toediening van dextrose moet egter voorkom word, aangesien dit as 'n diuretikum kan funksioneer. 'n Eenvoudige oplossing wat aan kalwers *per os* toegedien kan word, is die volgende: 10 g natriumkarbonaat, 5 g natriumchloried, 500 g dextrose gemeng met 5 l water. Die hoeveelheid vloeistof toegedien, hang van die graad van ontwatering af. 'n Kalf van 45 kg met 10% verlies aan liggaamsgewig behoort 6 l vloeistof per dag te ontvang. Nadat die beskadigde dermepiteel herstel het, kan melk weer geleidelik aan die kalf gegee word. Antibiotika *per os* en sistemies is noodsaaklik om sekondêre en soms primêre bakteriële infeksies te beheer[10].

BEDANKING

Mnr. A.M. du Bruyn en personeel van die seksie Fotografie word hartlik bedank. 'n Spesiale woord van dank aan Mnr. A.M. Spickett van die seksie Entomologie vir sy bydrae met die neem van skandeer mikrotofo's.

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Table 1: DISEASES AND PARASITES IN 1 009 EELS

Organism	Group	Disease	Affected Host Organism
1. *Aeromonas hydrophila*	Bacteria	Septicaemia	Systemic
2. *Pseudomonas putida*	Bacteria	Septicaemia	Systemic
3. *Flexibacter columnaris*	Bacteria	Columnaris	Skin & muscles
4. *Ichthyophthirius multifiliis*	Protozoa	White Spot	Skin & gills
5. Microsporidian *(Plistophora* sp.?)	Protozoa	Plistophoriasis	Muscles & serosa
6. *Myxidium giardi*	Protozoa	Myxoporidiasis	Gills
7. *Trichodina* sp. *(anguillae?)*	Protozoa	Trichodinasis	Skin
8. Nematode *(Paraquimperia* sp.)	Nematoda	—	Intestine
9. Metacercariae of avian trematodes	Trematode	—	Body cavity
10. Caligids	Copepoda	—	Buccal & branchial
11. —	Mineral	Calcium imbalance	Skeleton

stock which is certified disease free should be imported. As this is often impractical, imported fishes should, in any case, be held in quarantine while infections can be identified and, as is entirely possible, cured.

To turn to our eels, a stock assessment and aquaculture study of them is currently being undertaken at the J L B Smith Institute of Ichthyology at Rhodes University. In early 1977 the Institute employed an expert fish parasitologist, Dr P M Hine of New Zealand as a Research Fellow to undertake a survey of the diseases of young South African elvers. Dr Hine remarked upon the comparatively healthy state of our eels at present, compared with those which he has examined from Australasia and the Far East. Table 1 lists diseases and parasites found in a sample of 1009 live eels, of which 89% were juvenile "glass eels" and elvers and 11% were adult eels. The sample was made up of 880 specimens captured in the wild and 129 which had been kept in culture for some time.

The parasites in eels caught in two major rivers of our area, the Keiskama and Umtata, is given in Table 2. The comparatively low prevalence can be seen. The most serious, that of the ubiquitous "white spot" or "Ick" caused by the protozoan *Ichthyophthirius multifilis* is probably the commonest world wide pathogen of fish. It is primarily a problem of very young fish. These lack the ability to resist infection but eels above 10 g in weight appear to become immunocompetent and therefore, except in superinfections, it is seldom a problem in fish of greater mass.

Table 2: THE PERCENTAGE PREVALENCE OF PARASITES IN IMMATURE EELS CAUGHT IN THE KEISKAMA AND UMTATA RIVERS

	Glass Eels		Elvers	
Parasite	Keiskama %	Umtata %	Keiskama %	Umtata %
I. multifiliis	12,8	Nil	22,9	Nil
Plistophora sp.	6,8	9,5	2,9	Nil
Caligid	0,3	Nil	15,7	2,8
Paraquimperia sp.	Nil	Nil	Nil	2,8
Avian trematode	Nil	Nil	1,4	8,3

This underlines what has been previously said. At the present time fish farming is relatively new and our fish are comparatively healthy. Let us keep them that way. Since most pathogens are spread by the water with which fish are surrounded and intermediate hosts such as copepods or water birds live in or near it, a disease can spread through cultivated fish with wild fire rapidity. Prophylaxis rather than cure should be the aim. Most fish diseases are curable but their prevention, through constant vigilance, is much more desirable. Quarantining of imported fish is one important measure but care should be taken that no pathogens are introduced with any other imported article. Containers used to send fish away, for example, should on their return be sterilized. Another possible source of introduction of foreign infection is foodstuffs imported for use in the aquaculture industry.

The other extremely important aspect of prevention of disease is early and rapid diagnosis. Here the practising veterinary surgeon has a key role to play in any aquaculture operation. Correct and quick diagnosis first and the availability of preventive drugs to apply immediately thereafter, go a long way towards a healthy farm fish population.

The public health aspect of diseases which fish can transmit to humans can be a significant factor in many parts of the world though it is not likely, in the immediate future, to assume much importance in South Africa. Jackson[3] has listed a number of diseases transmissable by the eating of fish. Fish tissues and mucus can become infected with pathogens of alimentary canal diseases such as paratyphoid fever and bacillary dysentery. There is evidence that some fish can maintain cholera endemicity. As seen above, some fish are hosts of helminth worms, transmitted by ingestion to humans; such as the broad fish tapeworm *Dibothriocephalus latus* of Europe and the oriental liver fluke *Clonorchis sinensis*. Swimming pool granuloma or pseudotuberculosis is caused by the bacterium *Mycobacterium marinum* sometimes infecting fish and can be transmitted by entry through a small cut or injury while handling.

Nearly all such diseases are intestinal, the fish becoming contaminated through contact with human sewage and with human infection occurring by eating the uncooked fish. Both the introduction of raw, untreated sewage into farm ponds and the eating of raw or partly cooked fresh fish should be avoided. Cases of fish-transmitted diseases are rare in South Africa because here both these conditions are usually met. As the country becomes ever more cosmopolitan, however, outbreaks may in future occur among communities where raw fish may form an ingredient of a traditional dish. However, freshwater fish should never be eaten raw; about 15 minutes cooking at a temperature exceeding that of boiling water, at least, is sufficient to destroy any parasite the fish might harbour.

In conclusion, I should like to stress the need for ve-

terinary interest in aquaculture. Fish used as farm stock in this way will increase year by year. We are on a sound footing at present with a range of comparatively few pathogenic organisms compared with those of other countries. Our prophylactic measures carefully enforced will greatly reduce the chances of undesirable introductions, while timely diagnosis and curative measures promptly applied will minimise disease establishment and epidemics. It must be emphasized, however, that because the intensive cultivation of fish is so new in South Africa, we still have much to learn. Our knowledge in many aspects of this new branch of agriculture is still inadequate. Further research into the parasitology and epidemiology of fishes is badly needed in South Africa, as is also education and the spreading of knowledge in this new field among farmer and professional alike.

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JOURNAL REVIEW — TYDSKRIFRESENSIE

VETERINARY SCIENCE COMMUNICATIONS

Elsevier Scientific Publishing Company, Amsterdam
Publ. Price Dfl 139.00 p.a.

New journals are regularly appearing on the scientific scene to increase the burden on the veterinary scientist or clinician who tries somewhat helplessly to keep abreast with the relentless increase in knowledge. "Veterinary Science Communications" is, however, a new journal which is different from other journals because it offers, at least in part, a solution to the abovementioned dilemma.

The journal owes its origin and uniqueness to a marriage of two ideas. The one is the need established by Elsevier for a periodical that publishes brief scientific contributions with a high news value. These are referred to as "Letters", although they are cast into the customary layout for scientific articles. The second idea stems from the programme launched by a working group of the Commission of the European Communities on documentation and information in veterinary science for topical review articles on growing points in veterinary science. These to-the-point and concise "European Community Preview Articles" are intended to bridge the time gap between the appearance of the original scientific papers and the publication of classical review articles.

In order to expedite publication, the journal is produced directly from author-submitted camera-ready typescripts. This does not necessarily mean that articles will never be returned to authors for revision and retyping. With the necessary collaboration from contributors as regards the submission of error-free articles, delays should, however, be reduced to a minimum. A comprehensive guide to authors on the compilation and submission of articles is given in the first issue of the journal. The articles are printed on glossy paper and overall the journal gives a neat impression. The quality of the print is not uniform, but the method of production used makes this unavoidable.

The journal covers a wide field in pre-, para- and clinical veterinary science. It is therefore not only of value to the research scientist but also serves the clinician who wishes to remain abreast of current developments. Individual veterinarians may find the cost high but large practices and branches and groups of the veterinary association should find the investment worth their while.

RDB

CASE REPORT **GEVALVERSLAG**

CHONDROMA RODENS IN A DOG

A. LUCIA LANGE* and LEA STOGDALE**

ABSTRACT: Lange A. Lucia and Stogdale Lea. Chondroma rodens in a dog. *Journal of the South African Veterinary Association* (1978) **49** No. 1, 60–65 (En) Dept. Path., Fac. Vet. Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

The clinical and pathological characteristics of a chondroma rodens affecting the internal occipital protruberance, cerebellar meninges and the cerebellum of a dog, are described. The principle clinical findings were Horner's syndrome and ataxia. The histopathologic differentiation of this tumour from other fibro-osseous growths, is discussed.

INTRODUCTION

Chondroma rodens is a rare tumour which has been recorded in humans, dogs and cats. Jacobson[4] was the first author to describe this neoplasm. According to him it is "a tumour of intermediate aggressiveness in whose proliferation both spindle and cartilage cells play significant roles." He observed that, in humans, chondroma rodens usually affected the ribs, sternum, scapula and the shaft of metatarsal bones. Of the seven cases in dogs and one case in a cat so far described, only the skull bones were affected[14].

This case report describes a chondroma rodens in an 8 year old German Shepherd bitch, affecting the internal occipital bone with infiltration into the underlying meninges and cerebellum.

HISTORY AND CLINICAL FINDINGS

An 8-year-old German Shepherd bitch weighing 38 kg was referred to the Department of Medicine, Faculty of Veterinary Science, showing head tilt, ataxia, torticollis and myosis. Four months previously, the owners had noticed that the dog had developed a head tilt to the left and gradually became ataxic. Torticollis to the left and myosis of the left eye was observed 2 months later. During these 4 months daily treatment with dexamethasone tablets was prescribed. The corticosteroid therapy resulted in complete regression of the signs, which returned whenever treatment was stopped. She had remained bright and alert and had had a good appetite although her level of activity was decreased.

The clinical signs were principally confined to the locomotory and neurological systems. The dog was ataxic and tended to fall to the left. Her head was tilted to the left and there was hypermetria of the left fore leg but she did not circle (Fig. 1). Two days after admission she developed an intention tremor of the head and neck. When placed in lateral recumbency, the dog slowly righted herself but had difficulty in rising to her feet. All the spinal reflexes were normal. The left eye exhibited Horner's syndrome (Fig. 2): contraction of the pupil, enophthalmos and narrowing of the palpebral fissure due to ptosis of the upper eyelid and slight elevation of the lower eyelid. A thorough neurological examination of the eyes and the cranial nerves failed to reveal any additional abnormalities. No deformities of the skull bones could be palpated and otoscopic examination was normal. Other clinical findings of significance were the normal temperature, pain in both hip joints, increased inspiratory tone and her good condition.

Fig. 1 The patient had a consistent head tilt to the left and hypermetria of the left fore leg.

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Fig. 2 Horner's syndrome of the left eye.

CLINICAL DIAGNOSIS

Based on the history and symptoms the provisional clinical diagnosis was damage to the cerebellum and vestibular nuclei on the left side. The possible aetiologies considered were primary neoplasia of the brain, secondary neoplasia with brain involvement, abscessation, toxoplasmosis, *Coenurus cerebralis* infestation and thrombosis with consequent infarction of the brain. Laboratory tests and special examinations were performed to establish an aeriological diagnosis and hence, a prognosis.

Haematology, electrophoresis and blood chemistry tests were performed. The significant results are listed in Table 1. Of particular interest was the raised alpha globulin level indicating active cell destruction[3]. The extremely high level of serum alkaline phosphatase (SAP) was thought to indicate a bone abnormality. The serum alanine transaminase (SGPT) and the bromsulphalein (BSP) dye excretion tests values were only slightly elevated above normal, suggesting that most of the SAP originated from bone, rather than from the liver. The Sabin-Feldman dye test for toxoplasma antibodies was negative. Cerebro-spinal fluid was collected, analysed and cultured. An electroencephalogram was recorded and analysed. The findings of both procedures were normal, indicating that neither the sub-arachnoid space nor the cerebral hemispheres were affected.

Table 1: CLINICAL PATHOLOGY RESULTS

Test, units	Patient	Normal range
Haemoglobin, g/ℓ	176	120 – 180
Haematocrit	0,57	0,37 – 180
Leucocytes, x 10^9/ℓ	18,2	6,0 – 17,0
Neutrophils, %	94	60 – 77
Lymphocytes, %	3	12 – 30
Monocytes, %	3	3 – 10
Eosinophils, %	0	2 – 10
Total serum protein, g/ℓ	71	50 – 70
Albumin, g/ℓ	29,1	30 – 37
Globulin, g/ℓ	41,9	22 – 33
Alpha globulin, g/ℓ	22,1	3,1 – 18,6
SAP u/ℓ	1570	50 – 122
SGPT, mU/ℓ	72	6 – 25
BSP, % at 30 m	17	less than 5

Lateral and dorsoventral radiographs were taken of the skull. The lateral radiograph (Fig. 3) revealed a well

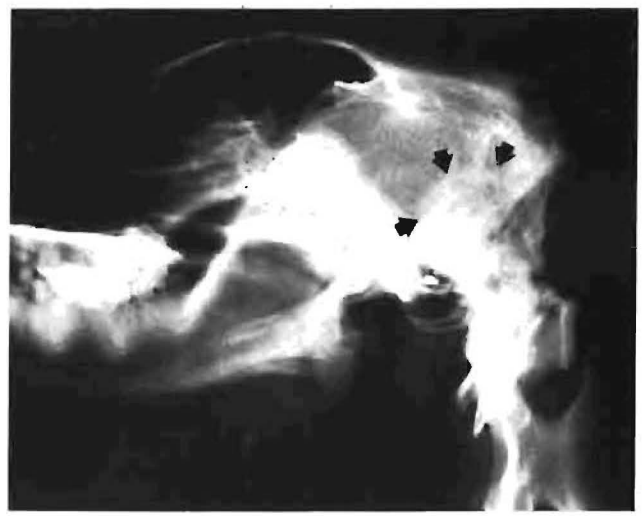


Fig. 3 Lateral radiograph of the skull. There is a well demarcated area of increased density (arrows) lying dorsal to the petrous temporal bones. The surface is undulating and the density is variable with numerous radiopaque granules scattered throughout the lesion.

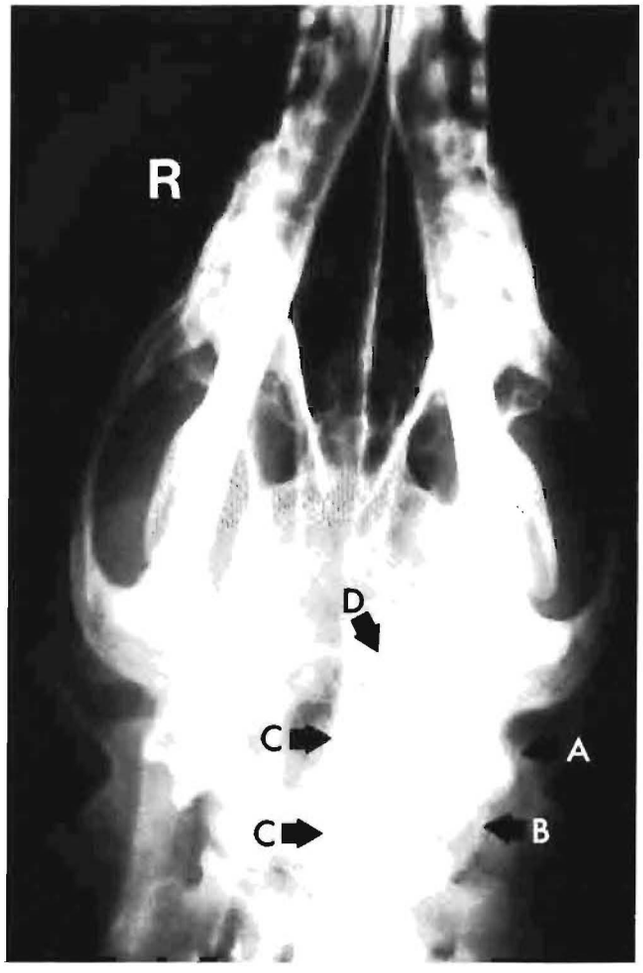


Fig. 4 Dorsoventral radiograph of the skull. The dense legion is situated on the left side of the head but has spread beyond the medium shadow caused by the external sagittal crest of the skull (C) to invade the right side (R). The tumour extends rostrally to the basisphenoid bone (D). The lesion projects laterally between the left occipital condyle and the mastoid process (A). The left paracondylar process has been destroyed (B).

demarcated area of increased density immediately dorsal to the petrous temporal bone and cranial to the internal occipital bone. There appeared to be some loss of bone substance and distortion of the temporal crest in this region. The lesion measured 2,5 × 3,0 cm and had an undulating surface. The density was variable with numerous small radio opaque granules scattered throughout the area. On the dorsoventral radiograph (Fig. 4) the dense lesion was situated on the left side of the head but had spread beyond the median shadow caused by the external sagital crest of the skull. It projected laterally between the left occipital condyle and the mastoid process. The left paracondylar process of the skull had been destroyed.

A radiological diagnosis of a neoplasm was made. It was thought to be cartilagenous or osseous in nature because of the presence of dense granules throughout the tumour mass. Lateral and dorsoventral radiographs of the thorax revealed no evidence of pulmonary metastasis.

Due to the invasive nature of this neoplasm, as assessed radiologically, and the extensive nervous involvement, as obvious from the clinical signs the prognosis was considered to be hopeless and the dog was humanely killed.

PATHOLOGY

Macroscopic

The only significant lesions found at post mortem examination were in the skull and cerebellum. During removal of the dorsal cranium from the underlying meninges and brain, it was found that the dura mater and leptomeninges over the left side of the cerebellum adhered closely to both the internal occipital bone and to the cerebellum. The meninges were irregularly thickened but intact and contained what appeared to be bony material (Fig. 5). An irregular nodular mass of bone was found ventral to the internal occipital protruberance. This mass extended into the cranial and occipital cavity, and was approximately 30 mm in diameter (Fig. 5). The left cerebellar hemisphere was enlarged (40 mm in diameter) and had an uneven surface which contained bony spicules. The enlarged cerebellar mass caused an indentation into the left cerebral hemisphere (Fig. 5).

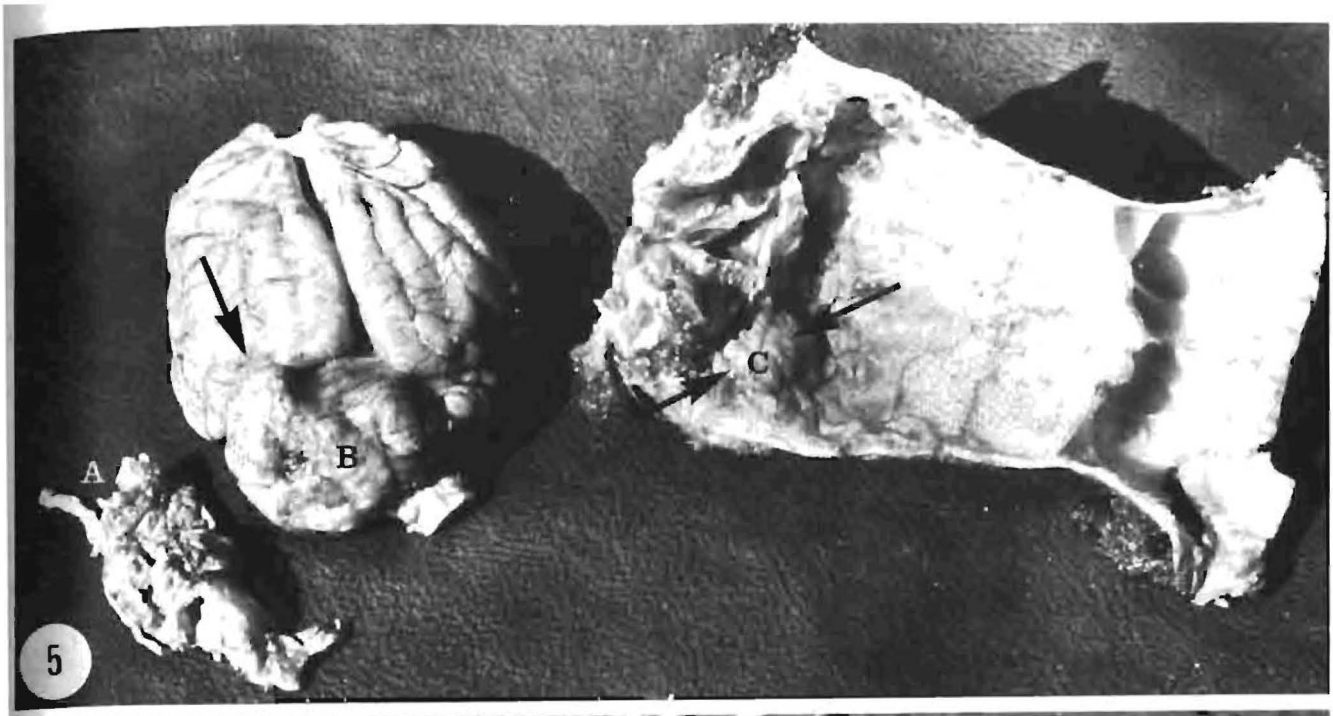
On cut section, the left cerebellar hemisphere was infiltrated with neoplastic cartilagenous and osseous tissue. Only a very thin layer of apparently normal cerebellar tissue remained on the periphery of this mass. Although the right hemisphere appeared to be normal from the outside, it was found that all the lobes were invaded by this tissue.

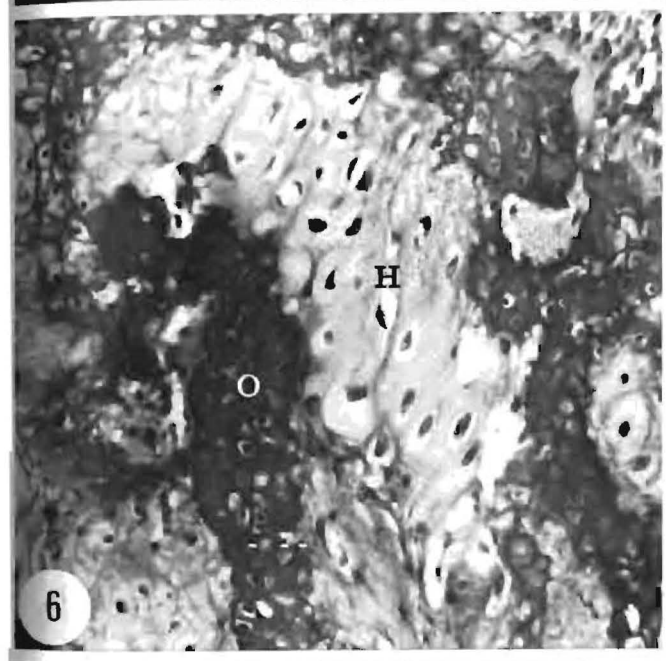
Specimens of the cerebellum, cerebrum, brain stem, spinal cord, meninges and internal occipital protruberance were taken for histopathological examination and were fixed in 10% formalin. Those specimens that contained bone and cartilage were decalcified in 8% formic acid. Sections were stained with haematoxylin and eosin (HE) and examined under the light microscope.

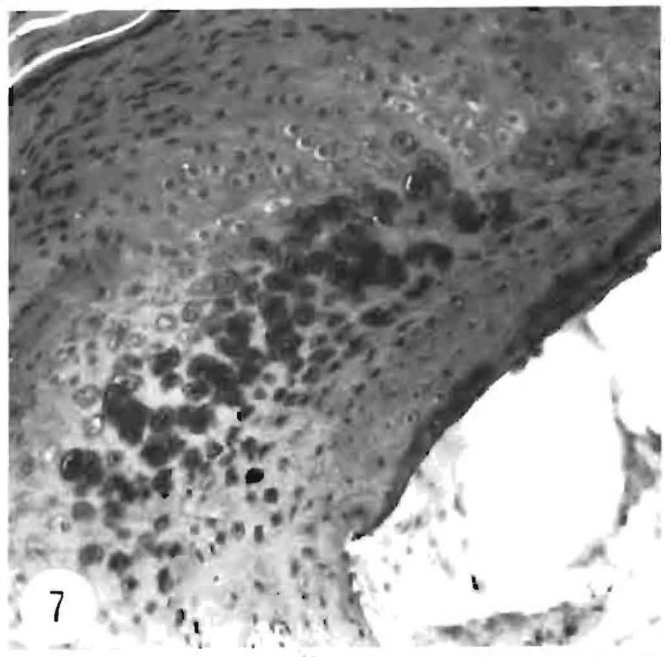
Microscopic

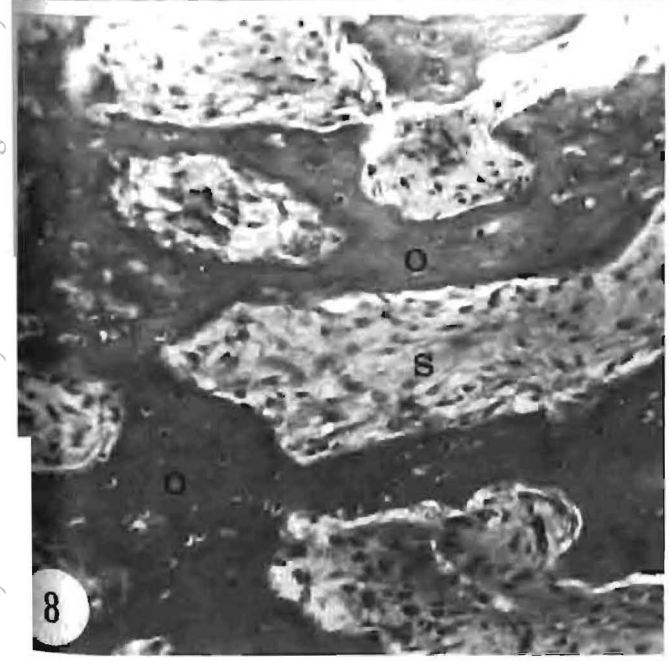
The cerebellum, meninges over the left cerebellar region and internal occipital bone showed similar microscopic changes. The cerebrum, brain stem and spinal cord exhibited no pathologically significant lesions.

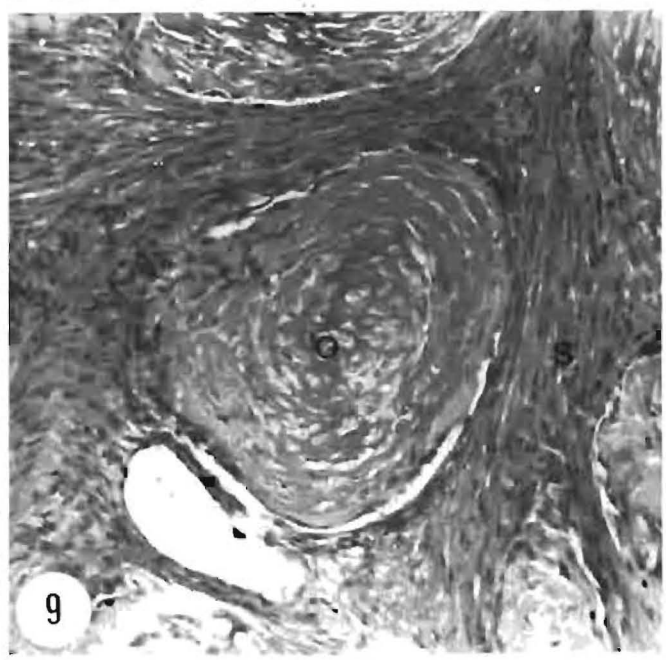
In the internal occipital bone scattered areas of normal tissue, containing Haversian canals, were seen. The majority of the bone, however, had been replaced by a neoplasm which was diagnosed as a chondroma rodens. It consisted of irregularly sized and shaped trabeculae of osteoid interspersed with islands of hyaline cartilage (Fig. 6). In some areas the hyaline cartilage was calcified (Fig. 7) and in others it appeared as if osteoid had replaced some of the cartilage (Fig. 6). Most of the abnormal osteoid was not calcified (Fig. 9) but some exhibited varying degrees of calcification (Fig. 6 & 8). Spindle cells were present around the cartilage and osteoid (Figs. 8 & 9). These cells were very similar in appearance to fibroblasts; each had a basophilic cigar-shaped nucleus with an elongated eosinophilic cytoplasm. Where the cells were closely associated with the osteoid, the nuclei were more vesicular, the nucleoli

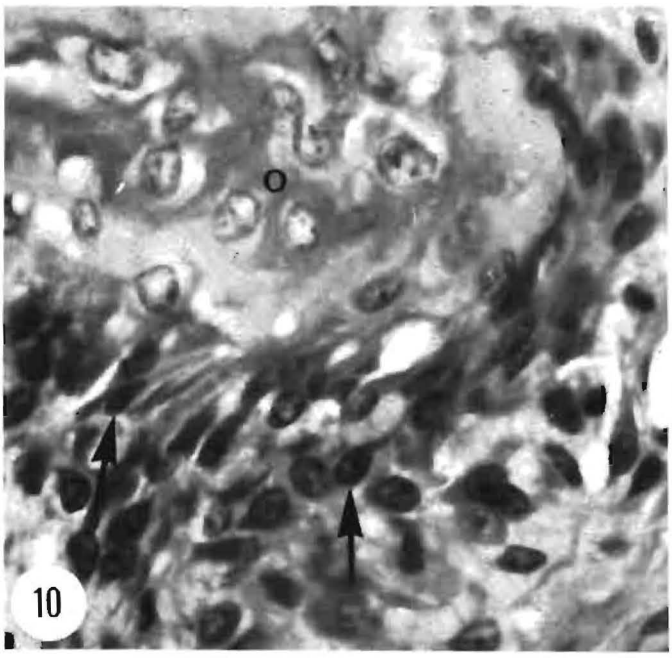


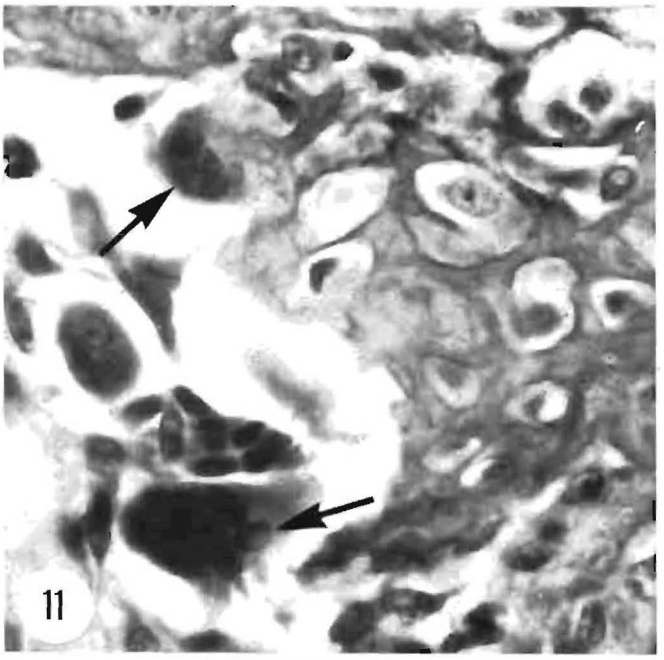


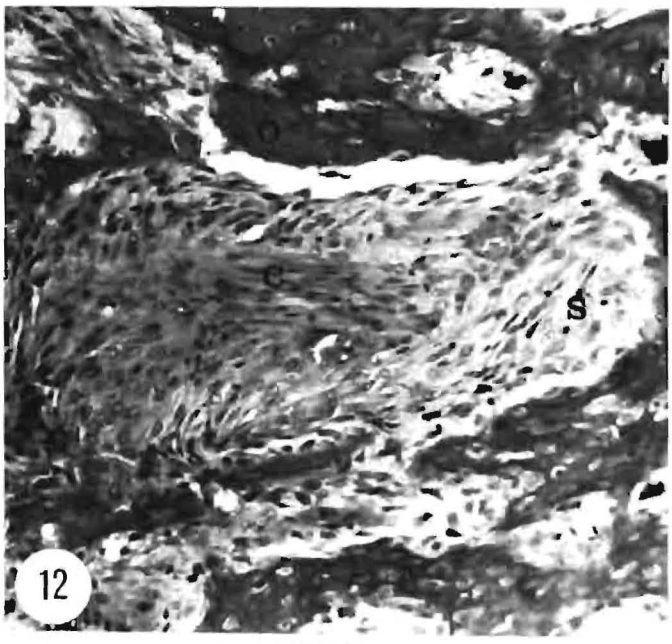


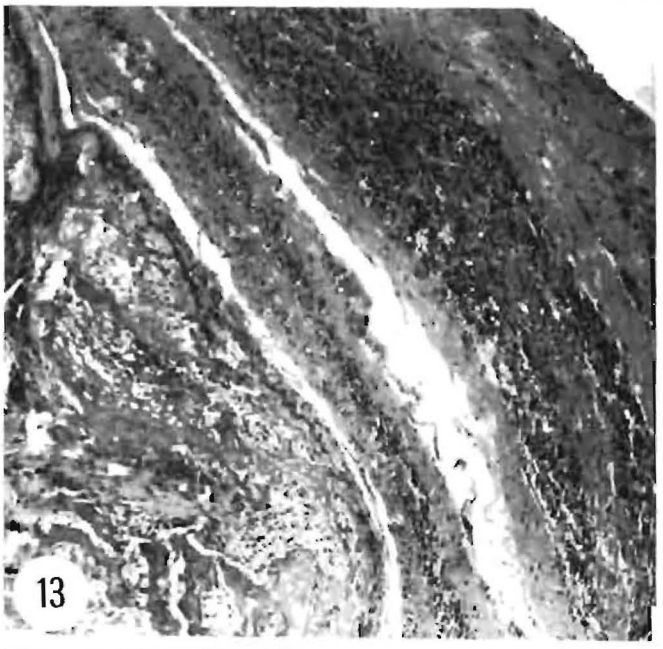


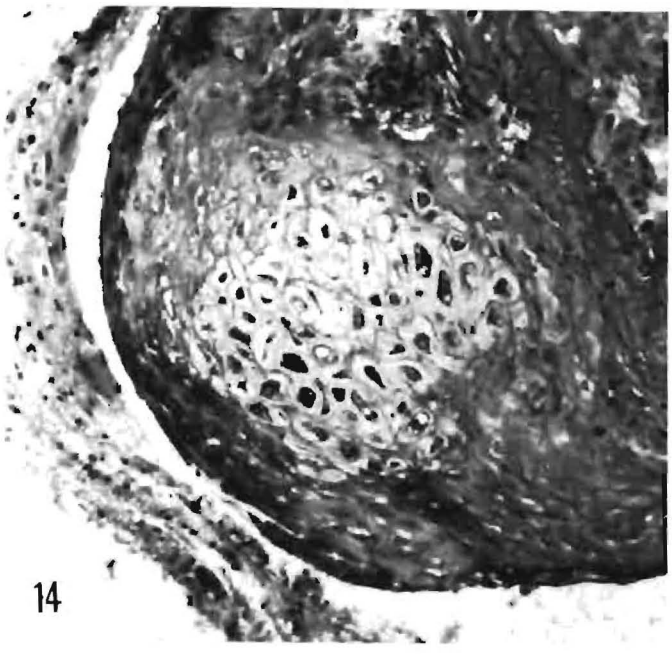












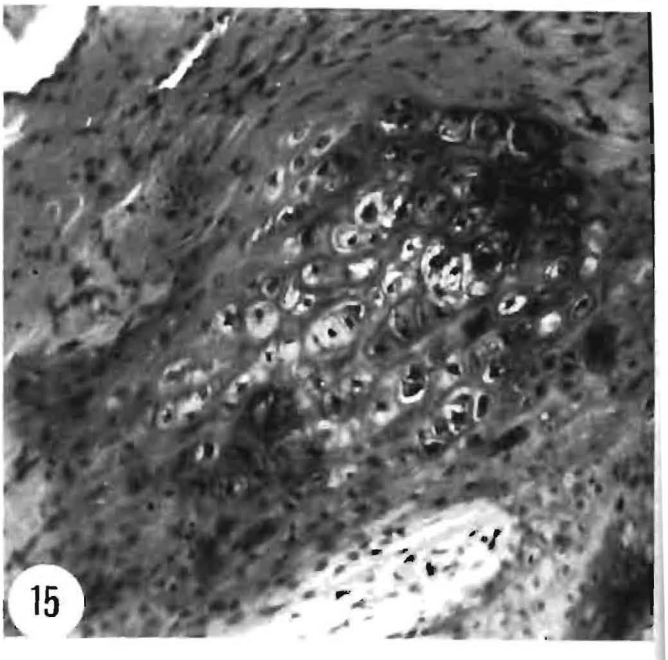


Fig. 5 A Irregularly thickened meninges of the left cerebellar hemisphere. B Unevenly enlarged left cerebellar hemisphere causing an indentation (arrow) into the left cerebral hemisphere. C Irregular, nodular mass of bone ventral to the internal occipital protruberance, extending into the occipital and cranial cavity (arrows).

Fig. 6 Hyaline cartilage (H) surrounded by partially calcified osteoid (O) HE X 284.

Fig. 7 Partially calcified hyaline cartilage HE X 284.

Fig. 8 Irregular osteoid trabeculae (O) surrounded by spindle-shaped cells (S) HE X 284.

Fig. 9 Concentrically arranged osteoid (O) surrounded by spindle-shaped cells (S) HE X 284.

Fig. 10 Osteoid (O) surrounded by osteoblasts (arrows) HE X 1139.

Fig. 11 Giant cells (arrows) closely associated with osteoid HE X 1139.

Fig. 12 Mature collagen (C) surrounded by spindle-shaped cells (S) and osteoid (O) HE X 284.

Fig. 13 & 14 Remnants of the atrofied cerebellum overlying the neoplasm HE X 284.

Fig. 15 Hyaline cartilage in the cerebellar neoplasm HE X 284.

were more prominent and their cytoplasm was more basophilic. In some areas it appeared as if they were responsible for deposition of osteoid matrix, i.e. osteoblasts (Fig. 10). Not all the osteoid tissue in the sections was closely associated with these cells. Many giant cells, resembling osteoclasts, were seen (Fig. 11). The spindle cells formed bands of collagenous tissue surrounding the hyalin cartilage and the osteoid (Fig. 9).

In the part of the tumour affecting the meninges, mature collagen, spindle cells and osteoid were present (Fig. 12). No hyaline cartilage was seen.

The cerebellum was extensively infiltrated with hyaline cartilage, osteoid and spindle cells and had a similar appearance to that described for the internal occipital bone. The cerebellar tissue was severely atrophied and only remnants remained. (Figs. 13 & 14). Hyaline cartilage islands were more numerous in the growth infiltrating the cerebellum (Fig. 15) than was the case in the neoplasm in the occipital bone.

DISCUSSION

The clinical signs of ataxia, head tilt to the left and hypermetria of the left fore leg, without circling, localised the lesion to the flocculonodular lobes of the cerebellum and the vestibular nuclei on the left side. The Horner's syndrome affecting the left eye indicated that the sympathetic nerves to that eye were damaged[6].

It was thought that the chondroma rodens arose from either the left internal occipital protruberance or from the dura mater in this area. It infiltrated into the left cerebellar hemisphere with subsequent distortion of the structures described above.

In Jacobson's description of this tumour in humans and animals, the origin was not clear[4]. Brodey, Misdorp, Riser & van der Heul recorded only one chondroma rodens in a study on 35 canine skeletal chondrosarcomas[1]. However, they do not mention the origin of the neoplasm. Misdorp & van der Heul described chondroma rodens as a soft tissue tumour and mention that it possibly arises from aponeuroses and secondarily infiltrates into the underlying bone[5]. Jacobson found that it appeared as if the spindle cells give rise to foci of cartilage and osteoid[4]. This is similar to the findings in the case under discussion.

Three distinct entities occur in chondroma rodens, i.e., spindle cells, cartilage and osteoid. Liu & Dorfman, as quoted by Misdorp & van der Heul, consider this growth to be "the cartilagenous counterpart of fibromatosis"[5]. Other important differential diagnoses, from the histopathological point of view, are fibrous dysplasia and ossifying fibroma[7]. Fibrous dysplasia exhibits spindle cells (fibroblasts), osteoid and many giant cells. Hyaline cartilage may be present in these growths but it is not a common finding. Ossifying fibroma consists of fibroblasts and osteoid, with osteoblasts lining irregular bony spicules[7]. Fibrous dysplasia is differentiated from ossifying fibroma by the presence of osteoblasts in the latter. It is clear that due to the presence of spindle cells and osteoid chondroma rodens is similar to both of these growths. The main differentiating feature of the chondroma rodens is the presence of cartilage and the osteoblastic activity.

Chondroma rodens is an infiltrative neoplasm and no metastasis has been described[1 4 5]. In the case recorded here it appeared as if the tumour arose from the internal occipital protruberance or in the underlying meninges with infiltration into the cerebellum and occipital bone.

ACKNOWLEDGEMENTS

Appreciation is extended to the technical staff of the Departments of Pathology and Medicine for their assistance, Mr R Watermeyer for preparing the photographs and Mrs F Fouche for typing the manuscript. Dr. D.M. Burstein is thanked for referring the case and Prof C Roos for taking and interpreting the radiographs.

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CASE REPORT **GEVALVERSLAG**

FATAL PARASITISM AMONG FREE LIVING BUSHBABIES *(GALAGO CRASSICAUDATUS)*

ABSTRACT: Evans L.B. Fatal parasitism among free living bushbabies *(Galago crassicaudatus)*. Journal South African Veterinary Association (1978) **49** No. 1, 66 (En) 25 Roehampton Way, 4051 Durban North, Rep. of South Africa.

Four cases of severe parasitism due to *Primasubulura otolicini* in adult bush babies *(Galago crassicaudatus)* are described.

INTRODUCTION

Inanda Game Park consists of an area of approximately 550 ha and is situated close to the Natal coast. It contains numerous species of herbivores but no mammalian carnivores. The only predators present are avian, e.g. eagles and owls. These avian predators would probably seize and devour any ailing small mammals such as bushbabies. Consequently, it is extremely rare for any of these small nocturnal mammals to be found or even seen, although at night they can be heard.

During the months of August and September 1976, four adult bushbabies *(Galago crassicaudatus)* were found either dead or dying by the warden and game guards of Inanda Game Park. As no severe climatic conditions had been experienced during these months, the warden became alarmed and sought veterinary advice.

NECROPSY

At necropsy it was found that the caecum was packed with nematode parasites *(Primasubulura otolicini)* forming a solid mass and completely obstructing the lumen. Small, cyst-like structures were present in the myocard but the cause of these was unknown. No other parasites were recovered. There was no food present in the gut and no fat present in the abdomen or surrounding the heart.

On necropsy three animals revealed identical findings and death was ascribed to parasitism.

DISCUSSION

Parasitism in the wild animals at Inanda Game Park is generally not a great problem, with the hosts and parasites apparently living in a harmonious relationship. Worm burden studies carried out on blesbok revealed a variety of nematode parasites (including *Cooperia yoshidae*, that had not previously been described in this species), but the numbers of parasites were not excessive and the animals showed no undue adverse effects from them.

However, for some unknown reason, parasite burdens in the Bushbabies apparently increased so rapidly as to place an excessive stress upon their hosts at a time of the year when food is not as abundant as at other times. The reasult was deaths among these animals.

The puzzling question is why this increase in parasite numbers should have occurred? The climatic conditions as mentioned above were not overly severe and parasitic mortality is not a regular feature of this time of the year. The game warden and game guards who have lived in the area for many years have no recollection of similar occurrences in previous years.

Interestingly enough, during the winter of 1977 there was only one reported bushbaby death. The necropsy findings were as described above. One wonders whether some factor may have influenced the bushbaby's natural susceptibility–resistance so that they were unable to maintain their parasite numbers at a nonpathogenic level. As there were no signs of any disease or any pathology other than that mentioned above and man exerts relatively little effect upon their environment in a protected area such as a game park, the possibility that these played a role in reducing their resistance can probably be discounted.

The deaths ceased equally suddenly. The possible explanation that may be inferred from this is that as the season advanced into spring (although not very noticeable at the Natal coast, where Inanda Game Park is situated) the available food improved and the animals were better able to cope with or eliminate their worm burdens.

If the mortalities had not ceased a further problem that would have arisen would have been how to set about dosing wild nocturnal bushbabies with suitable anthelmintics. The use of fenbendazole (Panacur–Hoechst Pharmaceuticals) in bananas is a possibility, but fortunately the need to determine whether this method would have been successful did not arise.

ACKNOWLEDGEMENTS

I would like to record my thanks to Dr. Anna Verster of the Veterinary Research Institute, Onderstepoort and Dr. I. Horak of the Faculty of Veterinary Science, University of Pretoria, for identification of the parasites.

I would also like to thank Mr. D. Chapman, Warden of Inanda Game Park, for all his assistance, co-operation and unfailing interest in wildlife.

*25 Roehampton Way, 4051 Durban North.

CASE REPORT **GEVALVERSLAG**

HYPOGLYCAEMIA IN A DOG WITH PANCREATIC ISLET CELL ADENOMAS

W.S. BOTHA,* C. IRVINE-SMITH**

ABSTRACT: Botha W.S.; Irvine-Smith C. Hypoglycaemia in a dog with pancreatic islet cell adenomas. *Journal South African Veterinary Association* (1978) **49** No. 1, 67–69 (En) Dept. Path. Fac. Vet. Science, Univ. Pretoria P.O. Box 12580, 0110 Onderstepoort, Rep. South Africa.

The clinical, clinical pathological, pathological and histopathological findings of hypoglycaemia in a dog due to functional pancreatic islet cell adenomas are described.

CLINICAL SIGNS

A 9 year old male dog of mixed breed weighing approximately 33 kg was presented to one of us (C. I-S) in a collapsed and semi-comatose state. The dog was recumbent, had very weak reflexes and a rectal temperature of 39°C when first examined. Unconsiousness set in within hours and bradycardia, constricted pupils and a mild dyspnoea were present.

Emergency treatment consisted of injecting 100 mg prednisolone sodium succinate***, 1 gm atropine sulphate and 500 ml of Ringer's lactate via a jugular catheter. A solution containing penicillin and streptomycin**** was given intramuscularly. A bicarbonate reserve test (see Table 1) indicated a low value (11,7 mg/l) and therefore 250 ml of 4,2% solution of sodium bicarbonate was administered via the jugular catheter. The following differential diagnoses were considered:

1. Acute poisoning;
2. Rupture of an internal organ;
3. Snake bite;
4. Acute hypoglycaemic crisis; and
5. Apoplectic stroke.

The dog regained consciousness after treatment and on the following morning had a rectal temperature of 38,5°C but was still very weak. Large quantities of urine were passed during the night due to the intravenous fluid therapy of the previous day. Blood was collected for clinical pathological examination and tests performed as indicated in Table 1. Blood glucose value was found to be a significantly low 41,0 mg/100 ml indicating a hypoglycaemic state. A presumptive diagnosis of a functional islet cell tumour secreting insulin was made and therefore the dog was treated with one ℓof an aqueous solution containing various electrolytes and invert sugars† by slow intravenous infusion and 33 mg prednisolone†† intramuscularly. The same dose of prednisolone†† was repeated twice daily for several days to increase the blood sugar value which stabilized at 75 mg/100 ml. The habitus and general condition of the dog improved and it was discharged. An oral daily dose of 2 mg Dexamethazone††† was prescribed on a reducing dosage regimen.

Three weeks later the animal was reported to have developed posterior paresis and a severe generalized weakness when excercised. The oral dose of Dexamethazone††† tablets was increased from 1 mg/d to 5 mg/d and supplemented with sugar water. This treatment maintained the dog in a near normal condition but periodic acute hypoglycaemic episodes occurred during the following months.

Nearly 6 months after initial examination a major hypoglycaemic crisis occurred; the blood sugar value was 33 mg/100 ml. The animal was in a comatous condition and the owner requested euthanasia.

Table 1: CLINICAL PATHOLOGICAL TESTS

Date	2.3.76	3.3.76	4.3.76	8.3.76	24.8.76	Normal values
Blood glucose mg/100 ml	—	41	75	75	33	70–100
Bicarbonate reserve mEq/1	11,5	23,4	—	—	—	18–24
S G P T mU/ml	—	6	—	—	—	6–25
B U N mg %	25,6	—	—	—	—	16–25
P C V mg %	53	47	—	—	—	45
W C C X10³	—	19,8	—	—	—	6–17
Neutrophils %	—	87	—	—	—	60–77
Lymphocytes %	—	5	—	—	—	12–30
Monocytes %	—	8	—	—	—	3–10

*Department of Pathology, Faculty of Veterinary Science, University of Pretoria.
**Practitioner, Box 67092, 2021 Bryanston.
***Solu-Delta-Cortef V, Upjohn (Pty).
****Streptopen, Glaxo-Allenbury (Pty) Ltd.

†Maintelyte Baxter Laboratories Morton Grove Illinois
††Delta Cortril V Pfizer Laboratories S.A. (Pty) Ltd.
†††Decadron, Merck, Sharp & Dohme (Pty) Ltd.

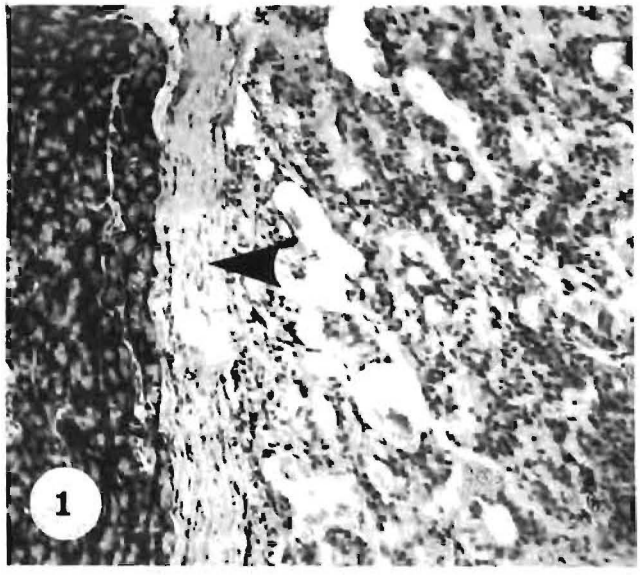

Fig. 1 Islet cell adenoma. The collagenous capsule is indicated by a arrow. H. & E. stain, X 100.

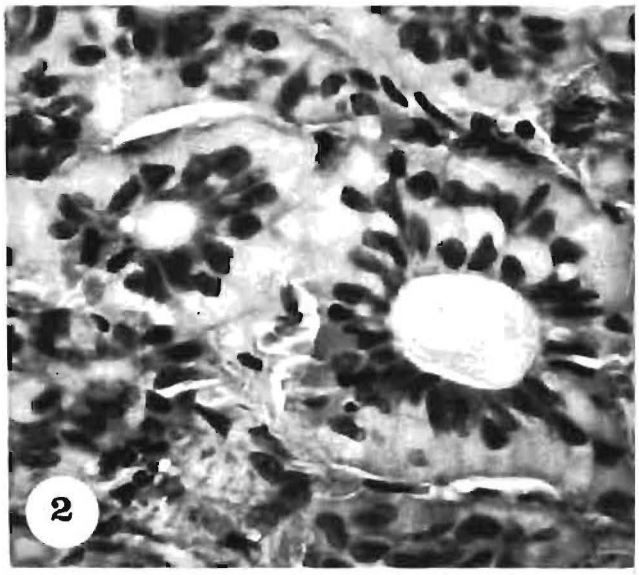

Fig. 2 The rossettes with homogenous intercellular material visibl in their centres. H. & E. stain, X 400.

PATHOLOGY

At post mortem examination the dog was found to be in good condition. Special attention was given to the pancreas and three small nodules were palpable within the substance of the right wing (duodenal portion) of the organ. On cut section these nodules appeared as firm, white, round, circumscribed adenomas with diameters of 5–10 mm. The liver was degenerated (fatty changes were suspected) and slightly enlarged but no other specific macroscopical pathological changes were noticed.

Upon histopathological examination all three tumours had the same morphological appearance. A diagnosis of multiple adenomas of the islets of Langerhans, was made. The neoplasms were well encapsulated (see Fig. 1) and a Masson's trichrome stain was used to show the well developed fibrous septa that subdivided the adenoma. Numerous dilated capillaries were present and many haemosiderin laden macrophages were found in the interstitial tissue.

The neoplastic cells resembled the normal cells of the islet of Langerhans. In HE stained sections the nuclei were round or oval and dark while the abundant cytoplasm was pale but had a granular appearance. The cell boundaries were not distinct. The neoplastic cells occurred in irregularly sized nests in many of which the cells were arranged in a rosette pattern (See Fig. 2). These rosettes contained a homogenous eosinophilic intercellular material in their centres which were positive to periodic acid Schiff stain. A Gomori aldehyde fuchsin stain did not confirm the presence of beta-granules in the neoplastic cells. The absence of these specific granules in insulin secreting islet cell tumours is well known[15]. The mitotic index was low and no sign of infiltration of the neoplasm into normal surrounding tissue was found.

Hydropic degeneration of the epithelial cells of the convoluted tubules in the kidney and of the centrilobular hepatocytes in the liver, was noticed. A mild bilirubin pigmentation was present in the renal tubular epithelial cells. Frozen sections of a lymph node stained with oil red O showed severe fatty infiltration into the interstitial tissue especially of the paracortical areas. The fat occured in varying sized globules and appeared to be lying free. Mild fatty changes were present also in the hepatocytes and the cells of the zona reticularis of the adrenal gland. The hypophysis was not examined.

DISCUSSION

In clinical terminology, hypoglycaemia may be defined as the state in which the blood sugar value falls below 50 mg/100 ml[2 4]. It is a serious condition because of the dependence of the brain tissue on glucose as a nutrient. The principal mechanisms hy which hypoglycaemia may develop in man are given by Sodeman[5] as impaired gluconeogenesis (Addison's disease, hepatic failure), tachyalimentation of glucose (post gastrectomy, abnormal "glucostat" (prediabetic state), excessive unregulated insulin released (functional islet cell adenoma) and by an unknown mechanism (thoracic mesothelial tumours). A lack of secondary signs and pathological changes may rule out hyperglycaemia in the dog caused by adrenal cortical insufficiency, hepatic failure, cachexia, hypothyroidism, inanition, distemper and repeated vomition[2 3]. Although sophisticated laboratory tests like those for the tolerance of glucose, leucine, tolbutamide and other substances have been described, the most practical and useful method of diagnosing hypoglycaemia due to islet cell neoplasms is by fasting blood glucose determinations[2 4]. In our case, the blood sugar values of 41 mg/100 ml and later 33 mg/100 ml (see Table 1) after fasting for 24 h showed a consistent hypoglycaemic state indicative of islet cell tumours.

The pathological features found in our case correspond well with those already recorded[2 3 4 5 7]. The hepatic and renal hydropic degeneration (may partly be due to glycogen infiltration) and fatty changes of the lymph nodes, liver and adrenal cortex were interpreted as changes secondary to the hyperinsulinism and administration of sugar water. Functional islet cell adenomas may be small (up to 20 mm)[1–4], as was the case with our dog. The rosette pattern of the cells in this case to-

gether with the presence of periodic acid Schiff positive intercellular material is deemed worthy of note. A rosette pattern but without the central homogenous material, has been described in humans[1].

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LETTER TO THE EDITOR — BRIEF AAN DIE REDAKTEUR

Sir

HYPOGLYCAEMIA IN A PREGNANT BITCH

A Chihuahua bitch, aged three years and in advanced stage of pregnancy, was presented for examination with symptoms of ataxia. The history was that it had eaten normally the previous evening and seemed normal when the owner left for work in the morning.

Examination revealed bradycardia (64/min) and ketosis could be smelled from the breath. Temperature was normal. Foetal heart beats were auscultated (±150 per minute) Capillary blood smear showed no evidence of babesiosis. A venous blood sample showed rapid sedimentation and lipaemia. Blood glucose estimation, using "Dextrostix", showed no perceptible colour change, indicating a glucose level somewhere between 0 and 25 mg/100 ml blood. A reading below 40 mg/100 ml blood is accepted as being diagnostic of hypoglycaemia. A venous blood smear showed moderate poikilocytosis which was considered indicative of liver dysfunction.

Treatment:

The dog was kept under constant observation during a period of three hours because I was not certain that I was dealing with a primary hypoglycaemia. At the end of three hours she was deeply comatose and commencement of treatment became imperative. 150 ml of a 5% dextrose saline solution was slowly administered intravenously and tioctic acid intramuscularly.

After fifteen minutes of dextrose saline administration there were signs of returning consciousness and after thirty minutes the bitch was fully conscious and able to walk. Heart rate increased during treatment and was normal next day. Glucose was administered *per os* for a few days and further recovery was uneventful.

Hypoglycaemia, associated with hyperinsulinism, is common in the dog but I cannot find any reference to its occurrence in pregnant bitches. One can see considerable similarities to pregnancy toxaemia in pregnant ewes. This bitch was fed only once daily so incorrect diet was obviously the direct cause of the condition.

W.J. Handcock MVB, MRCVS
2 Rivett St
6140 Grahamstown

Die mees belangrike 21 dae in 'n koei se jaar.

Die Probleem.

'n Hoë persentasie koeie het 'n residuele infeksie aan die einde van laktasie.

Daarby is alle koeie vatbaar vir 'n nuwe infeksie gedurende die vroeë stadia van die droë periode. Met 'n paar uitsonderings sal dit gedurende die eerste 10 tot 21 dae voorkom.

Die Antwoord.

Orbenin Droë Koei is ontwerp om beide hierdie probleme te oorbrug. Dit is geformuleer as gevolg van aanhoudende navorsing, beide in die laboratorium en in die veld.*

Orbenin Droë Koei is bakteriedodend teen streptokokke, penisillien sensitiewe en penisillien weerstandbiedende staphylokokke, die mees oorsaaklike organismes wat in residuele en nuwe infeksies gevind word.

Dit was duidelik uitgewys dat infeksie tydens kalwing gewoonlik van NUWE oorsprong en meesal 'n omgewings, of Gram -we infeksie is. Behandeling tydens daardie tyd met 'n breëspektrum Lakterende Koei produk soos Ampiclox L.K. is nodig om genoegsame antibiotiese konsentrasies te bereik om mastitis tydens kalwing te voorkom.

Orbenin Lakterende Koei en Ampiclox L.K. is bedoel om mastitis gedurende laktasie te beheer. Ampiclox word veral aanbeveel wanneer coliforme vermoed word.

Orbenin (kloksasillien) en Ampiclox (ampisillien/kloksasillien) is Beecham handelsmerke.

*Die jongste kliniese proef het 507 kuddes oor 'n tydperk van 3 jaar ingesluit – Brander G.C., Watkins J.H., en Gard R.P., Vet Rec. (1975) 97. 300-304.

Beecham Dieregesondheid — **Vordering in die Praktyk**

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BA 4138

CONGENITAL UNILATERAL AOTUS IN A BLACK RHINOCEROS *DICEROS BICORNIS BICORNIS* (Linn., 1758)*

The unilaterally earless adult black rhinoceros *Diceros bicornis bicornis* bull, as depicted, was caught in the Hluhluwe Nature Reserve and translocated to the Addo Elephant National Park during September 1977. Two other adult black rhino bulls with normal external ears made up the full consignment. They were introduced into Addo as breeding males in order to fill a vacancy which was left by the recent demise of the only adult bull in the Addo rhino population. Before this introduction the population consisted of 10 animals that were products of an introduction of seven black rhinos, in 1961 and 1962, from Kenya. These animals and their offspring all had normal pinnae.

Uni- or bilateral aotus in the black rhino has been recorded from at least seven discrete populations in eastern and southern Africa, inter alia the Hluhluwe Game Reserve[1]. It has also been suggested by Goddard[1] that a sex linked genetic character might be responsible for this congenital deformity. It will therefore be interesting to see what effect the introduction of this aotic male will have on a "normal" population with their full quota of ears.

REFERENCE

1. GODDARD J. 1969. A note on the absence of pinnae in the black rhinoceros. *East African Wildlife Journal* 7: 178.

*Submitted by Dr V. de Vos, Department of Nature Conservation, National Parks Board, Private Bag X404, Skukuza. 1350.

NUWE GRADUANDI VAN DIE FAKULTEIT VEEARTSENYKUNDE UNIVERSITEIT PRETORIA
NEW GRADUATES OF THE FACULTY OF VETERINARY SCIENCE UNIVERSITY OF PRETORIA

Op die oorkantse blad verskyn 'n klasfoto van die Finalejaar studente van 1977. Tydens 'n gradeplegtigheid op 18 November 1977 is die BVSc-graad aan 36 nuwe veeartse toegeken.

Na voldoening aan al die vereistes vir die graad kan die ander kandidate ook verwag om in die eerskomende maande te gradueer. **Die SAVV verwelkom hulle almal as lede van die professie.**

Tydens 'n afskeidsfunksie deur die doserende personeel van die Fakulteit is die volgende toekennings deur die genoemde instansies aan finaliste gemaak:

A photograph of the Final Year Class of 1977 appears on the opposite page. At a graduation ceremony on the 18th November 1977, the BVSc degree was awarded to 36 new veterinarians.

After meeting all the requirements for the degree the other candidates are expected to graduate early in 1978. **The SAVA bids them all a warm welcome to the profession.**

At a farewell function arranged by teaching staff of the Faculty the following awards were made by the donors mentioned below:

SA Biologiese Vereniging:
Die Theiler Gedenkmedalje aan
R F HORNER

Die Tak WWR en SAVV:
Die Kliniese Medalje aan
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SA Biological Society:
The Theiler Memorial Medal to
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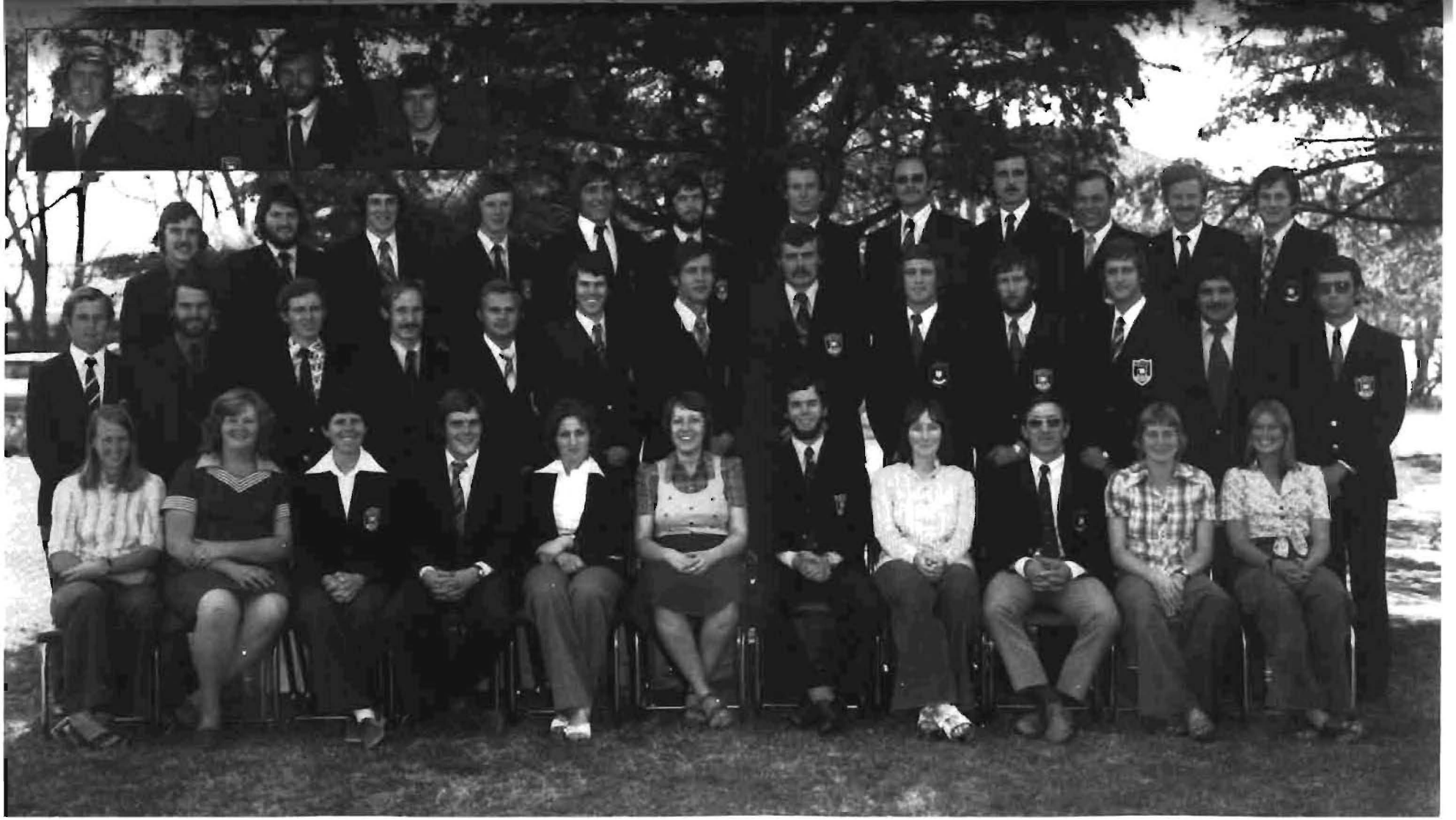

V.L.N.R./L. to R.

Agter/Rear: B Pollard, J P Niland, B M Anderson, W R Cunliffe, P J Whitfield, C W Moore, P V Andreae, R H Hassel, J van Aswegen, L Connoway, D J Porter, J M Fourie

Middel/Middle: N Kriek, K P Shulman, N H Christensen, H W Dickerson, A G Visser, J G Gage, J C van Niekerk, J B Morkel, G H Theron, L C Coetzer, H J J Steyn, C du T Smith, M Hornsveld

Voor/Front: E v d Horst, G Smith, L E Lunn, C R W Barnard, M F S Bailey, R C Jeppe, R O Gilbert, A Stettler, G P J du Preez, M Thomas, S A Smith

Inlas/Inset: M Strong, B C Bowles, R F Homer, B R Stevens. **Afwesig:** N Bainbridge

TOEKENNING **AWARD**

LEWENSLANGE ERE-ONDERPRESIDENT

Die konstitusie van die SAVV maak o.a. voorsiening vir verering van gewone lede wat uitmuntende dienste aan die veeartsenykundige beroep gelewer het. Dienooreenkomstig is 'n sekere benoeming deur drie lede van die Bestuur gemaak en het die Vereniging tydens sy Algemene Jaarvergadering gehou op 30 Augustus 1977 te Grahamstown eenparig vir

PROF. EMERITUS HENRI PIETER ALBERT DE BOOM

Prof H P A de Boom

verkies tot *Lewenslange Ere-onderpresident* van die Suid-Afrikaanse Veterinêre Vereniging.

Sowel binne as buite die grense van die Republiek is hy baie goed bekend as uitstaande wetenskaplike, 'n hoogaangeskrewe en uiters bekwame dosent en 'n man met 'n vriendelike en innemende persoonlikheid. In besonder is hy bekend vir sy jarelange diens op die Redaksie van die Tydskrif van die SAVV – as gewone lid sedert 1950 en as Redakteur van 1959 tot 1961 en weer vanaf 1970 tot 1975.

Hy is gebore in Pretoria in 1914, het daar skoolgegaan en in 1937 die BVSc-graad aan die Fakulteit Veeartsenykunde van die Universiteit van Pretoria behaal. In 1937 word hy in die Seksie Patologie van die NIV te Onderstepoort aangestel en in 1940 word hy deeltydse dosent in histologie en embriologie in die Dept. Anatomie van die Fakulteit Veeartsenykunde van UP. In 1955 word hy Professor en Hoof van die Dept Anatomie en hy beklee die pos tot aftrede as Professor Emeritus aan die einde van 1974.

In 1953 is hy vir 'n jaar na Cornell Universiteit in die VSA om verdere studie in histochemie, endo-krinologie, neuroanatomie en pedagogie te onderneem as houer van die Commonwealth (later die Harkness) Fund Fellowship. In 1961 word hy vir 15 maande besoekende professor in die Dept Anatomie van die New York State Veterinary College en weer in 1975 vir 'n jaar. Gedurende die jare 1968–1970 dien hy ook op spesiale uitnodiging van die Portuguese Regering as Professor en Hoof van die Dept Anatomie aan die Fak. Veeartsenykunde van die Universiteit van Lourenço Marques.

Hy is 'n aktiewe lid van verskeie wetenskaplike verenigings. Hy is dienende Bestuurslid en gewese Sekretaris en President van die SA Biologiese Vereniging. Van die SA Vereniging vir die Bevordering van die Wetenskap is hy etlike jare lank Raadslid gewees en ook President van die Seksie E 19. Hy is volle lid van die SA Akademie vir Wetenskap en Kuns. Hy is stigterslid van die Anatomiese Vereniging van SA en was President in 1973. Hy is lid van die SA Genetiese Vereniging. Vanaf 1965–1973 het hy die Fakulteit Veeartsenykunde op die Veeartsraad verteenwoordig en vir die tydperk 1976–78 is hy een van die SAVV-gekose lede op genoemde Raad.

As Lewenslange Ere-onderpresident het Prof de Boom ook sitting op die Raad van die SAVV en daar sal hy ongetwyfeld verdere diens aan die professie lewer. Ons wens hom van harte geluk met die onderskeiding – omdat hy dit terdeë verdien!

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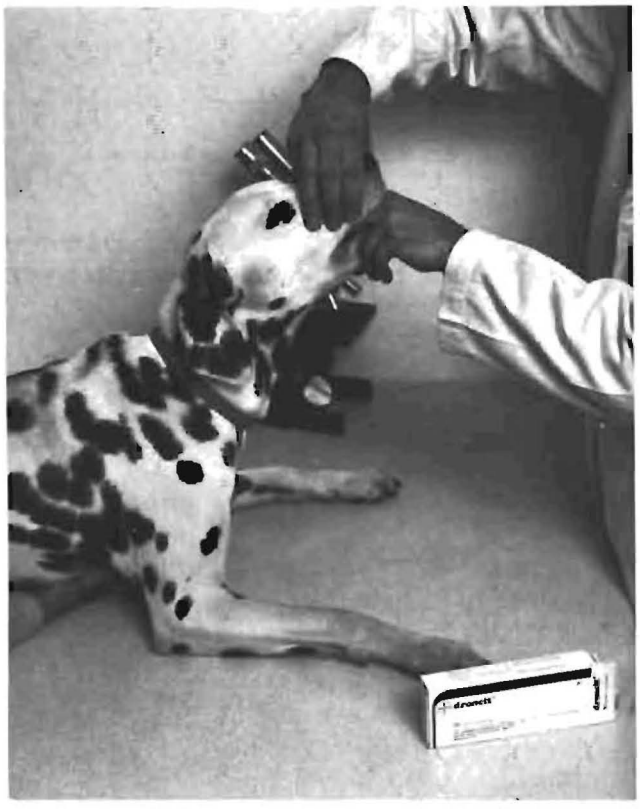

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