CHAPTER 4

Pathology of parasitic diseases

of

free-living mammals

Introduction

A few parasitic diseases of which the pathology had not yet been described, or the descriptions of which needed to be augmented, are listed here. My main contribution was the identification of the parasites and, in one case, supply of material. Together with Dr. Dewald Keet and Prof. Nick Kriek, the pathology of the lesions that were seen in buffaloes in the Kruger National Park were elucidated. I was co-promoter for Dr. Keet's M.Med.Vet thesis, and, of course, the first to identify the parasite as *Parafilaria bassoni*, an identification that was later confirmed by Dr. Odile Bain. The interesting aspect is that this parasite was for the first time recovered and described from the retro-orbital spaces of springbok, *Antidorcas marsupilalis*, from Marienthal in Namibia in 1964, and has since never been found again. The information on the parasite formed a part of the thesis and was rewritten to suit the format of the Onderstepoort Journal of Veterinary Research.

The references are in chronological order because of the nature of the contributions.

PLETCHER, JOHN M., HORAK, I.G., DE VOS, V. & BOOMKER, J., 1984. Nodular abomasitis in impala (*Aepyceros melampus*) caused by the nematode *Longistrongylus sabie*. *Journal of Parasitology*, 70, 907 - 910.

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PLETCHER, JOHN M., BOOMKER, JOOP, DE VOS, VALERIUS & GARDINER, CHRIS H., 1989. Lesions in the heart and lungs of greater kudu (*Tragelaphus strepsiceros*) caused by *Cordophilus sagittus* (Nematoda: Filaroidea). *Journal of Zoo and Wildlife Medicine*, 20, 465 - 470.

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NODULAR ABOMASITIS IN IMPALA (*AEPYCEROS MELAMPUS*) CAUSED BY THE NEMATODE *LONGISTRONGYLUS SABIE*

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ABSTRACT: The periodic occurrence of nodular abomasitis associated with the trichostrongylid nematode *Longistrongylus sabie* was observed in impala lambs of the Kruger National Park, Republic of South Africa. The condition was seen predominantly in animals less than 1 yr of age. Peak incidences occurred in the spring and fall, when more than 50% of the lambs studied had from several to numerous nodules in their abomasal mucosae. The nodular lesions in the lambs were macroscopically larger than, but microscopically similar to, those observed in domestic ruminants with ostertagiosis; however, *L. sabie* worm burdens were much lower than those in clinical cases of ostertagiosis, and no evidence of diarrhea could be found in any of the lambs studied. The physical condition of lambs with moderate to severe nodular abomasitis did not differ noticeably from that of lambs with mild involvement or those without lesions. Nodular abomasitis caused by this parasite was of minimal significance to impala herds in the Park under the circumstances prevailing at the time of the study.

During a recent parasitologic and pathologic study of free-ranging impala of the Kruger National Park (KNP), the periodic occurrence of a unique nodular abomasitis was noted. In many of the nodules the trichostrongylid parasite *Longistrongylus sabie* (Gibbons, 1977) and/or its eggs could be found. Herein, we consider some initial parasitologic information concerning this wildlife parasite and describe the gross and microscopic lesions it produces in the abomasal mucosa. Some comparisons are drawn between this condition in impala and ostertagiosis in domestic ruminants.

MATERIALS AND METHODS

Male impala from 3 age groups and some females were studied monthly over a period of 1½ yr. Each month several lambs (less than a year old), yearlings, and adults were taken at random from impala herds in the southern part of the KNP. External and internal parasite identification and enumeration as well as a complete pathologic examination were accomplished for each animal. More than 140 impala were included in the study. Recovery of parasites was carried out as described by Horak et al. (1982), and enumeration was accomplished by examining samples of digested abomasal mucosae and abomasal contents. Representative abomasal nodules were fixed in 10% neutral buffered formalin, embedded in paraffin and cut at 6 to 8 μm. Sections were stained with hematoxylin and eosin (HE) and the periodic acid-Schiff (PAS) technique.

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RESULTS

Parasitologic findings

Table I gives the numbers of 4th-stage larvae and adult *L. sabie* found in the first 60 impala studied. These animals were taken from the herds from January through July. Month by month evaluations of the total endoparasite and ectoparasite burdens are to be carried out, and pertinent information will be included in a subsequent report.

Pathologic findings

Nodular abomasitis occurred predominantly among lambs, with no apparent sex predilection. The condition was most prevalent in the spring (September–November) and fall (March–May). The number and size of mucosal nodules varied considerably. In some cases, more than 50 discrete nodules could be seen distributed throughout the abomasal mucosa (Fig. 1). Occasionally, coalescence of adjacent nodules produced irregular areas of mucosal thickening. Larger nodules measured 5 mm in diameter and were roughly circular, cream colored and raised 1 to 2 mm presenting a domelike appearance; many were slightly umbilicated (Fig. 2). It was often possible to extract the adult worms (usually 1 male with 1 or more females) by dissecting through nodules. The lambs with numerous nodules did not differ noticeably in physical condition from those with minimal or no abomasal involvement. Moreover, they showed no evidence of diarrhea, having well formed fecal pellets in their rectums.

Microscopically, individual nodules consisted of focal areas of mucosal hyperplasia (Fig. 3). The simple columnar mucus-secreting epithe-

TABLE I. *Mean intensity of 4th-stage larvae and adult Longistrongylus sabie listed by age groups/sex of host (ranges in parentheses).*

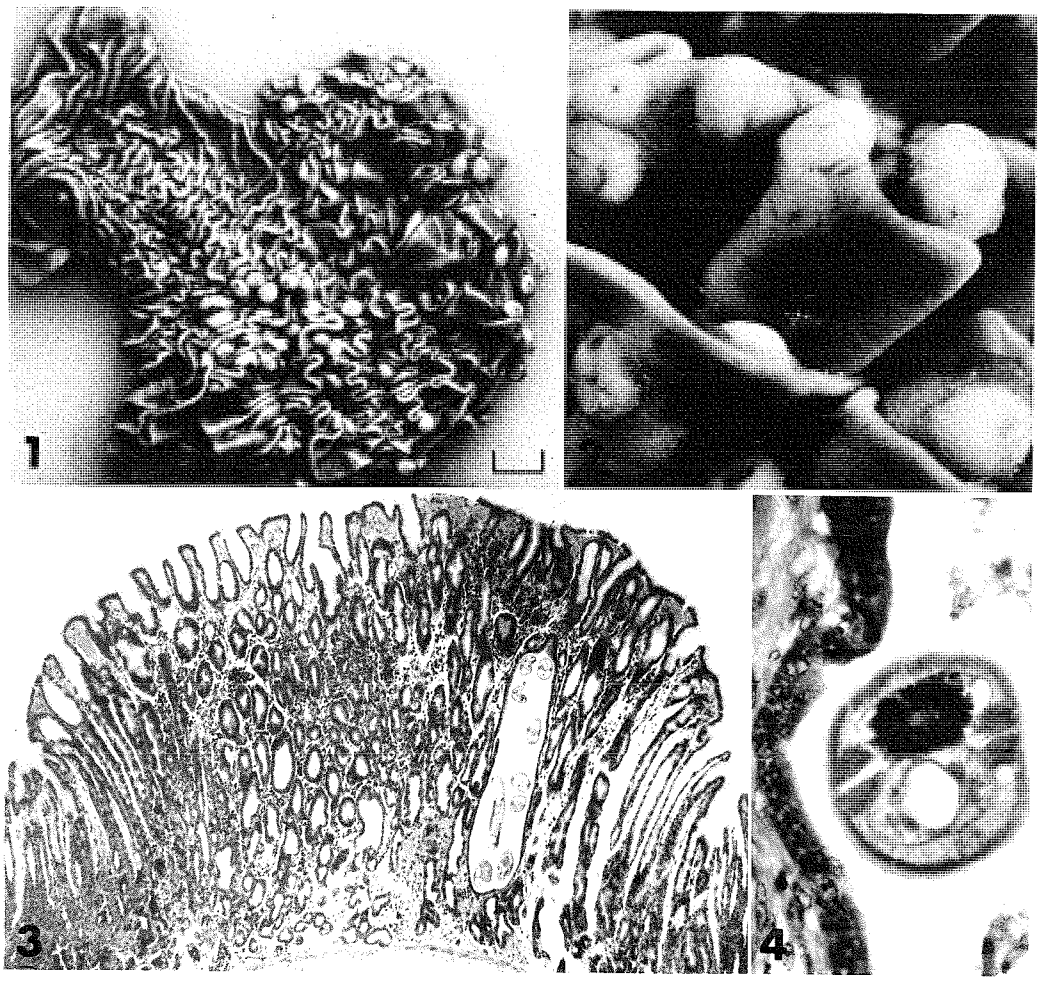
Impala age/sex	No. of impala	*Longistrongylus sabie*	
		No. of 4th-stage larvae	No. of adults
1–7 mo. (males and females)	15	11 (0–50)	55 (0–200)
13–19 mo. (males)	15	33 (0–130)	166 (0–450)
Adults (male)	22	66 (0–540)	97 (0–360)
Adults (female)	8	84 (0–430)	92 (0–410)

lium of the surface and gastric pits was not affected, but was elevated due to hyperplasia of the gastric glandular epithelium beneath and edema and inflammatory cell infiltration of the lamina propria. Gastric glands were often slightly dilated and elongated, when compared to those of the mucosa adjacent to a nodule. Moreover, these glands were lined by closely arranged cuboidal to columnar epithelial cells with large vesicular nuclei containing one or more prominent nucleoli and notably basophilic cytoplasm. Numerous mitotic figures were seen in these epithelial cells throughout the length of the glands, and differentiation to chief or parietal cells, seen in abundance in the adjacent mucosa, was lacking. The lamina propria within nodules was infiltrated by plasma cells, lymphocytes and eosinophils and often had a slightly loose appearance suggestive of mild edema. Frequently, oblique and cross sections through nematode parasites were seen in dilated glands within the nodules (Fig. 3). Morphologic features consistent with those of the trichostrongyles and the presence of 23 to 31 symmetrically arranged longitudinal cuticular ridges suggested the parasite to be *Longistrongylus sabie* (Fig. 4). Fibrosis of the lamina propria adjacent to parasitized glands was a frequent finding. Many nodules were devoid of worms but contained eggs, mucus and/or cellular debris located in central dilated and arborescent glandular spaces that were lined by undifferentiated or mucus-secreting epithelial cells. The submucosa beneath mucosal nodules was unaffected.

DISCUSSION

Nodular abomasitis caused by *L. sabie* appears to be a common, benign parasitic condition of impala lambs in the Kruger National Park. Similar lesions are associated with other *Longistrongylus* spp. in antelope of Kenya (Karstad, pers. comm.; Pester and Laurence, 1974). Its seasonal occurrence has been related to the resumed development of arrested fourth-stage larvae (Horak, 1978). The biannual occurrence noted in this study differs from that found in a similar study of impala in the northern Transvaal, where nodular abomasitis was seen only in November and December (early summer) after a prolonged period of arrested development from the fall to spring (Horak, 1978). It was suggested in that study that the normal rapid development of *L. sabie* through larval stages to adulthood, which occurs throughout the remainder of the year, is not associated with the formation of mucosal nodules. Although there may be an association between nodule development in impala lambs and release from arrested larval development, the factor(s) responsible for mucosal hyperplasia have not been identified.

The similarity of the lesions in impala to lesions in cattle and sheep with parasitic abomasitis caused by *Ostertagia* species is apparent and not unexpected, since both *L. sabie* and *Ostertagia* species are morphologically very similar and are included in the tribe Ostertagiea (Gibbons, 1977). The nodules produced by *L. sabie* are generally larger than those caused by *Ostertagia* species and are macroscopically discrete. Although they were larger, abomasal nodules never numbered more than 100 in the impala studied, reflecting the relatively small worm burdens (mean of 66 per lamb). Worm burdens of domestic ruminants with clinical ostertagiosis are hundreds of times greater (Raynaud and Bouchet, 1976); therefore, even though the resulting mucosal nodules are smaller, the entire mucosal surface is usually involved, presenting a "morocco leather" appearance. Histopathologically, the secondary nodules described in ostertagiosis (Jarrett, 1966) are identical to the nodules of *L. sabie,* with a substantial loss of well-differentiated parietal cells. An important development in the pathogenesis of ostertagiosis is the increase in pH of the abomasal contents at the time the adult worms emerge from the mucosa (Jarrett, 1966; Ritchie et al., 1966; Jubb and Kennedy, 1970; Thomson, 1978). The rise in pH is the result of hypochlorhydria caused either by a destruction/lack of differentiation of parietal cells in response to the presence of the worm in gastric glands (Jarrett, 1966; Ritchie et al., 1966), or, according to a recent study, a chemical associated with the parasite which inhibits acid secretion



FIGURES 1–4. **1.** Nodular abomasitis in an impala lamb. Larger nodules are 5 mm in diameter. Bar = 1 cm. **2.** Typical umbilicated nodules. **3.** Low magnification of a nodule showing hyperplasia of the glandular epithelium and one or more parasites within a dilated gland. H&E stain; ×20. **4.** Cross section of an adult *L. sabie* within a mucosal nodule. The cuticular ridges aid in identifying the parasite as belonging to the genus *Longistrongylus*. H&E stain; ×1,000.

(Eiler et al., 1981). A slightly acid or neutral abomasal environment is thought to enhance the survival of emerging adult parasites but also favors proliferation of bacteria, which closely parallels the period of severe diarrhea and loss of protein into the abomasal lumen in clinical ostertagiosis (Jarrett, 1966). It is possible that lambs experiencing a degree of abomasal disease which exceeds a threshold may sicken and die rapidly or become victims of predation, thus being unavailable for study and introducing a bias in our evaluation of the significance of this condition. However, the good condition and lack of diarrhea in impala lambs with nodular abomasitis as well as the burgeoning impala herds in the KNP, suggest that abomasal disease caused by *L. sabie* is not a serious problem at present in the Park. It must be assumed that most impala lambs do not develop serious hypochlorhydria as a result of *L. sabie*-related nodular abomasitis; the most reasonable explanation for this is the relatively low worm burdens. A balance between host physiology and parasitic numbers/pathogenicity has apparently evolved which favors the survival of both host and parasite.

The initial infection experienced by most lambs seems to produce a tolerance in later years; few yearlings or adults had abomasal nodules, al-

though on the average they harbored more of the parasites than did the lambs (Table I). The nature of this mucosal tolerance should be investigated further.

It should be appreciated that although nodular abomasitis is not a serious disease among the impala of the KNP at present, changes in the environment could destabilize the host/parasite relationship. For instance, a prolonged drought might force herds to concentrate near remaining water. Under such conditions, several parasitic diseases including nodular abomasitis may increase dramatically in significance.

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The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Air Force or the Department of Defense.

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HEPATIC LESIONS ASSOCIATED WITH *COOPERIOIDES HEPATICAE* (NEMATODA: TRICHOSTRONGYLOIDEA) INFECTION IN IMPALA (*AEPYCEROS MELAMPUS*) OF THE KRUGER NATIONAL PARK

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ABSTRACT: Intrahepatic biliary lesions were observed in two of 12 lambs, seven of 12 yearlings and 10 of 25 adult impala (*Aepyceros melampus*) surveyed in the Kruger National Park, Republic of South Africa. Lesions were associated with the nematode *Cooperioides hepaticae*, a trichostrongyloid parasite that inhabits the bile ducts of impala, and ranged from a mild chronic-eosinophilic cholangitis to foci of florid hyperplastic cholangitis with duct ectasia. The latter almost always contained viable worms and, after the worms died, the lesions appeared as foreign-body granulomas. Infection was acquired early in life; severe lesions were seen most frequently in yearlings. Adults were less severely infected, which suggested an acquired immunity. Although the incidence of infection was high, cooperiiasis did not appear to be a serious herd-health problem at the time of this study.

Key words: Intrahepatic biliary lesions, pathology, nematode, *Cooperioides hepaticae*, impala, *Aepyceros melampus*, field survey.

INTRODUCTION

During a study of parasitic infections and pathologic changes in free-living impala (*Aepyceros melampus*) of the Kruger National Park (KNP), Republic of South Africa, it was noted that a large number of the antelope had varying degrees of intrahepatic biliary lesions due to infection by a trichostrongyloid nematode (*Cooperioides hepaticae*). Species of the genus *Cooperioides*, with the exception of *C. hepaticae*, are intestinal parasites found in domestic sheep and several types of antelope in eastern and southern Africa (Daubney, 1933; Messer, 1952; Round, 1968). Like members of the genus *Cooperia*, *C. hepaticae* are generally considered of minor pathologic significance unless present in large numbers in combination with other trichostrongyles, and/or in association with a poor nutritional condition. *Cooperioides hepaticae* is the only species of the genus with adults occupying an extraintestinal location. Moreover, the species appears to be the only trichostrongyloid that inhabits tissues other than those of the gastrointestinal tract (Daubney, 1933; Ortlepp, 1938; Soulsby, 1968).

We have conducted a systematic study of endoparasite and ectoparasite frequencies and numbers and associated pathologic changes of impala in the southern part of the KNP. Impala are the most abundant antelope in the eastern Transvaal. It has become evident from our work that *C. hepaticae* infection is very common among these impala and can cause substantial lesions. This report describes gross and microscopic lesions produced by *C. hepaticae* in the livers of impala and discusses the host response to the infection. Other aspects of this parasite such as taxonomic morphology and life cycle are not addressed except in acknowledgement of the facts that the taxonomy of the genus *Cooperioides* has been reviewed (Gibbons,

TABLE 1. Intensity of *Cooperioides hepaticae* infection in impala by sex and group.

Age group	Number of animals			Mean intensity (range)	
	Female	Male	Total	Female	Male
Lambs (0–6 mo)	3	9	12	67 (0–265)	71 (6–295)
Yearlings (12–18 mo)	3	9	12	230 (15–680)	226 (10–683)
Adults (over 24 mo)	7	18	25	32 (0–230)	31 (8–225)

1978) and that the life cycle of *C. hepaticae* has not been described but is thought to be direct.

MATERIALS AND METHODS

All impala in this study were collected at the Kruger National Park, Republic of South Africa (25°12′ to 24°24′S and 31°36′ to 32°02′E). The details of the park habitat are described briefly by Krecek et al. (1987).

Impala of the appropriate age were selected at random, shot through the neck with a high caliber rifle and then exsanguinated by severing the major vessels in the neck. The majority of the impala studied were males from three age groups—1 to 6 mo (lambs), 12 to 18 mo (yearlings), and over 2 yr (mainly prime adults). A smaller number of females of similar ages were also surveyed.

Tissues taken for histopathologic studies were fixed in 10% neutral buffered formalin and prepared and stained according to commonly accepted methods. Hematoxylin and eosin as well as Masson's trichrome stains were applied to the sections studied. The entire liver of each animal was macerated and the number of *C. hepaticae* tabulated for each. Liver sections containing *C. hepaticae* were deposited at the Armed Forces Institute of Pathology (Washington, D.C. 20306, USA; Accession Number 2168059) and by the U.S. National Parasite Collection (Animal Parasitology Institute, USDA, Beltsville, Maryland 20705, USA; Accession Number 80361).

RESULTS

The data on the intensity of infection in the impala are summarized in Table 1.

The general physical appearance of the animals ranged from poor to very good. Those in poor condition were usually yearlings or young adults, and necropsy of these animals revealed heavy parasite infections, with the lesions of *Pneumostrongylus calcaratus* in the lungs and of *C. hepaticae* in the liver being most notable. Impala <6 mo old did not have gross lesions associated with either of these parasites. Prime adults were generally in good condition, although many had macroscopic evidence of both lungworms and biliary cooperiiasis, but to a lesser degree than did the yearlings. The prevalence of infection by both parasites appears to be the same for male and female impala.

Lesions associated with *C. hepaticae* were limited to the liver. In mildly infected animals (those with <100 nematodes in the liver), macroscopic changes were subtle, consisting of a slight prominence of the portal tracts within the liver. These areas offered increased resistance when the parenchyma was incised, suggesting mild portal fibrosis. In several animals, the bile duct tapeworm *Stilesia hepatica* was present along with *C. hepaticae*. Gross lesions specific for *C. hepaticae* were observed in heavily infected impala (those having >200 nematodes in the liver); these animals were most often yearlings. In addition to prominent portal fibrosis, focal yellow-white spherical to ellipsoidal nodules were seen within the liver. Nodules were as large as 1 × 2 cm and often could be seen at the surface of the liver but were present throughout the parenchyma as well. A heavily infected liver would contain ≥eight nodules, which, when incised, revealed a substantial fibrous capsule encompassing a greatly thickened and dilated bile duct containing many reddish nematodes and a cloudy fluid (Fig. 1). Nodules of similar size were occasionally found to be firm and, on incision, to contain a caseous material that had a gritty consistency. Nematodes were not seen in these lesions.

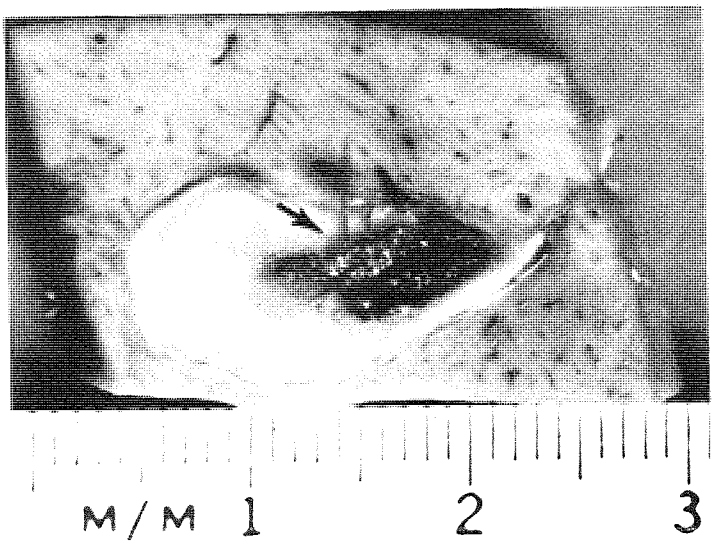

FIGURE 1. Sagittal section through an active *Cooperioides hepaticae* nodule. There are many reddish worms (arrow) in the greatly thickened and sacculated bile duct. Formalin-fixed specimen.

Microscopically, animals with early, or mild, infections showed a chronic-active eosinophilic cholangitis with an increase in periportal fibrous tissue. Eosinophils and plasma cells were the predominant inflammatory cells in the lamina propria, and the biliary epithelium was often noted to be slightly hyperplastic. Portal veins were frequently dilated and congested. In addition to these changes, severely infected animals had focal areas of moderate to extreme cholangiectasia with florid biliary epithelial hyperplasia often resulting in papillary structures which extended into the sacculated bile duct lumens. Within these lumens were adult *C. hepaticae*, their eggs, and mucoid material mixed with cellular debris (Fig. 2). The epithelium at the tips of the papillary structures often showed a marked squamous metaplasia; and eosinophils, as well as mononuclear cells with variably sized eosinophilic globules in their cytoplasm, were frequently seen within the epithelium. Remnants of both these cells also could be seen in the luminal debris. Connective tissue beneath the epithelium was infiltrated by many plasma cells and eosinophils, and these inflammatory cells, in addition to aggregates of lymphocytes, were prominent in the surrounding fibrous tissue. The hepatic parenchyma adjacent to these nodules appeared compressed, attesting to the expansile nature of the lesions. The firm nodules having no nematodes consisted of a central core of eosinophilic material containing mineralized debris and were surrounded by a zone of multinucleated giant cells and macrophages. At the periphery were inflammatory cells including eosinophils and plasma cells, but macrophages and lymphocytes predominated, and the development of lymphoid follicles was noted (Fig. 3). Biliary epithelium was not evident in these lesions.

DISCUSSION

The genus *Cooperioides* was originally described by Daubney (1933), who acknowledged the close relationship between it and the genus *Cooperia*. Various species have been recovered from the intestines of domestic sheep, springbok (*Antidorcas marsupialis*), Thomson's gazelle (*Gazella thomsoni*) and impala (*Aepyceros melampus*) (Daubney, 1933; Round, 1968); however, Ortlepp (1938) described *Cooperioides hepaticae* found "in small nod-

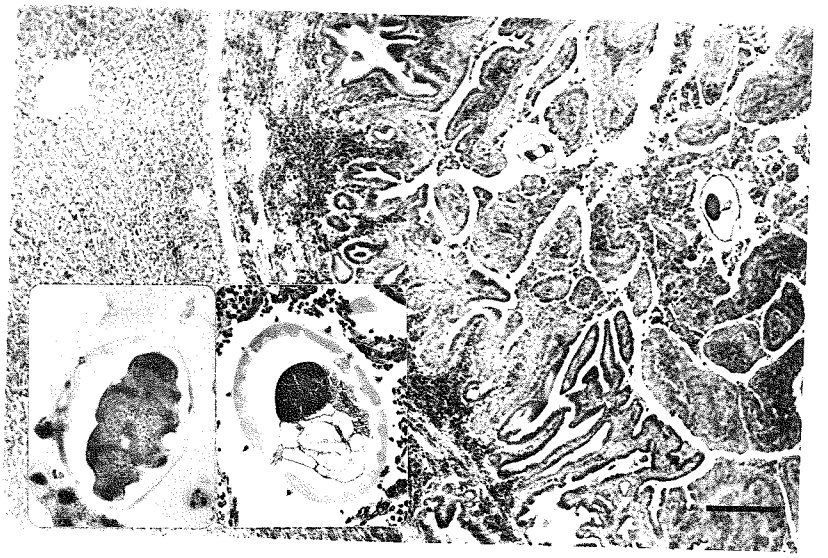

FIGURE 2. An active parasitic nodule. Note the papillary hyperplasia of biliary epithelium with superficial squamous metaplasia and the nematodes in cross section. H&E (bar = 1 mm). Insets: left, *Cooperioides hepaticae* egg measuring 30 × 40 μm; right, mature *C. hepaticae* in cross section.

ules in the terminal portions of the bile ducts" of an impala from the northern Transvaal. Messer (1952) described an identical nematode from the intrahepatic bile ducts of an impala in the eastern Transvaal. He referred to this parasite as "*Cooperia hepaticae*" and briefly described the pathologic changes associated with it (Messer, 1952). Mugera (1969) provided brief descriptions of the lesions produced by *C. hepaticae* and the tapeworm *Stilesia hepatica* in the liver of impala but it appears that he confused the lesions produced by the two species. Basson et al. (1971) mentioned *Cooperioides hepaticae* as a parasite of the biliary ducts of impala from the KNP, identified it as a "bile duct hookworm" and grouped it with true hookworms such as *Grammocephalus clathratus*, the bile duct hookworm of the African elephant (*Loxodonta africana*).

Gross and microscopic observations of *C. hepaticae* lesions and the intensity of infection suggest that there is a progression of events. An initial infectious phase proceeds through the development of active parasitic nodules and peak intensity. This is followed by subsequent destruction of adult nematodes and resolution of nodules. Mild eosinophilic cholangitis with a resulting increase in portal connective tissue occurs during the initial stages. These changes are first recognized at about 6 mo of age and probably reflect the migration of larvae and/or adult worms up the biliary tree. Similar lesions could occur as a result of the bile duct tapeworm *S. hepatica*; however, the occurrence of cholangiectasia and nodule formation is specific for biliary cooperiiasis. Although initial infection probably occurs early in life, substantial macroscopic lesions (parasitic nodules) are not observed until the animals approach 1 yr of age. Active parasitic nodules incite an immune response characterized by large numbers of plasma cells and eosinophils. Eosinophils and mononuclear cells with eosinophilic cytoplasmic globules were seen within the epithelium and in the luminal debris of parasitic nodules, and it seemed that both types of cells, as well as the plasma cells in the lamina propria, were intimately involved in the host response to the parasites. The cytoplasmic globules in the mononuclear cells appeared similar to Russell bodies, and their

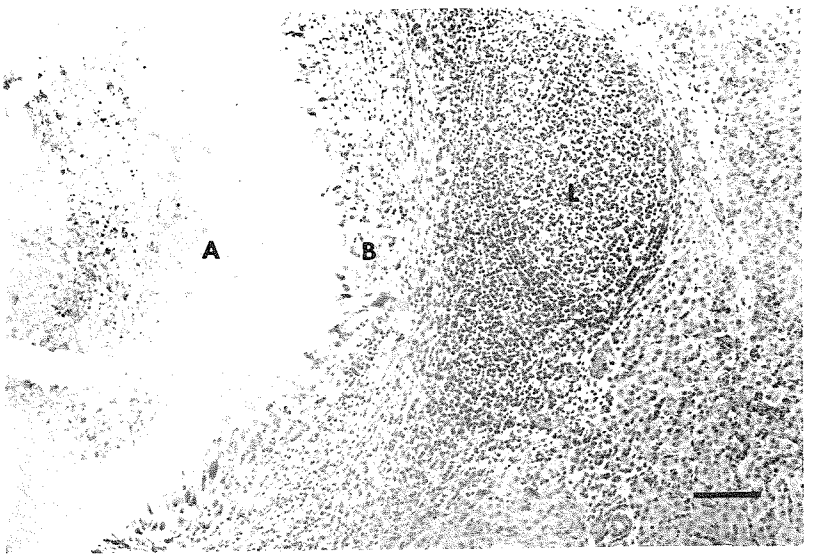

FIGURE 3. A resolving parasitic nodule with central core of partially mineralized eosinophilic debris (A), a zone of multinucleated giant cells and macrophages (B), and a peripheral zone predominantly of lymphocytes and macrophages with lymphoid follicle development (L). Note that biliary epithelium is not evident. H&E (bar = 250 μm).

possible role in the transport of antibody across the epithelium is the subject of continuing study. It has been demonstrated recently that eosinophils, in the presence of specific antibodies, can function as effector cells in the destruction of metazoan parasites (Zucker-Franklin, 1978). Once the adult worms are dead, the lesions appear to resolve as foreign-body granulomas. Macrophages and multinucleated giant cells predominate and lymphoid follicles are formed. The hyperplastic biliary epithelium observed in active nodules is obliterated in the process of resolution. Although many adult impala are infected, these tend to have fewer active parasitic nodules, and the prevalence of infection is lower than that for the yearlings. One can deduce from these findings that most adult impala acquire at least a partial immunity.

The florid biliary hyperplasia and metaplasia seen in active nodules is of interest. Apparently, biliary stimulation results from some parasite-associated factor(s) similar to that described in trematode-induced biliary hyperplasia observed in domestic ruminants (Isseroff et al., 1977).

Cooperioides hepaticae infection is one of the most common extraintestinal parasitic infections of impala in the KNP. Substantial lesions can accrue as a result of severe infection, particularly in yearlings, and it is highly probable that such lesions adversely affect the health of animals. However, it is doubtful whether *C. hepaticae* alone is a primary factor limiting the population of the large impala herds of the eastern Transvaal. Impala are the most numerous antelope in the KNP, despite a high prevalence of infection. Conversely, it is reasonable to assume that biliary cooperiiasis, in concert with lung worms, gastrointestinal helminths and ectoparasites could cause excessive losses in certain environmental settings. The depletion of impala herds in the eastern Transvaal during the dry seasons of 1949 and 1950 is an example (Messer, 1952). Game farmers and conservationists should be familiar with the conditions that tend to en-

hance these various parasitic diseases so that appropriate preventive measures can be applied when feasible.

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Parafilariosis in African buffaloes (*Syncerus caffer*)

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ABSTRACT

KEET, D.F., BOOMKER, J., KRIEK, N.P.J., ZAKRISSON, G. & MELTZER, D.G.A. 1997. Parafilariosis in African buffaloes (*Syncerus caffer*). *Onderstepoort Journal of Veterinary Research*, 64:217–225

This is the first report on the occurrence of *Parafilaria bassoni* in the African buffalo (*Syncerus caffer*). Previously this parasite has been recorded only in springbok (*Antidorcas marsupialis*) in Namibia. Haemorrhagic perforations (bleeding points), the usual lesions seen in infected animals, were caused by gravid female parasites ovipositing embryonated eggs. These lesions occurred mainly on the dorsal and lateral sides of buffaloes. Complications of these lesions developed in a small number of buffaloes because of secondary bacterial infection [subcutaneous abscesses (3/178)] and as a consequence of a localized Type 1 hypersensitivity [large cutaneous ulcers (7/178)]. Red-billed oxpeckers (*Buphagus erythrorynchus)* appeared to play an important role in the epidemiology of this parasite as well as in the pathogenesis of the lesions. They reduced the likelihood of spread by ingesting blood containing embryonated eggs, and caused the development of large ulcers by feeding on superficial necrotic skin. From the results of an ELISA test it was determind that *P. bassoni*-infected buffaloes occur throughout the Kruger National Park complex, with a seroprevalence of approximately 34%.

Keywords: African buffalo, *Antidorcas marsupialis, Buphagus erythrorynchus*, eosinophilic arteritis, Kruger National Park, *Parafilaria bassoni*, red-billed oxpeckers, springbok, *Syncerus caffer*

INTRODUCTION

Parafilariosis is a condition commonly seen in cattle (Pienaar & Van den Heever 1964) and rarely in equids, in South Africa (Ortlepp 1962a); it has not been reported in other ruminants. During 1992, large ulcerated skin lesions on African buffaloes, *Syncerus caffer*, were reported by game rangers from the Sabi Sand Game Reserve (SSGR), a privately owned reserve adjacent to the Kruger National Park (KNP). The ulcers occurred mainly on the dorsal and dorsolateral aspects of the thorax of adult animals and resembled those caused by *Stephanofilaria* spp. in cattle and buffaloes (*Bos bubalis*) in India (Sharma Deorani 1965; Patnaik & Roy 1967). Their occurrence was strictly seasonal, lesions appearing in about November and disappearing towards the middle of March in the following year, thus suggesting insect transmission. Initially, the lesions are inconspicuous bleeding points arising from small cutaneous ulcers, similar to those caused by *Parafilaria bovicola* in cattle. Red-billed oxpeckers, *Buphagus erythrorhynchus*, were attracted to the bleeding points to feed on the exuding blood, simultaneously removing embryonated eggs.

The condition has long been suspected to occur in African buffaloes in the KNP because of the presence

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of non-specific lesions in their subcutis and its musculature, but parasites were never found (Young & Van den Heever 1969). Basson, McCully, Kruger, Van Niekerk, Young & De Vos (1970) necropsied 100 buffaloes in the KNP and found some of them to have an eosinophilic cellulitis and panniculitis, but did not recover any adult nematodes. They did, however, find sheathed microfilariae in the infra-orbital skin and suggested that these could be either *Stephanofilaria* spp. or *Parafilaria* spp. or both (Basson *et al.* 1970).

This paper is a report on the cause and pathogenesis of the lesions, as well as on the distribution and prevalence of parafilariosis in buffaloes in the greater KNP complex.

MATERIALS AND METHODS

Study area

The KNP is a large reserve, about 20 000 km^2 in extent. It is situated in the north-eastern corner of South Africa, adjoining Zimbabwe in the north and Mozambique in the east (Fig. 1). The vegetation is diverse and several types and subtypes of Lowveld, Mopane veld and Bushveld are recognized (Acocks 1988). The SSGR is a privately owned and managed reserve on the western border of the KNP (Fig.1). It is approximately 570 km^2 in size and forms part of the greater KNP complex. The vegetation is classified as Lowveld (Acocks 1988).

Animals

A buffalo herd in the SSGR was monitored from October 1993 to April 1994. One dominant female in the herd was identified and immobilized according the method described by Bengis (1993). A radio transmitter (Telonics, 932E Impala Avenue, Mesa, Arizona, USA) was fitted around her neck. The herd was tracked on the ground twice a week with the aid of a radio receiver (Yaesu 2M FT-290R 2, Yaesu C.P.O. Box 1500, Tokyo, Japan) and an H-antenna. When the herd was difficult to find, a light aircraft was used to track it.

Buffaloes showing lesions suggestive of parafilariosis were immobilized and a skin-biopsy specimen was taken. When the lesion was a bleeding point, an incision 20 mm in length was made in a ventral direction, starting 30 mm above the lesion. An index finger was used as a probe to locate the granuloma (Fig. 2a) caused by the nematode in the subcutis. On locating the granuloma, the incision was extended in that direction so as to include the granuloma in the biopsy specimen. Tissue blocks, 15 mm square and containing all the layers of the skin, were removed from the edge and centre of ulcerated lesions. In all instances the biopsy wounds were packed with a sulphonamide-containing ointment (Acrisulph, Kyron Laboratories, 84 Main Reef Road, Benrose, 2094, South Africa) and sutured with vertical interrupted mattress stitches and no. 1 nylon. The biopsy specimens were fixed in 10% buffered formalin. Tissue blocks of the specimens were processed routinely, embedded in paraffin wax, sectioned (4–6 µm thick) and stained with haematoxylin and eosin for light microscopy.

Buffaloes were examined from a vehicle, by binoculars, and all lesions were recorded on a silhouette diagram of a buffalo. The lesions of each buffalo were plotted on a separate diagram. The behaviour of oxpeckers towards lesions was monitored and categorized as follows: ingestion of crusts around bleeding points; the extent of beak penetration into a bleeding point; attention given to large cutaneous ulcers; the number of oxpeckers feeding on a single ulcer; the frequency at which buffaloes fended off the oxpeckers; the reaction of the oxpeckers to the behaviour of the buffaloes; and the way in which buffaloes attempted to evade the oxpeckers.

Parasites

At first, attempts were made to remove the worms surgically from the subcutis of immobilized buffaloes. Then two buffaloes were killed at an interval of three weeks. The animals were skinned and the worms dissected from the bright green granulomas in which they were present (Fig. 2b) Worms collected in this manner were fixed in cold, 70% ethyl alcohol.

A third buffalo was killed during January 1994 and its skin cut into pieces of approximately 40 x 70 cm. These were placed in normal saline at 40 °C on expanded metal sheets in plastic trays and incubated for 24 h. The saline was replaced every 4 h; each volume of saline was sieved through a sieve (150 µm apertures). The residue was inspected visually and the worms removed and fixed in boiling 70% ethyl alcohol. The remaining residue was washed into a container, fixed by heating to 60 °C and preserved by adding 10% buffered formalin.

A total of 15 specimens consisting of fresh blood and blood crusts from bleeding points of 15 buffaloes were collected in glass tubes. Water was added and the mixture was left for a period of 12 h for haemolysis to occur. The resulting suspension was centrifuged for 4 min at 3 000 *g*. The supernatant was discarded and an unstained smear prepared from the residue. This preparation was examined under a standard microscope at 50x magnification.

Seroprevalence

Serum samples collected from 184 buffaloes from 11 localities throughout the KNP complex, together with nine positive and six negative controls, were submitted for diagnosis. Each locality represented one

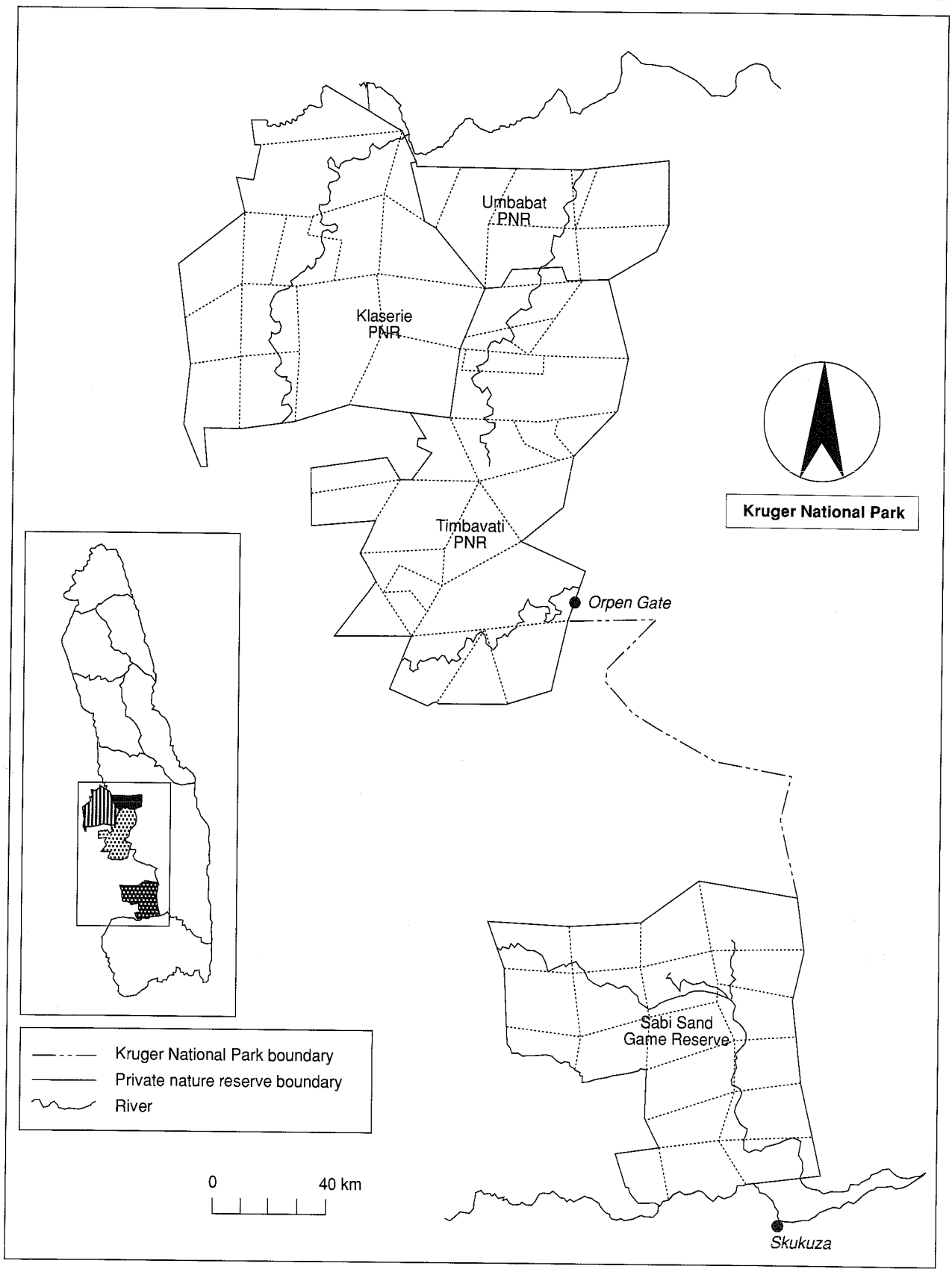

FIG. 1 Map of the Sabi Sand Game Reserve and other private nature reserves in relation to the Kruger National Park. Together they form the greater KNP complex

cluster and from these clusters samples were picked at random, with replacement. Positive control samples were obtained from animals in the SSGR and their status confirmed by the presence of the parasites as well as histopathology. Negative control samples were obtained from buffaloes born and bred in confinement. They were removed from the cows at about 3 months of age. These animals were regarded as negative as they had never shown bleeding points and had never had contact with free-ranging buffaloes. An ELISA test as described by Voller, Bidwell & Bartlett (1980) was employed. This test was developed for polypeptides of *P. bovicola* exoantigens with molecular masses of 41 and 36 Kda (Sundquist, Bech-Nielsen & Zakrisson 1989). Antigens were purified and characterized by chromatofocussing to eliminate cross-reactivity with antigens produced by concurrent nematode infections (Sundquist *et al.* 1989).

RESULTS

Animals

The lesions seen on the animals were categorized into three macroscopically discernible types that occurred at different times during summer. The primary lesion, the bleeding point or haemorrhagic perforation (Fig. 2c) was observed from the beginning of November to the beginning of February. Two secondary lesions developed subsequently: subcutaneous abscesses (Fig. 2d), which were seen from the middle of December to the middle of January; and large cutaneous ulcers (Fig. 2e) which appeared from about the middle of January and had healed by the middle of March, leaving a conspicuous scar (Fig. 2f).

The bleeding points were equally distributed on the left and right sides of the buffaloes (81 points on the left side and 84 on the right). The dorso-ventral distribution, however, was not uniform: 15,85% occurred dorsally, 58,53% laterally and 25,6% ventrally. The cranio-caudal distribution was even less uniform, with 83% of the lesions recorded cranial to the loins. Of these, 20,6% were seen on the neck, 23,6% on the shoulders, 39,4 % on the ribs, 4,5% on the loins and 11,5% on the hindquarters. No lesions were encountered on the head or the tail.

Each bleeding point was located centrally in a poorly circumscribed swelling raised about 3–5 mm above the surface of the skin. These openings were about 1 mm in diameter and occluded by a coagulum of serum in which the heads of the female nematodes were embedded, though not visible externally. Intermittent haemorrhaging from the bleeding points occurred for up to 2 d from the time of their first appearing. The volume of blood seeping from these bleeding points could not be estimated as it was rapidly consumed by oxpeckers. On visual inspection of the subcutis, the female nematodes were found in their migration tracts which were enveloped in an elongated, brilliant green granuloma, measuring about 20 x 3 mm. The area immediately surrounding the granuloma appeared unaffected macroscopically (Fig. 2a).

Histologically, sections cut transversely through the migration tract, revealed a central core of necrotic eosinophils, cellular and fibrin. A pronounced granulomatous reaction consisting of epithelioid cells in palisade formation and a dense infiltrate of eosinophils, plasma cells and lymphocytes, together with oedema and fibroplasia, surrounded the necrotic core. Vascular lesions that occurred in the surrounding tissue included chronic proliferative phlebitis and a perivascular eosinophil and lymphocyte infiltration.

The subcutaneous abscesses were well-defined swellings, measuring up to 60 x 60 mm. The swellings contained circumscribed cavities filled with necrotic debris. The adjacent subcutis was pale green and slightly oedematous. Three out of 178 buffaloes had such abscesses as a complication.

Histologically, the walls of the abscesses consisted of granulation tissue, containing vast numbers of eosinophils. Fibrin and large numbers of erythrocytes, eosinophils and neutrophils accumulated in their centre. Vascular lesions in the surrounding tissue included eosinophilic arteritis, eccentric endarterial fibrosis, fibrinoid necrosis of the vessel wall, proliferative endarteritis and recanalization of thrombi.

Large cutaneous ulcers developed, mainly on the dorso-lateral area immediately behind the shoulders, but were also to be seen on the top of the hump of infected buffaloes. One ulcer was noted on the loin and two on the hindquarters. Ulcers were seen on seven of 178 buffaloes and only on adult animals. They varied in size from 50x40 mm to 300 x 200 mm, were well-circumscribed, had ragged, elevated edges and an irregular base. Even the largest lesions developed rapidly, the entire process taking about 10 d. During the early phase of development, intermittent haemorrhage occurred from the ulcerated areas, but this was caused mostly by oxpeckers feeding on the wound. The adjacent subcutis had a yellowish-green tinge, with signs of haemorrhage and oedema. Pruritus was never seen.

Microscopically, there was a sudden transition from normal to ulcerated skin. A marked acanthosis occurred at the junction, which progressed to full-thickness necrosis with ulceration. The ulcerated surfaces were covered by a scab consisting of fibrin containing numerous eosinophils. Below the scab a prominent layer of immature granulation tissue containing masses of eosinophils was seen. The dermis was relatively unaffected, with the exception of marked perivascular infiltrates of eosinophils. The inner layer

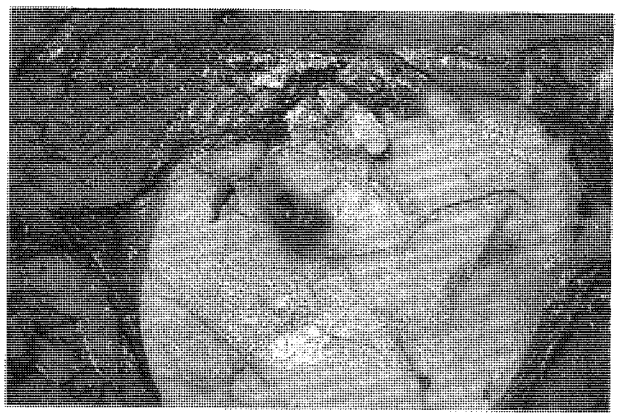

FIG. 2a A brilliant green subcutaneous granuloma containing a gravid female filariid

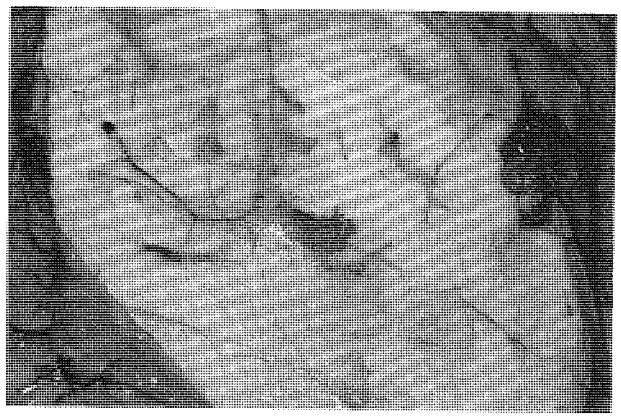

FIG. 2b An exposed female filariid

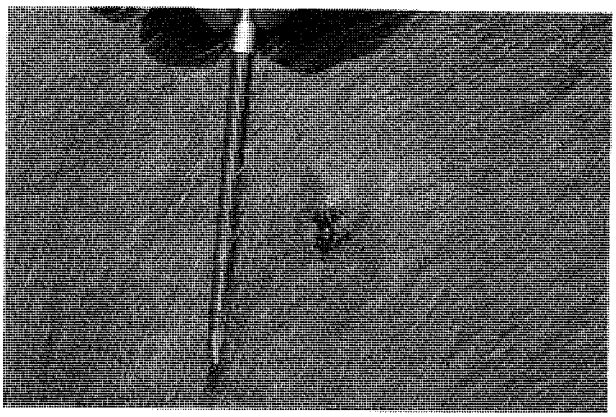

FIG. 2c A typical bleeding point

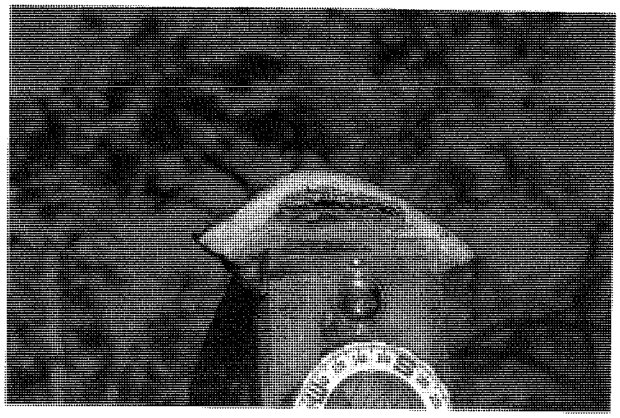

FIG. 2d A subcutaneous abscess

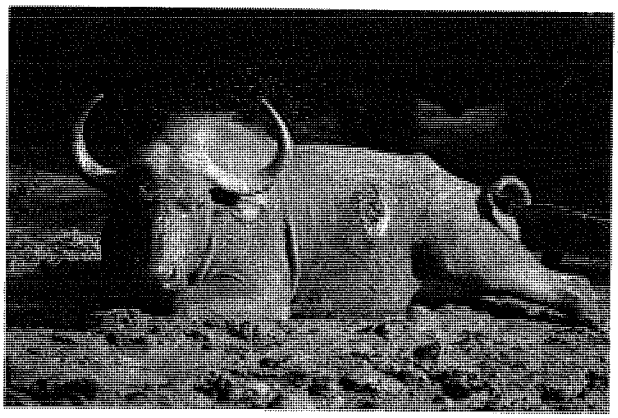

FIG. 2e A large cutaneous ulcer

FIG. 2f A conspicuous scar that remains visible for a long time

of the dermis and the subjacent subcutaneous interface showed severe oedema and marked vascular involvement and contained numerous eosinophils. Mostly arteries were involved, a marked eosinophilic vasculitis, segmental medial hyperplasia and extensive recanalization of thrombi being the most common lesions present. Marked endothelial proliferation occurred in the affected vessels and some showed mild villous endarteritis. These lesions were accompanied by pronounced endothelial hyperplasia and subendothelial oedema, as well as eccentric, chronic, eosinophilic endophlebitis and early villous

endophlebitis. In addition to the eosinophils, the perivascular reaction also contained plasma cells and a few lymphocytes. In the central area of the lesion eosinophilic granulomas occurred deeper in the dermis. These exhibited the Splendore-Hoepplé phenomenon, the eosinophilic mass containing necrotic nematode fragments.

All the cutaneous ulcers healed spontaneously towards the beginning of March, leaving conspicuous scars characterized by greyish-white alopecic areas sometimes with marked hyperkeratosis that remained visible for years.

Microscopically, the regenerated epidermis covering the large ulcers, contained only a few hair follicles, sebaceous glands and dilated sweat glands. Excessive keratinization was a constant feature. The outer dermis showed few changes, while the inner dermis consisted of maturing connective tissue. Vascular lesions were advanced and consisted of marked, eccentric, medial and endothelial hyperplasia and hypertrophy. Vascular occlusion and angiogenesis in close association with affected blood vessels were also seen.

Oxpeckers were attracted to bleeding points and ulcers. Wet streaks and dry crusts were rapidly ingested with scissor-like movements of their beaks. Occasionally, they inserted the full length of their beaks into a perforation to feed on the contents. Up to 12 oxpeckers were seen feeding simultaneously on one large ulcer. They also fed off biopsy wounds within minutes of the buffalo being revived after having been immobilized. The attacks were so vigorous that wound dehiscence occurred within 12 h. During daylight hours buffaloes continually fended off the oxpeckers. On occasion they were seen to use their horns up to seven times per minute to chase the birds off. White areas of alopecia developed on the buffaloes' shoulders owing to the trauma caused by the impact of their horns. The oxpeckers reacted aggressively when buffaloes tried to fend them off. They made growling sounds, puffed themselves up and assumed a threatening posture. The animals also tried to protect the ulcers from the oxpeckers by positioning their bodies close to shrubs so that the wounds would not be visible or accessible. Temporary relief from harassment was also obtained by wallowing in mud.

Parasites

Surgical removal of the nematodes from the subcutis of immobilized buffaloes was relatively unsuccessful and only fragments of female worms were recovered, while entire female worms were recovered only by dissection of the subcutaneous granulomas. Incubation of pieces of skin in saline was more successful and one entire male, two entire females and two entire early fourth-stage larvae were recovered. A total of 23 nematodes were collected by use of the various techniques. A considerable number of these nematodes were already dead at the time of collection.

The nematodes were identified as *Parafilaria bassoni* because of the similarity of the morphological characteristics with the species described by Ortlepp (1962b) from the orbital connective tissue of a springbok (*Antidorcas marsupialis*).

Embryonated eggs were found in all the blood crusts and wet blood streaks that were examined, but no free microfilariae. No attempt was made to count the number of eggs, but from one to seven eggs were noticed on each slide.

Seroprevalence

All six the negative controls tested negative and eight of the nine sera from buffaloes with parafilariosis, tested positive. These trends indicate a specificity of 100% and a sensitivity of 89%. Twenty-five of 71 (35,21%) male buffaloes tested positive and 37 of 113 (32,74%) females, a total of 62 of 184 (33,7%) animals examined. The youngest seropositive buffaloes were 2 years old. Twenty-three (12,5%) suspicious readings were recorded and 99 (53,8%) buffaloes tested negative.

DISCUSSION

Parafilariosis has been recorded in various Asian buffalo species (Srivastava & Dutt 1959; Patnaik & Pande 1963; Sahai, Singh & Varma 1973; Chauhan, Arora & Ahluwalia 1974) but never in African buffaloes. This is the first description of parafilariosis in African buffaloes. The lesions in buffaloes and the life cycle of the parasite resemble those seen in cattle parasitized by *P. bovicola*. Most typical lesions (bleeding points) heal without complications while some develop into abscesses or large distinct cutaneous ulcers. Red-billed oxpeckers appear to play an important role in the epidemiology of the parasite by feeding on blood containing embryonated eggs, and in the pathogenesis of lesions by removing superficial necrotic skin while feeding on large cutaneous ulcers, enlarging these ulcers. Infected buffaloes occur throughout the entire KNP complex and the prevalence of the infection is approximately 34%.

The external appearance of bleeding points seen on buffaloes is similar to that seen on cattle infected with *P. bovicola*, with the difference that oxpeckers may have removed most of the oozing blood on buffaloes. No difference was seen between the histopathology of the granulomatous reaction surrounding nematodes or the polymorphonuclear cell composition seen in buffaloes and that reported in cattle (Pienaar & Van den Heever 1964). Patnaik & Pande (1963) attributed complications of lesions caused by *Parafilaria* in Asian

buffaloes to contamination with bacteria when wallowing, and myiasis. Abscesses that developed from bleeding points caused the skin to slough, leaving an ulcer (Srivastava & Dutt 1959). In our study, none of the abscesses developed into ulcers and it appears that abscessation is an uncommon complication in African buffaloes. Concurrent myiasis was never observed and wound-breeding blowflies were not observed to feed on these lesions. The known vectors of *Parafilaria* are dung breeders and they do not deposit eggs or larvae in open wounds. However, myiasis was observed during the study period in wounds caused by lions on the withers of two buffaloes.

The histopathologic changes in the large ulcers and underlying tissues suggest that the pathogenesis of the lesion is a localized Type 1 hypersensitivity reaction of which the severity is enhanced by an excessive anamnestic response. At the subcutaneous site of dermal penetration the female, while ovipositing, becomes enveloped in an eosinophilic parasitic granuloma. The fact that the female is relatively stationary leads to the continuous local deposition of eosinophils involved with immune reactions directed against metabolic products secreted by the nematode. It appears that female parasites do not survive ovipositing, as the enveloping immune reaction develops into an impermeable mass which restricts and kills her, as evidenced by the number of dead and decaying worms that were recovered. Female worms may also be killed by oxpeckers when they feed on the bleeding points.

Oxpeckers are usually associated with the larger mammals in the KNP where they play a beneficiary role by removing especially ticks. Once they have settled on a buffalo, they move to an area where blood is present on the skin. They have been incriminated as aggressively and actively attacking and enlarging open cutaneous wounds (McLachlan & Liversidge 1982). In this study it appeared that they were causing haemorrhage by removing necrotic debris and damaging granulation tissue that developed in the ulcer, and then feeding on the blood. The carnivorous behaviour of this otherwise mutualistic companion of the buffalo must reduce the possibility of vectors becoming infected, which in turn could ultimately reduce the prevalence of *P. bassoni* in buffaloes in the complex.

The continuous presence of feeding oxpeckers annoyed infected buffaloes during the day, their action clearly inflicting pain, and they made it difficult for the buffaloes to feed or ruminate. In addition, since the majority of large ulcers occurred on the dorsal and dorso-lateral aspects of the buffaloes, we presume that it was more convenient for the birds to enlarge existing wounds. The ulcers in different buffaloes began to heal simultaneously during March, despite the fact that the birds continued feeding on them.

The reproductive phase of *P. bassoni* appears to be shorter than that of *P. bovicola*. Nevill (1984) found that in cattle the first bleeding points appeared in June and disappeared the following May, with a peak during October and November. In buffalo, the first bleeding points appeared only in November and persisted to the end of February. The synchronous healing of the large ulcers during March, when the hypersensitivity reaction diminishes in the absence of living and/or dead adult nematodes, supports this assumption of a short reproductive phase.

Adult *P. bassoni* has so far been recovered only from the orbital connective tissue of springbok in Namibia (Ortlepp 1962b). Although *P. bovicola*, which cause similar lesions in cattle, occur primarily in the subcutis, Chauhan *et al.* (1974) and Chauhan, Arora, Agrawal & Ahluwalia (1976) recovered a juvenile male *P. bovicola* from the anterior chamber of the eye of an Asian buffalo, and Ortlepp (1962a), a gravid female *P. multipapillosa* from the posterior chamber of the eye of a horse. Nevill (1980) successfully infected cattle with infective third-stage larvae of *P. bovicola* per conjunctiva. Adult *P. bassoni* occurred in the orbital connective tissue of all five springbok examined (Ortlepp 1962b). He assumed that this site was not abnormal for this parasite (Ortlepp 1962b). In view of the above findings it seems that the eye is a normal site of entry of infective larvae of this genus. Unfortunately the eyes of buffaloes killed during this study were not examined for the presence of nematodes.

The factors governing the predilection sites of *P. bovicola* during oviposition in cattle are undetermined (Nevill 1984) but the dorsal and lateral aspects of the body seem to be preferred. The same tendency was observed with *P. bassoni* in buffaloes, 74,4% of bleeding points occurring in these regions. Since haematophagous or partially haematophagous flies are the intermediate hosts of all the *Parafilaria* spp. this may be to ensure that the vectors, which usually feed around the face, are attracted to blood containing embryonated eggs. It may also be that the higher up on the body the bleeding points occur, the longer the blood streak will be, thus providing a larger feeding area and greater volume of blood for the vectors. In cattle only 7,8% of lesions occur on the ventral aspects (Nevill 1980) as opposed to 25,6% in buffaloes. This suggests that in buffaloes a wider spectrum of vectors may be involved, some of which may be attracted to the shaded areas of the body.

The presence of embryonated eggs in fresh and crusted blood collected from primary lesions provides an easy method to immediately confirm a preliminary diagnosis of parafilariosis. However, the collection of specimens was complicated by the following factors: it took considerable time to immobilize and sample a buffalo after a fresh bleeding point had been seen and clotting may have set in; oxpeckers were quick to attend to the fresh bleeding spot and remove crusts;

buffaloes do not have a thick hair coat and it was often necessary to virtually scrape off remnants of crusts left behind by the oxpeckers; and the buffaloes were often covered with mud, which was of necessity included in the sample.

According to Sundquist *et al.* (1989) the 4l and 36 Kda antigens are specific for *P. bovicola*, and the ELISA test performed on bovine material showed a 95% specificity and a 92% sensitivity. Infected cattle were identified even before bleeding points appeared. The results obtained with the sera of the buffaloes suggest that the test is genus-specific. Cross-reactivity with antigens of other nematode genera (Neppert 1974) was not observed in any of the studies described by Sundquist, Zarkrisson, Bech-Nnielsen & Bianco (1988) and Sundquist *et al.* (1989).

Four to five months are required to develop a positive titre in cattle (Sundquist *et al.* 1989). It is not known for how long a positive titre persists in buffaloes but cattle have to be re-infected annually for the continuity of the life cycle from one season to the other, thus to maintain a positive titre (Sundquist *et al.* 1989). Suspicious reactions in buffaloes could either reflect recent infections or animals losing their positive titre because of not being re-infected. The absence of serologically positive buffaloes younger than 2 years of age can be ascribed to the fact that the majority of buffalo calves in the KNP complex are born between January and April (Pienaar 1969). During this period the number of bleeding points declines to such an extent that the possibility of newly-born calves being infected is probably very low. One can therefore assume that they are not infected shortly after birth but only during the following summer season.

The presence of this parasite throughout the KNP complex suggests that it must have been present for a long time and may even be endemic.

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LESIONS IN THE HEART AND LUNGS OF GREATER KUDU (*TRAGELAPHUS STREPSICEROS*) CAUSED BY *CORDOPHILUS SAGITTUS* (NEMATODA: FILARIOIDEA)

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Abstract: Lesions in the heart and lungs resulting from infection by the filarial nematode *Cordophilus sagittus* were observed in 31 of 42 free-ranging greater kudu (*Tragelaphus strepsiceros*) of the Kruger National Park, Republic of South Africa. Animals less than 1 yr old were free of lesions whereas many of those over 1 yr old, irrespective of sex, were affected. Adult worms were found free in the right ventricle of the heart, in coronary arteries, and in the pulmonary artery and its branches. In the coronary arteries, worms were usually found coiled within aneurysmal lesions, which were often visible on the epicardial surfaces and occasionally on the endocardial surfaces of the right and left ventricles. Within pulmonary arteries, the presence of the parasites provoked a unique intimal proliferative response similar to that seen in canine dirofilariasis.
Key words: *Cordophilus sagittus,* kudu, *Tragelaphus strepsiceros,* vasculitis.

INTRODUCTION

Cordophilus sagittus (v. Linstow, 1907) Monnig, 1926 is a member of the nematode family Filarioidea. The parasite infects the chambers and vessels of the heart and the branches of the pulmonary artery of certain antelope. Von Linstow[5] described the original specimen, which was found in the heart of a bushbuck (*Tragelaphus scriptus*) in the Cameroon. Turner[4] reported *C. sagittus* in a bushbuck from Malawi and suggested that kudu (*T. strepsiceros*) may also be parasitized. McCully et al.[3] described the pathologic lesions associated with the parasite in kudu, bushbuck, and African buffalo (*Syncerus caffer*). Cordophilosis is also known to occur in nyala (*Tragelaphus angasi*) in the Kruger National Park (KNP), Republic of South Africa.[2] Young and Basson[6] reported fatal cordophilosis in eland (*Taurotragus oryx*) translocated to the KNP.

The purpose of this study was to identify pathological lesions associated with *C. sagittus* in free-ranging greater kudu. The location of the parasites within the coronary vessels differs from that described previously.

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MATERIALS AND METHODS

Free-ranging kudu from the Malalane area of the KNP were shot in the neck, exsanguinated, and necropsied. Most of the kudu were taken between 0700 and 0900 hr on the day of their necropsy. Forty-two animals were examined as part of a study of external and internal parasitic infections and pathological lesions in the kudu. Filarial worms from the heart and lungs were fixed in alcohol and later identified as *C. sagittus.*[4,5] Tissue samples containing lesions of cordophilosis were fixed in 10% neutral buffered formalin, embedded in paraffin, and processed by standard methods.

RESULTS

Macroscopic findings

Lesions resulting from *Cordophilus* infection were found in 31 of 42 kudu examined (Table 1). Macroscopic lesions were observed in both the heart and lungs of most kudu over 1 yr old. Pale round-to-oval cyst-like lesions measuring 1–2 cm in diameter were usually visible from the epicardial sur-

Figure 1. Heart of a greater kudu with aneurysmal lesion (arrow) of coronary artery caused by adult *Cordophilus sagittus.*

faces of the ventricles but were only occasionally visible from the endocardial surfaces (Fig. 1). Dissection revealed these cystic lesions to be parasitic aneurysms within branches of the coronary arteries. Up to six worms (both sexes) could be found coiled within an aneurysmal cavity. It was not unusual to find as many as five aneurysms in a heart. They were most often located adjacent to the intermediate or longitudinal grooves. Adult worms were also found free in the right ventricular chamber and in the pulmonary artery at the base of the heart as well as along its course into the pulmonary parenchyma. Parasites were also found in the smaller branches of the pulmonary arteries, particularly in the distal diaphragmatic lobes (Fig. 2), where their presence provoked a florid, sharply demarcated villous proliferation of the intima (Fig. 3).

Table 1. Distribution of lesions caused by *Cordophilus sagittus* by age group and sex in greater kudu from Kruger National Park, Republic of South Africa.

Sex and age	No. lesions	Lesions		
		Lungs only	Heart only	Heart and lungs
Male				
1 yr	2	0	0	0
1–2 yr	2	1	0	3
Adult	0	2	0	8
Female				
1 yr	5	0	0	0
1–2 yr	1	1	1	2
Adult	1	0	2	11
Totals	11	4	3	24

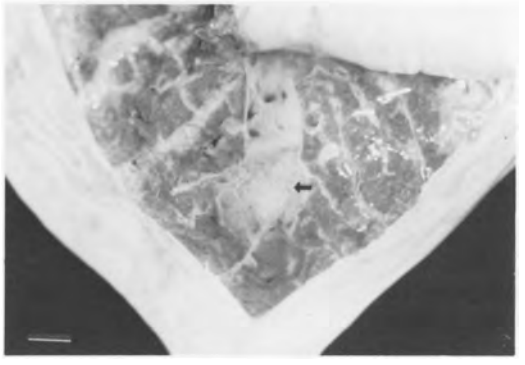

Figure 2. Cut surface of distal diaphragmatic lobe of lung from a greater kudu. Florid villous proliferation of the intima of the pulmonary artery (arrow) is caused by adult *Cordophilus sagittus.* Bar = 1 cm.

Histological findings

Histological sections of parasitic aneurysms in the branches of the coronary arteries revealed a greatly dilated vascular space containing worms. The walls of these affected arteries and their branches were thickened above and below the parasitic aneurysms (Fig. 4). Focal areas of ventricular myocardial scarring were noted in the more heavily parasitized hearts. Often a mild villous proliferation of the intima was present but never to the degree observed in the affected pulmonary arteries. The normal architecture of the coronary artery wall was replaced by fibrous tissue. Bundles of medial smooth muscle cells were isolated by thick bands of fibrous tissue or had been pushed to the periphery by fibrosis of the intima. External and internal elastic lami-

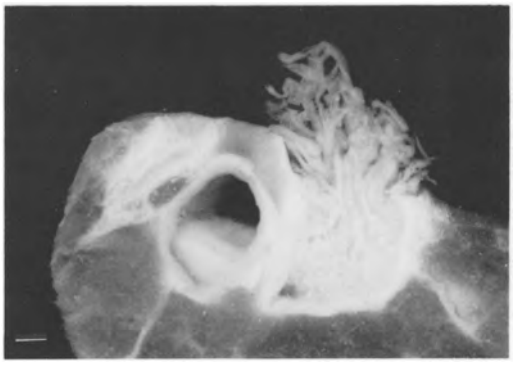

Figure 3. Cut section of distal diaphragmatic lobe of lung from a greater kudu infected with *Cordophilus sagittus*. Note the extended fronds (arrow) of the villous proliferation of the pulmonary artery. Bar = 1 mm.

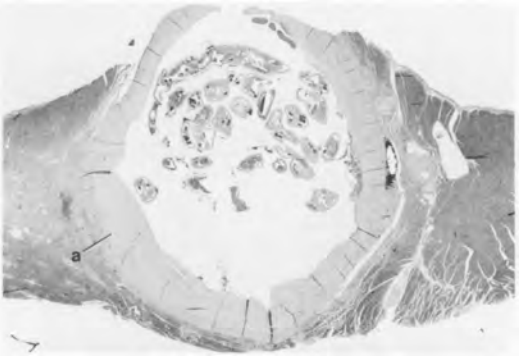

Figure 4. Histological section of heart from a greater kudu infected with *Cordophilus sagittus* revealing thickened wall of the coronary artery (a) and numerous sections of adult parasites within the lumen. H&E, ×6.

nae were disrupted and distorted. Thick-walled arterioles were prominent within the fibrous tissue surrounding the affected coronary vessels, and nodular collections of lymphocytes were occasionally seen in the adventitia. Foci of mineralized debris (parasitic "mummies") could sometimes be found within the walls of the parasitized arteries (Fig. 5). Arterioles in the adjacent myocardium were prominent as a result of smooth muscle hyperplasia, and venules were dilated. Perivascular inflammatory infiltrates consisted of lymphocytes and eosinophils. Focal areas of fibrosis and lymphocytic interstitial myocarditis were also noted. Changes in the myocardium were prominent in areas adjacent to parasitized arteries, but changes gradually diminished distal to these areas. In a few kudu, microfilariae were observed in the myocardium, where they seemed to elicit a moderately severe, but localized, eosinophilic myocarditis. Multinucleated giant cells were often closely associated with these microfilariae. Eosinophilic lymphadenitis and focal areas of eosinophilic pneumonia were attributed to the presence of microfilariae, but such lesions were few in number.

Microscopic changes observed in the parasitized branches of the pulmonary arteries were similar to those in coronary arteries, with one notable difference: a localized florid villous intimal proliferation enveloped worms within branches of the pulmonary arteries (Fig. 6). Fronds of intimal tissue measuring 20–200 μm in diameter extended from the intimal surface of the vessel. Each frond was composed of a fibrovascular core that contained occasional smooth muscle cells and was covered by one to multiple layers of endothelial cells (Fig. 7). Eosinophils and plasma cells were often abundant within these intimal fronds. Arterioles adjacent to the parasitized pulmonary arteries had thickened walls as a result of medial smooth muscle hyperplasia, and accumulations of lymphocytes and eosinophils were

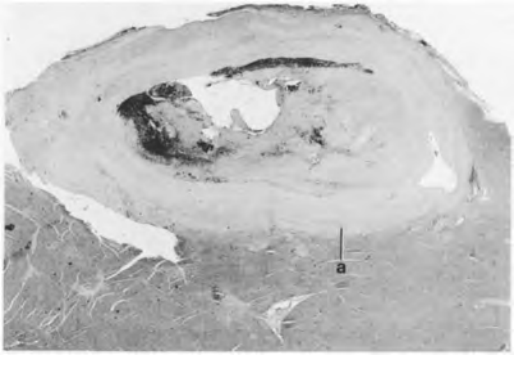

Figure 5. Histological section of heart from a greater kudu revealing thickened wall of a coronary artery (a) surrounding a focus of mineralized debris. H&E, ×5.

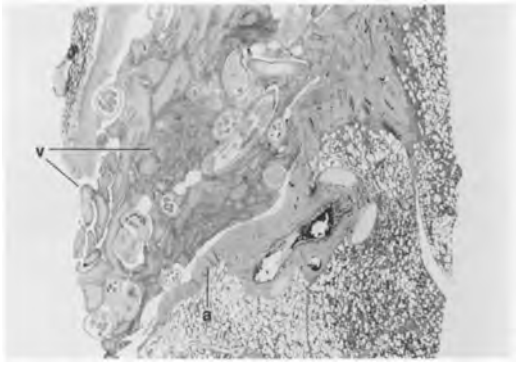

Figure 6. Histological section of distal diaphragmatic lobe of lung from a greater kudu. The lumen of a branch of the pulmonary artery is occluded by an intimal proliferation that envelops adult *Cordophilus sagittus*. a, wall of pulmonary artery; v, proliferation of intima of pulmonary artery. H&E, ×15.

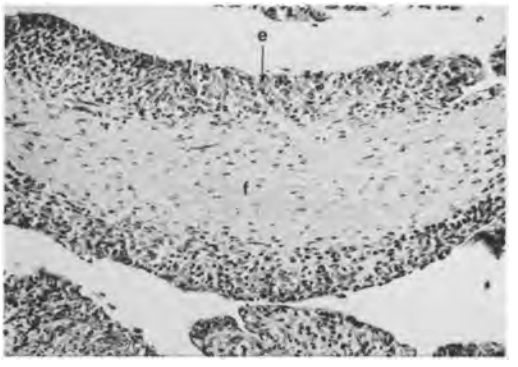

Figure 7. Histological section of frond composing the intimal proliferation of the pulmonary artery from a greater kudu. Note that the fibrovascular core (f) is covered by multiple layers of endothelial cells (e). H&E, ×150.

in adventitial tissues. The surrounding lung parenchyma was essentially normal except in those few kudu that had localized areas of eosinophilic pneumonia associated with the presence of microfilariae.

Adult male and female parasites exhibited morphological characteristics (i.e., coelomyarian musculature, a small intestine, and lateral internal cuticular ridges) of a filarial nematode (Figs. 8, 9).

DISCUSSION

The presence of *Cordophilus sagittus* and associated lesions in the hearts and/or lungs of 31 of 35 kudu over 1 yr old suggests a high rate of infection. The absence of adult worms and associated lesions in a number of kudu lambs studied could be the result of lowered susceptibility to cordophilosis in this age range. However, because many filarial parasites have lengthy prepatent periods, it is more likely that lambs receive infective larvae via one or more species of biting insects at an early age followed by a long period of migration and maturation of parasites. This may culminate in aggregations of adult parasites in the right ventricle of the heart and in coronary and pulmonary arteries as observed in the adult animals in this study. It seems reasonable to assume that whereas some infective larvae mature in the right heart and pulmonary arteries, others pass through the lungs and left heart to find their way into the coronary arteries. Once the worms reach maturity, it is unlikely that further migration occurs.

Aneurysmal lesions similar to those in this study have been reported in kudu, bushbuck, and an African buffalo,[3] but the *C. sagittus* adults were supposedly said to be found in coronary veins rather than arteries. In that study, hypertrophy and hyperplasia of medial smooth muscle cells of "smaller branches" of the coronary arteries were ascribed to hypertension created by obstruction of the venous drainage. It seems unlikely that the parasite changed its site of predilection from coronary veins to arteries. It may be that the intimal proliferation and transmural fibrosis that characterized the well-developed arterial lesions obliterated the histological features of the parasitized coronary vessels thus causing them to be mistaken for veins.

The medial smooth muscle hyperplastic changes observed in smaller coronary and pulmonary arteries and arterioles of parasitized kudu are reminiscent of those observed in the lungs of dogs and cats with certain nematode parasites (e.g., *Dirofilaria immitis*) that live in or migrate through pul-

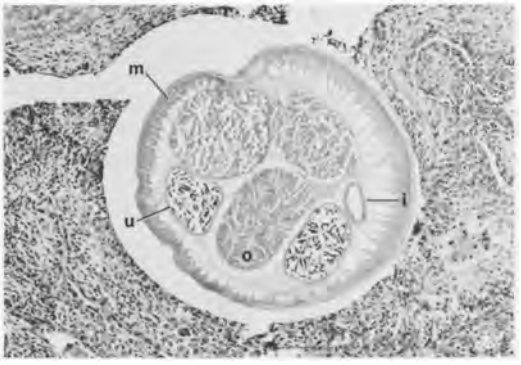

Figure 8. Histological section of lung from a greater kudu containing cross section of an adult female *Cordophilus sagittus* surrounded by fronds of intimal proliferation. Note the coelomyarian muscles (m), small intestine (i), and sections of ovary (o) and uteri (u) containing mature microfilariae. H&E, ×150.

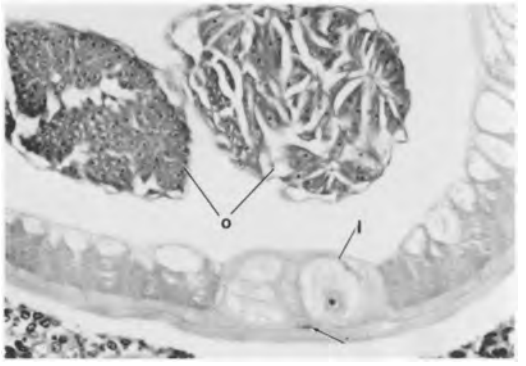

Figure 9. Higher magnification of adult female parasite (*Cordophilus sagittus*). Note sections of ovary (o) and a lateral internal ridge of cuticle (arrow) at the level of the lateral chord (l). H&E, ×300.

monary arteries.[1] Theories on the pathogenesis of such lesions include pulmonary hypertension, inflammatory stimulation by the parasites, thrombosis, and local anaphylaxis in response to parasitic antigens.

The absence of microfilariae in the tissues of most kudu in this study contrasts with descriptions of microfilariae and associated inflammation that were reported in the myocardium and lymph nodes of most kudu in another study.[3] It was conceded in that study that the microfilariae seen in lymph nodes may have been from an unidentified subcutaneous filarial nematode found in the sternal region of several of the kudu. No subcutaneous filarial nematodes were found in the kudu in our study. In only a few kudu, microfilariae and associated lesions were observed in the myocardium, lymph nodes, and/or lungs.

The most significant and interesting lesions of cordophilosis in kudu were the aneurysms in the coronary arteries and branches of the pulmonary arteries and the associated proliferative endarteritis lesions seen most strikingly in the pulmonary vessels. Both of these lesions appear to result from the presence of mature worms; however, the pathogenesis of arterioaneurysms such as these in response to filarial parasitization is not clear. Mechanical, toxic, and/or immune-mediated damage could initially weaken the artery wall with subsequent dilatation resulting from blood pressure. The tendency for these parasitic aneurysms to occur at specific locations (in the epicardium adjacent to the intermediate and longitudinal grooves) is most likely related to vessel diameter and the increased hydrodynamics of coronary circulation of this segment. Although villous intimal proliferations were present in many parasitic aneurysms of the coronary arteries, they never attained the floridity observed in the parasitized branches of the pulmonary arteries, where the aneurysmal dilatation of the vessel lumens were filled with both fine and coarse intimal villi that embraced the parasites. It was apparent in some cases that worms had been incarcerated through the process of intimal proliferation, and calcified remains with attending granulomatous inflammation were occasionally observed within fibrous tissue adjacent to the vascular lumen. Villous intimal proliferation in response to a pulmonary arterial filarial nematode is not unique. A similar response is seen in canine dirofilariasis.[1]

As is often the case with diseases in wildlife, it is difficult to assess the importance of cordophilosis to the general health of kudu herds. The physical condition of heavily

parasitized animals did not differ noticeably from that of animals with few parasites, or even from those free of infection. Pulmonary lesions were remarkable in microcosm; however, even in the heavily parasitized kudu, pulmonary arterial lesions due to *C. sagittus* were not considered significant.

The coronary vascular lesions produced by *C. sagittus* would seem to portend greater physical disability than do the pulmonary lesions; however, the lesions described herein were limited to the parasitized coronary arteries and the adjacent myocardium. Inflammatory changes were generally modest, and neither the livers nor lungs of parasitized kudu showed signs of cardiac decompensation.

The apparent benign nature of *C. sagittus* infection in these kudu is in contrast to cordophilosis reported in eland translocated from Addo Elephant National Park in the Western Cape Province to the Kruger National Park in the eastern Transvaal.[6] During this episode, numerous fatalities attributable to *C. sagittus* occurred among the eland, which apparently had no previous exposure to this parasite. Hearts from dead eland showed many more parasitic aneurysms than did the kudu hearts in our study. Reports of similar fatalities in domestic cattle appear in the files of the Pathology Section, Onderstepoort Veterinary Research Institute.

Although it is seemingly a subclinical condition in kudu (most probably nyala and bushbuck as well), game farmers and wildlife managers should be aware of cordophilosis. In these and other susceptible animals, including domestic cattle, infection may result in severe disease with fatal consequences if, having had no previous exposure to *C. sagittus*, animals are translocated to endemic areas or are otherwise exposed to the parasite.

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