Onderstepoort J. Vet. Res. (1967), *Vol.* 34 (1), 137–160

Printed in the Republic of S. Afr. by the Government Printer, Pretoria

THE PATHOLOGY OF *CORDOPHILUS SAGITTUS* (v. LINSTOW, 1907) INFESTATION IN THE KUDU [*TRAGELAPHUS STREPSICEROS* (PALLAS, 1766)], BUSHBUCK [*TRAGELAPHUS SCRIPTUS* (PALLAS, 1766)] AND AFRICAN BUFFALO [*SYNCERUS CAFFER* (SPARRMAN, 1779)] IN SOUTH AFRICA

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Introduction

Cordophilus sagittus (v. Linstow, 1907) is a member of the family Filariidae which lives in the chambers of the heart, coronary veins and branches of the pulmonary artery of certain antelopes. This nematode was first described as *Filaria sagitta* by Von Linstow (1907). The original specimen was found in the heart of a bushbuck (*Tragelaphus scriptus*) in the Cameroon. Turner (1925) reported its presence in *T. sylvaticus* (= *T. scriptus*) from Malawi, and published additional information on its morphology. He stated circumstantial evidence suggested that kudus may also be parasitized in this country.

Mönnig (1926) revised the classification and transferred the parasite to the genus which he named *Cordophilus*. His specimens consisted of parasites in the following localities:—free in the left ventricle of a kudu from Transvaal, in a thrombus in the left ventricle of a bushbuck (*T. sylvaticus* = *T. scriptus*) from Kenya, in a myocardial tumor of an ox (*Bos taurus* Linn.) from Moçambique, and free in the heart of a bushbuck from Tanganyika.

Consideration of the above-mentioned reports makes it apparent that investigations were based mainly on the morphological studies of the nematode and its taxonomic status, and that relatively little information was published about the lesions evoked by the infestation. Mönnig (1926) records the presence of a myocardial tumor in an ox, and Turner (1925) describes two cysts situated at different sites within the myocardium. One cyst, the size of a hazel nut, protruded above the myocardial surface and contained four female and three male worms. The othe. cyst appeared as a flat opaque area which harboured a male and a female worm

The predilection sites of the adult parasite and the associated macroscopic- and microscopic lesions within the heart, coronary veins and pulmonary arteries appear to be worthy of further recording. A detailed description of the pathology should not only be of veterinary and medical interest but also of value to helminthologists engaged in the study of the life cycle of the parasite and to biologists concerned with the health and conservation of game.

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Received for publication on 15 July, 1966.—Editor

Materials and Methods

Specimens were collected during the process of conducting routine necropsies on one of five bushbuck and 12 of 13 kudus which had died or had been destroyed for various reasons in the Kruger National Park. Additional specimens were obtained from six kudus which had been shot specifically for studies on *C. sagittus* and other parasitic conditions. For the sake of convenience, autopsies of the latter group were conducted at the Veterinary Investigation Centre of Skukuza situated in the southern region of the park. Lesions were examined and photographed. Specimens of various organs were preserved in a neutral buffered 10 per cent formalin solution as well as in Susa's fixative (Cowdry, 1952). *C. sagittus* and other parasites were placed in an alcohol-glycerine mixture. Preserved tissues were processed according to the usual routine methods and then embedded in paraffin wax. Sections, 3·0 microns in thickness, were cut with a sliding microtome and stained with haematoxylin-eosin. Selected sections were subsequently stained with the AFIP modifications of Masson's stain and Hart's elastic stain

Haematoxylin and eosin stained sections from a case of *C. sagittus* in an African buffalo [*Syncerus caffer* (Sparrman, 1779)] were found in the files of the Department of Pathology at Onderstepoort. These were used for comparison with the new materials.

Results

Macroscopic Findings

With the exception of an old and emaciated kudu cow, all animals were in good condition. The emaciation was attributed to the badly worn teeth, pronounced louse infection (*Linognathus* sp.) and the presence of *C. sagittus*.

(*a*) *Observations on affected kudus*

Photographs of macroscopic lesions resulting from *C. sagittus* infestation appear on Plates 1 and 2.

In the heart, adult worms were found only in the conus arteriosus from where some partially extended into the pulmonary artery. They were very often present in aneurysms of the coronary veins located just beneath the epicardium of the left ventricle. Some were present in the ascending tributaries of the great coronary vein which lie in parallel to and near the intermediate and longitudinal grooves [Plate 1 (1 & 2)]. The diameters of the parasite-filled aneurysms were often remarkably large when compared with undilated adjacent segments of the vein [Plate 1 (3)]. Other macroscopic lesions included dilatation and marked hypertrophy of the right ventricle [Plate 1 (2)]. Fibrosis of the myocardium and an increased prominence of smaller branches of the coronary veins were observed near the apex of the left ventricle [Plate 1 (1 & 2)]. On the cut surface there were, in some cases, scarred areas of myocardium in the vicintiy of the affected portions of the coronary veins.

Adult parasites were always found in the branches of the pulmonary artery. In a few cases some were near the semilunar valves within the largest segment of the pulmonary artery [Plate 1 (5)]. They were found, however, more often in the medium-sized and small branches of the pulmonary artery, especially those accompanying the small bronchi measuring a little less than 1·0 to 2·0 cm in diameter [Plate 2 (7 & 8)]. Smaller branches which accompanied bronchioles were also

involved. At sites where the worms were found coiled in masses, there were frequently rather extensive proliferative reactions into the lumen from the arterial wall [Plate 2 (8 & 10)]. Many of the vessels had dilated and showed thin walls, while others were distended greatly and obviously aneurysmic [Plate 1 (6)]. After removing the worms and washing the arteries the projections from the intima appeared white and resembled Turkish towelling [Plate 2 (8)] or mops of string [Plate 2 (10)]. It was apparent that once the exuberant intimal proliferations filled the lumen at the point of their attachment, they grew along the course of the lumen of the artery. This resulted in many long stringy proliferations [Plate 2 (11)]. No specific change was observed in the lung tissue. Lesions in arteries, however, could be palpated through the tissue as either bulbous enlargements or more firm portions of the vessel.

Of the primary sites, heart chambers, coronary veins and pulmonary artery and its branches, the adults were most consistently found in the branches of the pulmonary artery. A number of the cases contained them in the branches of the pulmonary artery in the absence of parasites in the coronary vein. When present in the coronary vein, others could always be found in the branches of the pulmonary artery.

It is beyond the scope of this paper to describe all other parasites or disease conditions which were encountered in kudus. Attention will nevertheless be drawn to an, as yet, unidentified subcutaneous filarial worm which was found towards the end of the investigations. The unexpected discovery naturally prompted a careful examination of the subcutis of the next, but at the same time also the last kudu which became available for necropsy. In both animals parasitized subcutaneous nodules were encountered over the sternal region. Microscopic examination of scrapings prepared from nodules revealed microfilariae. The subcutis of previous animals had not been examined closely, so that it cannot be stated unequivocally that they were parasitized.

(*b*) *Observations on a bushbuck*

Parasites were observed exclusively in the pulmonary artery. There was no evidence that the cardiac ventricles and coronary veins were infested. Lesions were observed in the pulmonary artery. They were similar in appearance to those of the kudu, and appeared in the smaller branches of the pulmonary artery which accompanied bronchi.

Microscopic Findings

(*a*) *Observations on affected kudus*

Photographs of microscopic lesions resulting from *C. sagittus* infestation appear on Plates 1 to 6. Information gained from the study of sections, prepared from nine kudus comprises the basis of the major portion of the part covering the histopathological descriptions of this report.

Heart:—As there was no single heart, which contained all lesions representative of those observed in the entire group, a description of a picture composed of various types of lesions will be necessary. There were lesions which were produced by adult parasites and others which were due to microfilariae, while some were considered to be of a rather non-specific nature. Although the latter lesions are believed to represent a phase of the disease, it could not be determined with certainty whether they were produced by adults or microfilariae of *C. sagittus*.

The most striking cardiac lesions were the frequently observed aneurysms of the tributaries of the coronary veins. The interference of blood flow resulting from the aneurysms coupled with the endoplebitis obliterans of the coronary vein, had in some cases produced significant secondary lesions in the myocardium. The marked dilatation of the tributaries of the coronary vein resulted in large thin-walled aneurysms, which usually contained adult filarial worms [Plate 1 (4)]. Microscopically, various segments of the worms indicated that some were dead and others alive at the time when tissues were collected. Some of the dead ones were degenerative and sometimes completely enveloped by host connective tissue and impregnated with mineral [Plate 1 (4)]. In response to irritants of the living metazoon, the histologic layers of the vein, particularly the intima, were greatly thickened by proliferative fibrous tissue. The parasites and the host response virtually filled the lumen of some of the aneurysms. As the worms were coiled in an entangled mass, there were longitudinal, transverse and oblique sections of the various body levels in the histological preparations. Although in some of the aneurysms the exuberant proliferative reaction of the intima practically filled the lumen with little evidence of the parasite remaining, it was more common that parasites were present at such a site. The intimal proliferations were composed of either a highly cellular non-differentiated type of reaction or cells of fibroblastic nature. Other proliferations were composed of less cellular but quite dense and more mature connective tissue.

The exuberant tissue in the lumen extended as projections of various shapes and sizes, ranging in appearance from a fine villose one to relatively big, coarse, club-shaped proliferations which in some aneurysms freely anastomosed and in others merged to form a large, dense mass of tissue. Such large dense masses sometimes became hyalinized connective tissue and occupied the lumen of some of the aneurysms with only a few narrow attachments to the intima. Mineralized adult worms (parasitic " mummies ") were sometimes incorporated into such chronic reactions [Plate 3 (13)].

Depending apparently upon the sojourn of the parasite at a particular site, the cells composing the cores of projections extending into the lumen were either fibroblasts or fibrocytes. The more active, still-proliferating clubs were covered by multiple layers of immature, quite-undifferentiated cells. It was not possible to determine whether these cells represented metaplasia or hyperplasia of the endothelium or whether they originated from the underlying primitive mesenchymal cells. The projecting reactive tissues were very often infiltrated with eosinophiles, lymphocytes and plasma cells [Plate 3 (16)].

The wall of the coronary vein was often fibrotic at a distance from the dilated portions. Therefore, depending upon the distance from the aneurysm, the wall was either thin and fibrotic or substantially, though irregularly, thickened by fibrous tissue. Some portions of the vein wall, particularly the intima, were necrotic. The increase in thickness was principally due to intimal fibrosis, but the media and adventitia were likewise affected, though to a lesser degree. In contradistinction to the aneurysmic portions, there were sections of the coronary veins in which the sclerosis of the intima and other layers occluded the lumen, thus representing chronic phlebitis obliterans. Scar tissue had completely replaced the smooth muscle in some of the vein walls [Plate 3 (15)].

It was thus apparent that the response by the host either took the form of projections into the lumen or of marked fibrosis of the vein wall. In either instance the initial reaction appeared to be in the intima where hyperplasia of endothelial cells and/or primitive mesenchymal cells of the subendothelial part of the intima

occurred. These cells were hyperchromatic and appeared to be multiplying rapidly and growing profusely. They either elevated the endothelium or replaced it by sheets of undifferentiated cells which extended into the lumen thus forming polypoid projections of various sizes. The vein wall was sometimes uniformly thickened when similar cells proliferated and when connective tissue developed in the intima without bulging into the lumen as individual projections.

In some of the hearts there were indications of myocardial necrosis in the immediate vicinity of lesions of the coronary veins. There were many polymorphonuclear leucocytes in areas of recent necrosis. Lymphocytes, plasma cells and cardiac histiocytes were present in the more subacute lesions. Phagocytosis of necrotic debris was common. The myocardium in these areas was oedematous. Additional alterations included the loss of striations, granular appearance due to swollen mitochondria and other unequivocal evidence of degeneration and necrosis. There was hyperplasia of the sarcolemmal cells which resembled fibroblasts. More extensive lesions in the late stages of repair were present in the myocardium of some of the other hearts. Dense scar tissue which had replaced necrotic cardiac muscle was mute evidence of areas of previous infarction [Plate 4 (19)]. However, in some of the hearts which had extensive lesions involving tributaries of coronary veins there was no indication of infarction.

The coronary arteries had not escaped completely the effect of adult parasites. Some of the smaller branches of the coronary arteries had greatly thickened walls as a result of marked hypertrophy and hyperplasia of the smooth muscle in the media. This presumably was in response to the coronary hypertension which was necessary to overcome the obstruction to the venous drainage caused by lesions in the coronary veins. Although the lumina of some were somewhat smaller than normal, there was no evidence of complete occlusion of any coronary artery. Fibrosis of the intima of some of the small branches contributed towards the narrowness of their lumina. In one area of myocardial necrosis, small branches of the coronary artery showed a secondary panarteritis with inflammatory cells prominent in all vascular layers. This manifestation was believed to have been the result rather than the cause of the myocardial lesion.

A few sections of the right ventricle contained thickened sclerotic areas of endocardium which resulted from proliferation of fibroblasts [Plate 2 (12)]. The presence of adult worms in the heart chamber appears to be the most plausible explanation for such a lesion in this form of verminosis.

Microfilariae with their basophilic stippling were present in the capillaries of many heart sections but lesions in response to them were of a rather minor nature. Microfilariae were found frequently in the myocardium and sometimes there was a host reaction to them [Plate 4 (23 & 24)]. Microgranulomas with round cells predominating encircled some microfilariae. Others had necrotic centres without any parasitic remnants. Reactions to microfilariae were very severe in some, less so in others and apparently of a very mild nature in a few hearts.

Lesions of small veins and capillaries were prominent in the myocardium of some hearts. They were invariably associated with an apparent increase in both the number present in an area of myocardium and the thickness of their walls. The endothelial cells were hyperplastic and their nuclei were hyperchromatic [Plate 3 (14)], and in addition the lumen of some capillaries appeared to be filled by these along with inflammatory cells [Plate 3 (18); Plate 4 (20)]. Others contained in addition homogeneous, slightly basophilic fibrin-like thrombi [Plate 4 (21)].

Similar material appearing as fibrinoid necrosis was present in the wall of some of the vessels [Plate 4 (22)]. There were often inflammatory cells of mononuclear types around the vessels or between the myocardial muscle bundles [Plate 3 (17)].

Lung:—The most significant lesions were in medium-sized and small branches of the pulmonary artery. Just as in the coronary vein, the adult parasite had caused dilatation of some of the branches of the pulmonary arteries resulting in aneurysms. In these circumstances the lumina usually also contained a most remarkable response of the vessel wall. Microscopically, aneurysm formation [Plate 6 (32)] was indicated by two principle features. One of these was the remarkably increased diameter of certain segments of branches of the pulmonary artery, the measurement of which was several times that of the accompanying bronchi and bronchioli. The other one was the comparative thinness of the wall of such segments which indicated that they were unduly stretched and weakened. In a few aneurysms some portions of the wall consisted only of connective tissue and were completely devoid of the muscle of the media.

Adult parasites were present in the lumina of the aneurysms as well as in other segments of the pulmonary artery [Plate 6 (34)]. Most of the parasites appeared as though they were still viable at the time of the death of the animal. These were interspersed between the intimal projections which extended into the lumen. Some of the parasites, however, were dead. They appeared in various stages of degeneration while some showed mineralization.

The host response in the pulmonary artery was severe and its appearance had wide spectral aspects. In some respects it was similar to the response in the coronary veins. There were, however, distinct differences because of additional features which were only present in the pulmonary artery.

Generally speaking, in the affected portions of the pulmonary arteries, the wall was unevenly fibrotic with intimal thickenings distributed irregularly around the lumen. As a result of the fibrosis, the intima of some branches was as much as three times as thick as the media. This was partially due to a thinning of the media in some arteries, especially in the segments containing aneurysms. All layers of the wall were thin in some segments of the aneurysms.

In the affected segments of the pulmonary artery, the majority of which were not aneurysmic, the tissue reaction, which extended from the intima into the lumen, varied from relatively early lesions to the advanced chronic ones. Both extremes were sometimes present in branches of the pulmonary artery of one animal along with lesions which would fall into intermediate groups. Early lesions alone were not seen in any cases but some of the purely chronic types were present in certain cases. The relatively early lesions of acute verminous arteritis, were present in arteries ranging from the small arterioles to the largest branches in which lesions were observed macroscopically. The arterioles, particularly the intima, contained many eosinophiles. The lumen of some were practically filled with spindle-shaped cells and eosinophiles [Plate 5 (25)]. The former seemed to be continuous with the endothelium, but as previously described in the coronary veins, it was not possible to determine if this represented hyperplasia of the endothelium or hyperplasia of the underlying primitive mesenchymal cells. Palisading of such spindle-shaped cells, and the oval shape of their nuclei probably indicate their fibroblastic nature.

As the size of the affected branches of the pulmonary artery increased, the host response appeared better organized. Just as in arterioles the intima was thickened by endothelial hyperplasia which appeared to be the initial proliferative reaction.

From the intima folds arose which became projections extending into the lumen. They had cores of either fibroblasts or fibrocytes with accompanying collagen, and were covered by sheets of the specifically unidentified hyperplastic cells described above [Plate 5 (30)]. Numerous eosinophiles, lymphocytes and plasma cells covered the surface, and were in the core or stroma of these projections. Similar inflammatory cells were present in the adventitia of some vessels. While this was the basic composition of the intimal projections, the difference in their mass and the degree of maturity of the components altered their appearance correspondingly. Their size and shape ranged from the small delicate villose ones [Plate 5 (30)] to large frond-like forms which were composed of fibroblasts. Between these two extremes there were club or racket-shaped ones of various sizes. Necrosis was common in the larger ones with an abundance of amorphous purplish material which was prominent when sections were stained with haematoxylin and eosin. Areas of fibrinoid necrosis were also present. It was apparent that sometimes the reaction had extended within the lumen of the arteries in a linear fashion. The mass of projections in the centre of the lumen frequently had only a few minor attachments to the intima [Plate 6 (33)] at the point of sectioning, the main attachments being quite often relatively far removed.

The lumina of the aneurysms in branches of the pulmonary artery were also filled largely by parasites and exuberant host response. Conglomerations of the projections of various sizes and shapes occupied the central portions. In some aneurysms the projections, which were indicative of a very chronic stage, consisted of large, thick cores of dense hyanilized collagen containing few inflammatory cells, and covered by a single layer of flat endothelial cells. Others contained combinations of the very chronic proliferations and the less chronic, large club-shaped ones, with either quite active or very mature quiescent cores, covered by thick layers of the rather primitive cells [Plate 5 (28)]. Sometimes in the lumen there were fine villose projections infiltrated with eosinophiles and other cells which indicated a more acute reaction. It was not uncommon for acute secondary reactions to be present on or budding from the surface of the large club-shaped chronic proliferations [Plate 5 (29)]. A few of the lumina were practically filled by proliferative reactions which had fused to form a large mass of connective tissue. Some of these masses were extensively necrotic and mineralized. Others were hyalinized and contained mineralized parasites. Their mural surface sometimes appeared to be a perfect mould of the surrounding intimal surface of the arterial wall with an artefactual space between the two. It is obvious that the two surfaces had been in contact during life.

Some of the projections of tissue filling the lumen had involuntary muscle in their cores. This was demonstrated well by the Masson trichrome stain. The smooth muscle fibres were prominent, and directly continuous with that of the media of the artery [Plate 6 (31)]. Such projections which were thus composed of components from both the intima and the media, were most often near points in the arterial wall where there was injury incidental to aneurysm formation. This feature was never observed in the coronary veins.

The adventitia of many of the affected arteries had an increased amount of more dense appearing connective tissue. In one kudu, in particular, there was a number of acutely affected vessels in which there was not only a severe endovasculitis but also a severe perivasculitis [Plate 5 (27)]. This consisted of an inflammatory infiltrate of numerous eosinophiles and small round cells, and proliferation of capillaries. Adjacent thin-walled veins were also involved by the inflammatory

reaction. Although the proximity of vessels to bronchioles also caused the peribronchiolar tissue to be slightly involved, it was quite clear that the response was to an irritant primarily involving the vessels.

There were thin-walled vessels which appeared to be veins adjacent to some of the very small affected arteries [Plate 5 (26)]. Their hyperplastic endothelial reaction, with a concomitant infiltration of eosinophiles, was very similar to that of the arteries which they accompanied. There was medial hypertrophy of the walls of some of the arterioles which, coupled with endothelial hyperplasia, gave a prominent appearance to the wall and a narrow appearance of the lumen.

Lymph-nodes:—Sections from lymph-nodes were available for study from some of the kudus harbouring *C. sagittus*. They showed changes referable to the presence of microfilariae. Some lesions were more severe than others but basically they were all of the same nature. The more severe and the oldest lesions resulted in a diffuse chronic lymphadenitis. It primarily affected the medullary portion but the cortex was also affected to some extent. There was a marked increase of fibrous tissue in some of the lymph-nodes, and all appeared to contain more small newly formed vessels than normal. There were numerous microfilariae either within blood vessels or appearing to be situated in lymphatic spaces [Plate 6 (35)]. In response there were many plasma cells and Russell-Fuchs bodies. The microfilariae were often in the centre of microgranulomas. Syncytial-type multinucleated giant cells were observed in response to the irritant. Numerous eosinophiles were present, particularly in the intermediate sinuses [Plate 6 (36)]. In the small blood vessels there were hyperplastic cells interpreted as being from the endothelium.

(*b*) *Observations on additional wild ruminants*

Though the specimens were not a part of this study originally, this opportunity is taken to report the occurrence of adult *C. sagittus* in two other wild ruminants, namely an African buffalo (*Syncerus caffer*) and a nyala (*Tragelaphus angasi*, Gray, 1849).

In the files of the Pathology Department at Onderstepoort, a previously unreported case of a buffalo which had the parasite under discussion in the coronary vein was found. The specimen originated in Zululand. In the dilated lumen of the coronary vein there were segments of partially mineralized adult parasites. Coarse villose intimal projections of the nature found in the kudu partially filled the lumen. A lung section from the affected buffalo did not contain segments of parasite or lesions in branches of the pulmonary artery.

The parasite from a nyala which was collected in the Mkuzi Game Reserve in Zululand is recorded in the files of the Helminthology Section at Onderstepoort. It was removed from a cyst in the heart and sent to Onderstepoort where it was identified as *C. sagittus* by Ortlepp, 1961.

There were no histopathologic preparations available for study from either the bushbuck or nyala.

Discussion

The presence of *C. sagittus* in 18 out of 19 kudus examined, indicates a very high infestation rate in the Kruger National Park. The question of its significance as a health problem in kudu and bushbuck is at present difficult to answer. As illustrated in Plate 1 (2), one of the hearts photographed, demonstrates hypertrophy

and dilatation of the right ventricle approaching *cor pulmonale*. Medial hypertrophy of the arterioles of the lung could have been the result of pulmonary hypertension. The scarred areas of the heart, while not evidence of very massive necrosis, cannot be passed off lightly because at an earlier stage these areas could have affected heart function significantly. The fact that parasitized antelopes can be confronted frequently by lions and other predators makes any impairment to the heart a serious matter. In the event of coronary aneurysms rupturing, the animal would very likely die as a result of cardiac tamponage. In view of the lesions of the coronary veins, it is surprising that antelopes with more extensive myocardial lesions were not encountered. Such antelopes, however, could more easily have fallen prey to lions, and thus would have passed unnoticed. It might also be a reflection of the well-developed collateral venous drainage of the heart. Obstructions would presumably have developed gradually in response to the growth of the parasites and the development of aneurysms and the intimal lesions. This sequence of events would permit sufficient time for the collaterals to dilate in order to compensate for any venous inefficacy occurring.

The significance of the effect of this parasitism on the yearly increase in the population of wild ruminants can probably only be determined by comparisons with similarly situated species in areas where the parasite is not prevalent.

The occurrence of *C. sagittus* in two additional *Tragelaphus* spp. was not surprising in view of the very high incidence of the parasite in kudu. On the other hand it was less expected for the parasite to occur in the more distantly related zoologic species, the African buffalo.

The demonstration of *C. sagittus* in the heart of an ox by Mönnig (1926) is reason to include this nematode in the differential diagnosis of bovine myocardial verminosis. There is a considerable resemblance between the gross appearance of the *Cordophilus* lesions in the coronary veins and the superficially located *Cysticercus* cysts. In the routine examination of bovine hearts in an abattoir, especially if done hurriedly, the heart so affected would be condemned for food without further delay. The *Cordophilus* lesions would probably only be recognized specifically if they were incised, exposing the parasite and demonstrating the true nature of the lesion. A close examination of the hearts of cattle from areas where there are affected kudu and bushbuck is, therefore, indicated. There appears to be no other nematode which frequents and produces aneurysms in the coronary vein of cattle and hence no further comparisons can be made.

With further observations it may prove to be that the occurrence of *C. sagittus* in both the ox and African buffalo is of an incidental nature. It would appear that the primary hosts are the tragelaphine species.

Lesions in branches of the pulmonary artery of the above-mentioned ruminants are very similar to some of those which are associated with the heart worm *Dirofilaria immitis* (Leidy, 1856) in the dog and some other hosts. Thrombosis and aneurysm formation in the branch of the pulmonary artery supplying the diaphragmatic lobe of the lung have been reported in association with dead heart worms in a dog (Smith & Jones, 1961). Haythorn & Ryan (1917) recorded a case of a dog, which in addition to having an aortic aneurysm due to *Spirocerca lupi* (Rudolphi, 1809) had *D. immitis* in response to which there was a " shaggy polypoid proliferation of the intima of the pulmonary artery ". This expression describes some of the lesions observed in the pulmonary artery of the above-mentioned antelopes quite well. Adcock (1961) described the presence of rugose projections of the intima of the pulmonary artery which moulded themselves against the heart worm. This type of

formation was not observed in antelopes but some of the other lesions, which were described and illustrated, are essentially the same as those seen in *C. sagittus* infestations.

The precise cause of the proliferative reaction in pulmonary arteries containing filarial worms such as *D. immitis* may be difficult to pinpoint. Hennigar & Ferguson (1957), quoted by Adcock (1961), suggested that this might be due to hypersensitivity or mechanical irritation. Adcock pointed out that it appeared as though the inflammatory, proliferative and fibrotic changes of the intima were in response to contact between viable parasites and arterial intima. This would also appear to be the case with *C. sagittus* in antelopes. There can be no doubt that the adults of these two species of nematodes stimulate proliferative vascular changes. Whether they do so either through metabolic products, mechanical irritation, antigenic substances, toxin or possibly even by some other means is, however, very difficult to determine. The fact that there are changes in many of the branches of the pulmonary artery which have a diameter too narrow for even one small male *Cordophilus* to enter, suggests that either the microfilariae or an irritant from the adult parasite can have an effect on the intima at some distance away from the adult parasite itself. This was quite apparent in the antelopes.

The secondary pulmonary lesions which are often seen in canine dirofilariasis such as marked congestion, haemorrhage, oedema, haemosiderosis, pneumonitis and infarction were not seen in antelopes. It must be remembered, however, that these were apparently healthy animals which did not show signs of congestive heart failure generally associated with terminal cases of canine dirofilariasis. Some antelopes showed myocarditis, myocardial oedema, degeneration and necrosis but only one kudu had areas in the myocardium which were considered to have been areas of previous infarction.

Reactions to the microfilariae in the antelopes appear to be consistent with those to other filarial parasites. Besides the direct inflammatory response in the heart there was circumstantial evidence that suggested an association between microfilariae and proliferative changes in the small blood vessels. This parasitic stage was obviously the cause of the changes in lymph-nodes. It must be mentioned that when specimens were being collected the lymph-nodes were not labelled so that the site at which they were situated is unknown. This technical fault possibly precluded determining precisely which of the two species of microfilariae were responsible for the lymphadenitis. Had the nodes been those draining the subcutis of the sternum, it would have been strong circumstantial evidence for the subcutaneous filaria being the source of the microfilariae.

The complete life cycle of *C. sagittus* is still undetermined. It is a viviparous species and microfilariae do circulate in the blood. In common with some of the other members of the family Filariidae one may assume that this species may also be transmitted biologically by a blood-sucking insect.

Consideration of the nature of the lesions produced by *Cordophilus sagittus* makes it apparent that they are specific for certain ruminants. It is, therefore, proposed that the name "Cordophilosis" be applied for this form of vascular filariasis. If necessary, the adjectives bovine, syncerine or tragelaphine should precede the proposed name to indicate the host or hosts that are affected.

Summary and Conclusion

1. An account is given of the lesions caused by *Cordophilus sagittus* infestations in an African buffalo, a bushbuck and 18 kudus.

2. The distribution of adult parasites and the host response in the kudu are given in detail.

3. Adult worms appeared most frequently in medium-sized and small branches of the pulmonary arteries, less frequently in coronary veins and only occasionally in the right ventricle.

4. Aneurysms filled with parasites and showing a rather remarkable proliferative intimal reaction frequently appeared in the coronary veins and in the medium-sized and small branches of the pulmonary arteries.

5. Non-aneurysmic segments of the pulmonary arteries also reacted to the parasitic infestation. This manifestation might be summed up as a " proliferative polypoid villose verminous endarteritis ".

6. Attention is drawn to the close resemblance between this manifestation and certain types of reactions in the pulmonary arterial tree of dogs caused by another filarial worm, *Dirofilaria immitis*.

7. The possible significance of the lesions described above as a health problem in wild ruminants is discussed.

8. Knowledge on the incidence of *C. sagittus* infestation in cattle and buffalo appears to be restricted to single cases in each. Careful examination of the myocardium of these species when the opportunity avails itself may yet reveal a higher morbidity rate.

9. It is proposed that the derivative from the generic name of the nematode namely, " Cordophilosis " be used for the disease.

Acknowledgements

The authors wish to express their gratitude to the following: the National Parks Board for providing the opportunity of doing autopsies on antleope in the Kruger National Park and to their staff for their co-operation and assistance; Prof. J. D. Smit, head of the Department of Pathology for permission to use the African buffalo material and for helpful criticism; Mr. A. M. du Bruyn and his staff for their expert production of photographs; Mr. J. L. de B. van der Merwe and his technicians for the preparation of the sections, and Mrs. W. M. Willemse for clerical assistance.

References

ADCOCK, J. L., 1961. Pulmonary arterial lesions in canine dirofilariasis. *Amer. J. Vet. Res.* 22, 655–662.

ANON., 1960. Manual of histological and special staining technics. Armed Forces Institute of Pathology, Washington, D.C. 2nd ed. New York: The Blackiston Division, McGraw-Hill Book Co., Inc.

COWDRY, E. V., 1952. Laboratory technique in biology and medicine. Baltimore, Md.: Williams and Wilkins.

HAYTHORN, S. R. & RYAN, A. H., 1917. Aortic aneurysms in dogs with report of six cases. *Med. Res.*, 35, 411–424.

HENNIGAR, G. R. & FERGUSON, R. W., 1957. Pulmonary sclerosis as a result of *Dirofilaria immitis* infection in dogs. *J. Amer. vet. med. Ass.*, 131, 336–340.

MALLORY, F. B., 1938. Pathological technique. Philadelphia, Pa.: W. Saunders.

MALLORY, F. B., 1942. Pathological techniques. Philadelphia, Pa.: W. B. Saunders.

MÖNNIG, H. O., 1926. Three new helminths. *Trans. Roy. Soc. S. Africa*, 13, 291–298.

ORTLEPP, R. J., 1961. Onderstepoort: Unpublished record.

SMITH, H. A. & JONES, T. C., 1961. Veterinary pathology. Philadelphia, Pa.: Lea & Febiger.

TURNER, W. Y., 1925. The morphology of *Filaria sagitta* v. Linstow, 1907, from the heart of *Tragelaphus sylvaticus* in Nyasaland. *J. Helminth.*, 3, 15–18.

VON LINSTOW, O. F. B., 1907. Nematoden aus dem Königlichen Zoologischem Museum in Berlin. *Mitt. Zool. Mus. Berlin*, 3, 251–259.

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PLATE 1.—Photographs prepared from kudu specimens.

1. Heart showing two aneurysms (A & B) in tributaries of the great coronary vein. Aneurysm (A) in the tributary lying in the intermediate groove; Aneurysm (B) in the left longitudinal groove

2. Aneurysm (A) in a tributary of the great coronary vein. Notice also dilatation and hypertrophy of the wall of right ventricle

3. Opened aneurysm from heart (2). Compare size of lumen with that of vein on either side

4. Low magnification photomicrograph of aneurysm of coronary vein (A) containing adults of *C. sagittus*. Notice the mineralized parasites (B) surrounded by fibrous reaction of the wall of vein.

5 Adults of *C. sagittus* in the pulmonary artery near semilunar valves

6 Adults of *C. sagittus* (A) and intimal reaction filling an aneurysm (B) of a branch of the pulmonary artery (C)

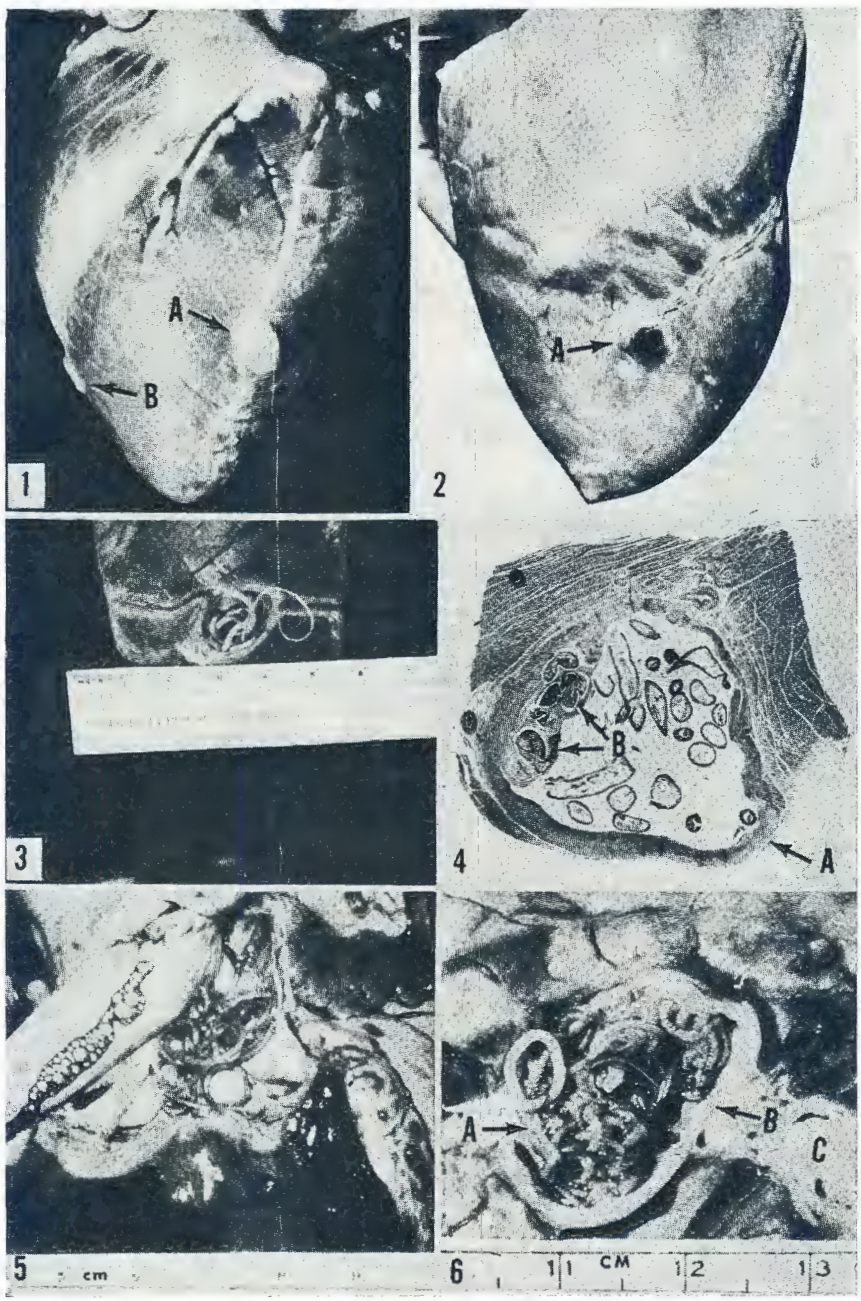
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PLATE 2.—Photographs prepared from kudu specimens

7. Lung. Parasites and villose proliferations in a branch of the pulmonary artery (A) accompanying a small bronchus (B)

8. Lung. Same vessel (A) in (7) after worms were removed and the specimen cleaned. Notice Turkish towelling appearance

9. Lung. Adults of *C. sagittus* and intimal response in another branch of a pulmonary artery

10. Lung. Cleaned specimen of (9). Notice that mass of villose proliferations, appearing as a ragmop, extends from one branch of pulmonary artery (A) into another branch

11. Lung. Picture of villose proliferations from a small branch of a pulmonary artery (A) against a black background illustrates their long stringy nature

12. Heart. Endocardium of right ventricle. Proliferation of fibroblasts (A) is shown along with a focus of superficial necrosis (dark area)

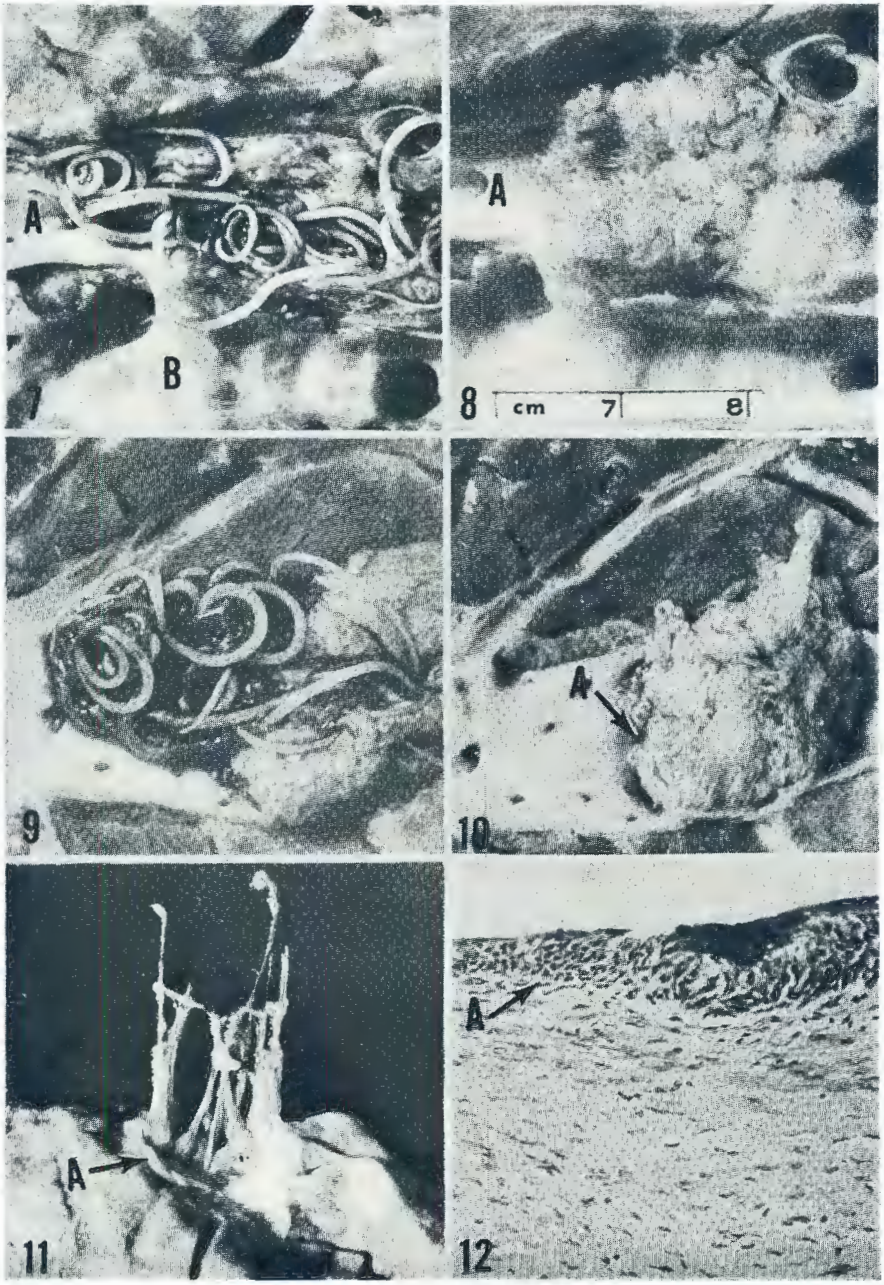
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PLATE 3.—Photographs prepared from kudu specimens

13. Tributary of coronary vein. The lumen (A) is almost occluded by fibrous reaction which also involves vessel wall. Notice remains of a dead parasite (B) incorporated in the reaction

14. Coronary vessel in myocardium. Notice endothelial hyperplasia within vessel (A) and reaction in interstitium of myocardium

15. Tributary of coronary vein (A). Chronic phlebitis obliterans with fibrous tissue replacing the smooth muscle of the media. Branch of coronary artery (B)

16. Coronary vein with proliferative reaction (A) and inflammation of vessel wall

17. Myocardium showing a round cell infiltrate interstitially

18. Myocardium. Proliferating cells filling lumen of small vessels (A) and a surrounding interstitial cellular reaction

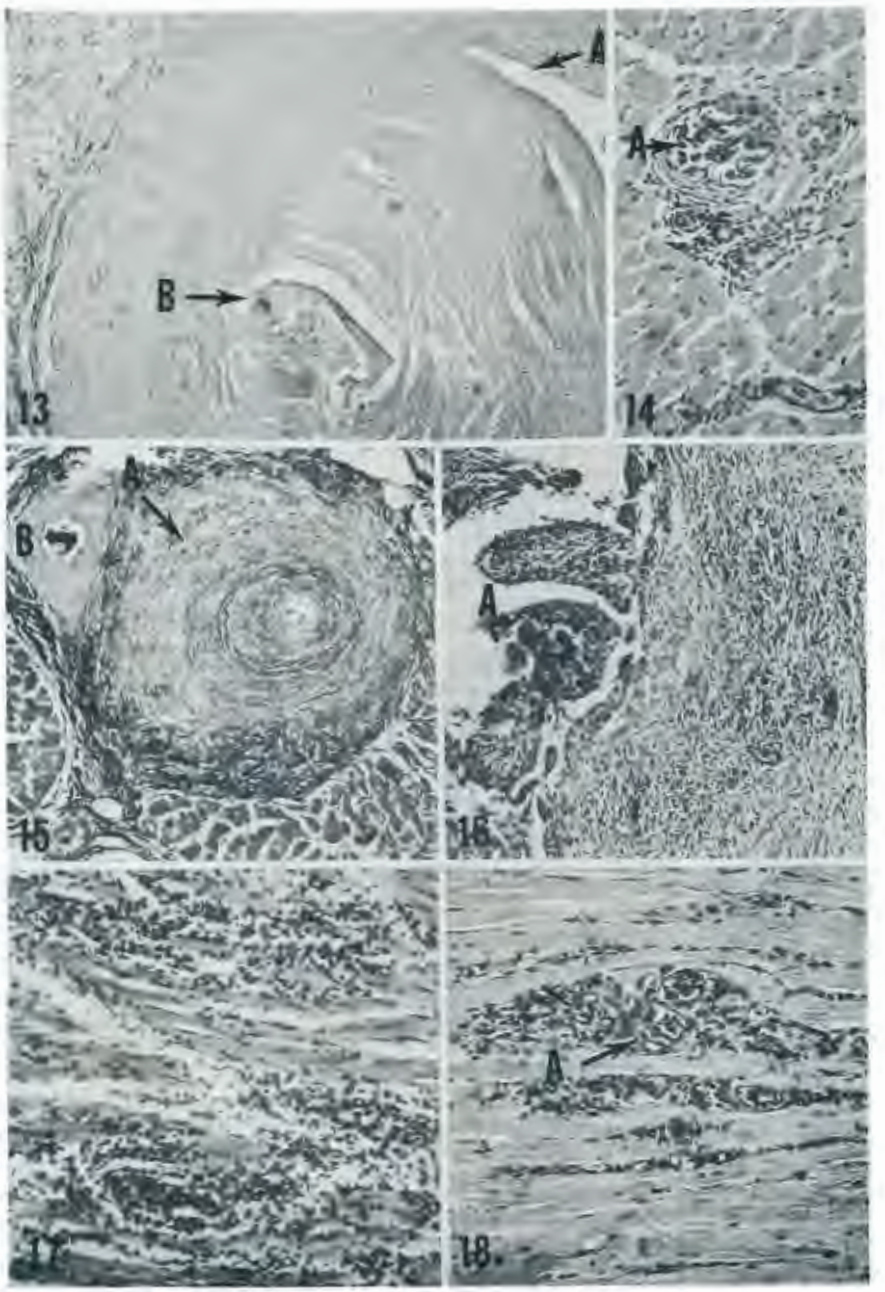
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PLATE 4.—Photographs prepared from kudu specimens

19. Myocardium showing scar tissue

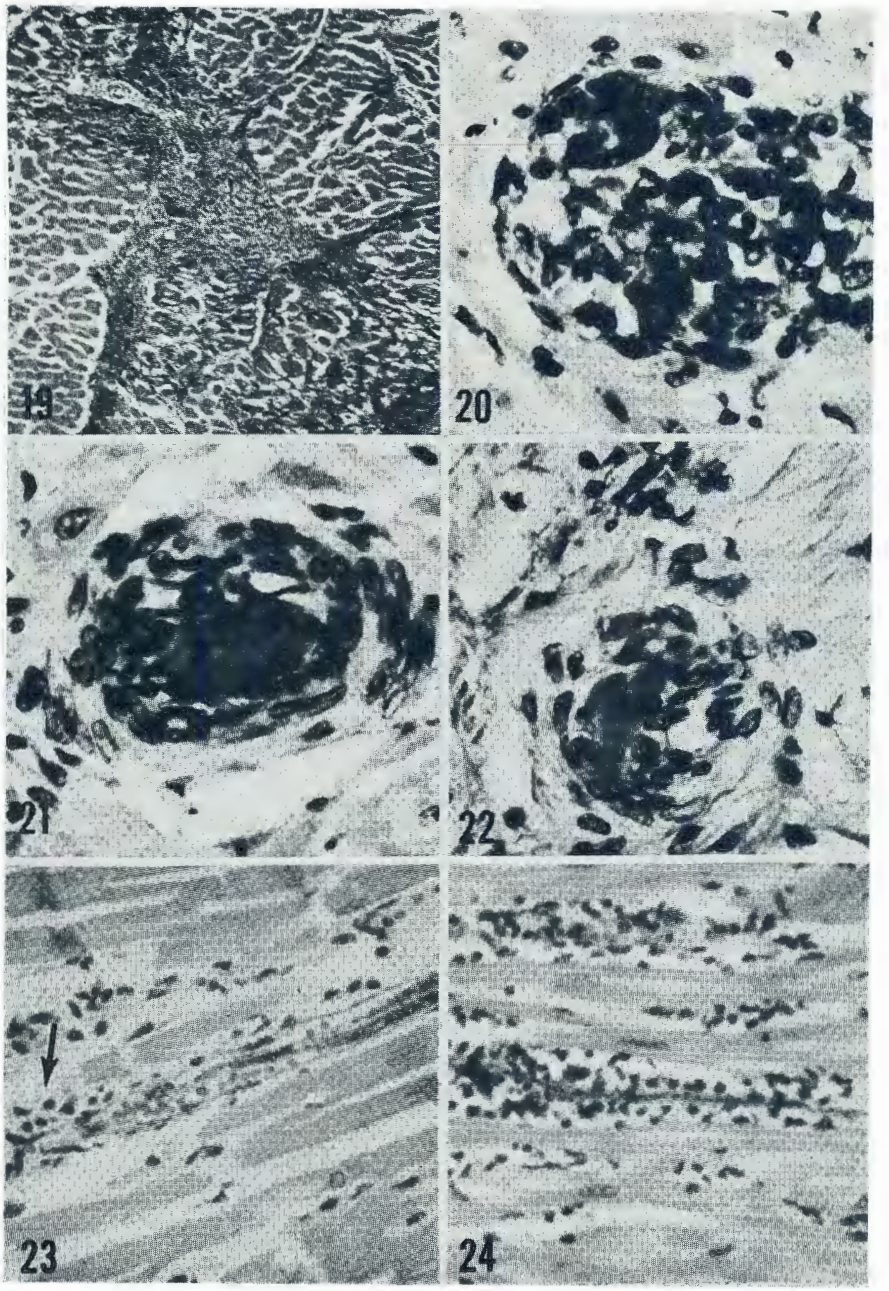
20. Small vein, myocardium. Leucocytes and hyperplasia of endothelial cells almost completely occlude the lumen

21. Small vein, myocardium. Along with hyperplasia of endothelial cells there was sometimes a homogeneous slightly basophilic fibrinous thrombus

22. Small vessel, myocardium. Fibrinous material was sometimes located subendothelially in vessel wall

23. Small vein, myocardium. Small round cells (arrow) were sometimes found around vessels in which microfilariae were present

24. Myocardium. Inflammatory cells surrounding this microfilaria were typical of reactions to many of the microfilariae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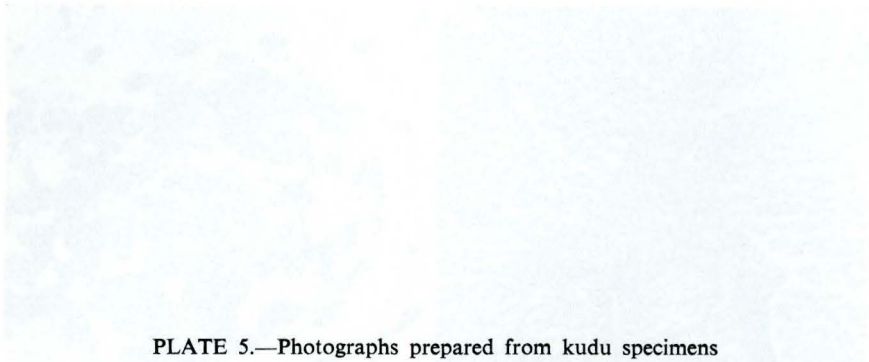

PLATE 5.—Photographs prepared from kudu specimens

25. Lung. Small branch of pulmonary artery. Lumen is practically filled by hyperplastic endothelial cells and eosinophiles

26. Lung. Small artery and vein adjacent to bronchiole (A). Hyperplastic endothelial cells and eosinophiles fill lumen of both the artery (C) and vein (B), while the adventitial tissue is infiltrated with inflammatory cells

27. Lung. Pulmonary vessel. Lumen of vessel contains rather active subacute reaction composed of villose proliferations and inflammatory cells. Adventitia and surrounding tissue also show inflammation

28. Large coarse projection (A) within the lumen of a branch of pulmonary artery with a segment of arterial wall (B) showing. Notice sheets of hyperplastic cells (C) covering the endothelial surface of (A) and (B).

29. Reaction within lumen of a branch of pulmonary artery. Active, chronic proliferation from the intima which shows mature coarse, club-shaped reactivated projections (C) infiltrated with inflammatory cells (A). Notice the small villose projections on their surface (B)

30. Contents of lumen of a branch of pulmonary artery. Small villose (A) and large coarse (B) projections were occasionally present within the same lumen

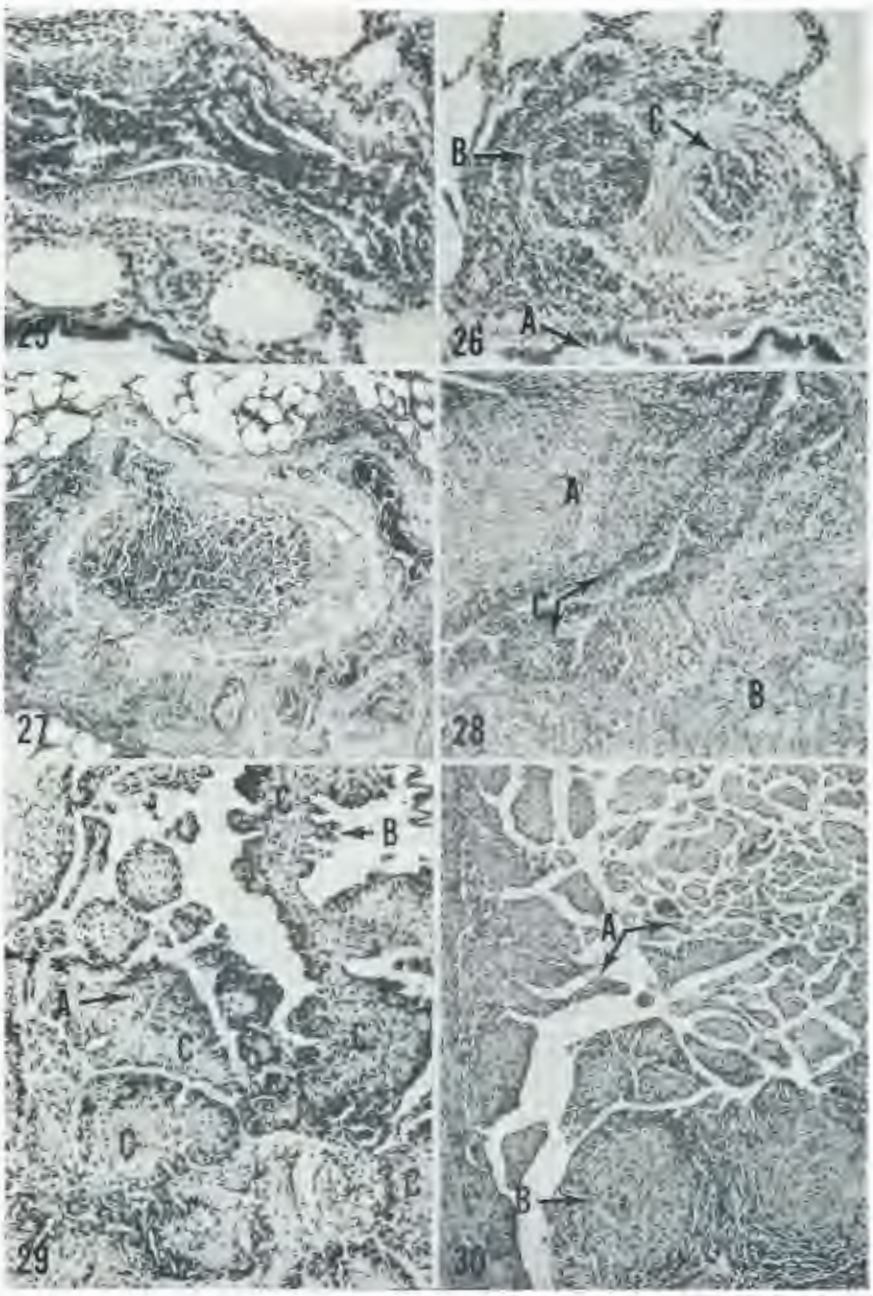
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PLATE 6.—Photographs prepared from kudu specimens

31. Lung (Masson stain). Wall of pulmonary artery (A) and projections into lumen (B). Notice that smooth muscle of the media (C) is continuous with that of the projections

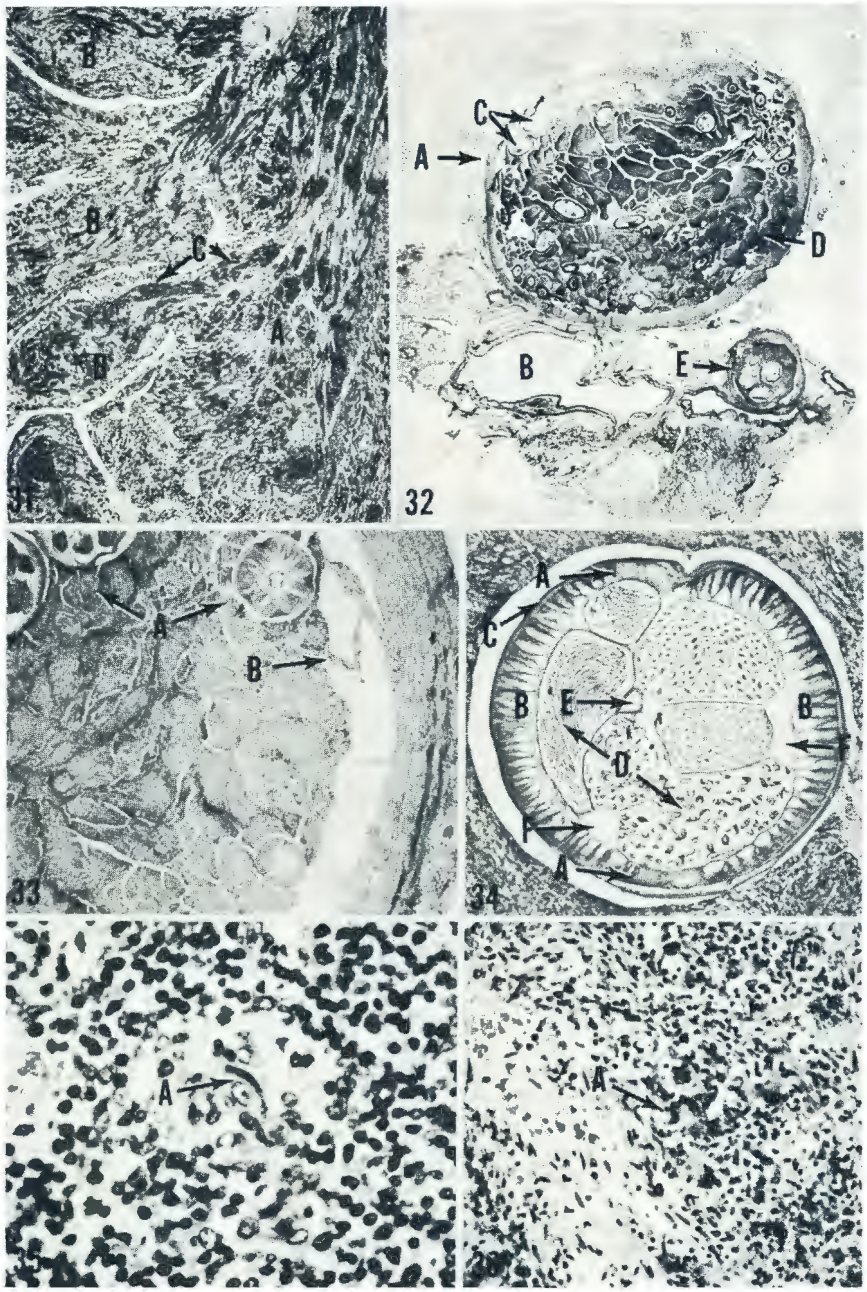
32. Lung. Aneurysm (A) within a branch of pulmonary artery accompanying a bronchus (B). The aneurysm which is much larger than the small bronchus which it accompanies contains segments of numerous parasites (C) and the chronic proliferative club-shaped projections (D). Notice smaller arterial branch containing a dense connective tissue mass (E) with incorporated dead parasitic remnants

33. Lung. Branch of pulmonary artery containing parasites (A) and reaction. In this artery projections were growing along course of the vessel's lumen with few attachments (B) to intima at this point

34. Lung. Transverse section of adult *C. sagittus* within lumen of a branch of pulmonary artery. Notice lateral cord cells (A), muscles (B), cuticula (C), segments of uterus filled with developing microfilariae (D), digestive tube (E) and body cavity (F)

35. Lymph-node. Lymphadenitis in response to microfilaria (A)

36. Lymph-node. Microgranuloma, which is adjacent to a trabecula, containing syncytial-type of giant cells (A)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