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ELAEOPHOROSIS IN WHITE-TAILED DEER: PATHOLOGY OF THE NATURAL DISEASE AND ITS RELATION TO ORAL FOOD IMPACTIONS

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ABSTRACT: The lesions of naturally occurring elaeophorosis in white-tailed deer (*Odocoileus virginianus*) were studied. Arterial changes caused by adult *Elaeophora schneideri* occurred mainly in cephalic arteries and were characterized by circumferential intimal thickening, disruption of the internal elastic lamina, and verminous thrombosis. Microfilariae caused focal necrosis and fibrosis in the myocardium, but produced only minor changes in other tissues. Radiographic studies indicated that *E. schneideri* can cause impairment of the cephalic arterial circulation in white-tailed deer. Eleven of 14 (78%) infected deer had oral food impactions, with sublingual impactions being most common. Seven deer with impactions had other oral pathologic conditions, such as gingivitis, loose or absent premolar and/or molar teeth, and remodeling and/or lysis of mandibular bone. The evidence indicates a relation between food impactions and infection by *E. schneideri* in white-tailed deer, but no definitive connection was established.

INTRODUCTION

Elaeophorosis due to the arterial worm, *Elaeophora schneideri*, is manifest as a variety of syndromes in wild and domestic ruminants in North America (Adcock and Hibler, 1969; Worley et al., 1972; Robinson et al., 1978; Pence and Gray, 1981). In white-tailed deer (*Odocoileus virginianus*), oral food impactions have been associated with infection by *E. schneideri* (Prestwood and Ridgeway, 1972; Hibler and Prestwood, 1981).

There are no detailed reports describing pathologic changes in white-tailed deer naturally infected with *E. schneideri*. In experimentally infected deer, nematodes produced plaques in the intima of carotid arteries. Microscopically there was subintimal thickening of carotid, leptomeningeal, and ventral spinal arteries; thrombosis of vessels of forehead skin; and vasculitis of retinal veins (Titche, 1976; Titche et al., 1979).

The pathogenesis of food impactions in white-tailed deer has not been determined. Prestwood and Ridgeway (1972) suggested that clinical manifestations were related to diminished blood flow caused by *E. schneideri* in the arterial system supplying the floor of the mouth. Experimental infections, however, produced few instances of arterial lesions that would significantly alter circulation, although one fawn died due to obstruction of a coronary artery by an immature *E. schneideri* (Titche, 1976; Titche et al., 1979). The purpose of this study was to describe the pathology of naturally occurring elaeophorosis in white-tailed deer and to further determine the pathogenesis of clinical manifestations.

MATERIALS AND METHODS

White-tailed deer with suspected elaeophorosis (facial swellings due to oral food impactions) were captured alive on South Island, Georgetown County, South Carolina. The deer were given 1.0 mg/kg xylazine hydrochloride (Rompun, Haver-Lockhart, Birmingham, Alabama 35214, USA) and delivered to Athens, Georgia. Sedated deer were anesthetized and maintained on fluothane (Halothane, Ayerst Laboratories, New York, New York 10017,

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USA). Electromyography was performed unilaterally on oral and facial muscles including buccinator, masseter, mylohyoideus, temporal, and tongue by using methods outlined by Bowen (1978). Subsequently, the carotid arteries were catheterized in the mid-cervical region and cephalic arteriography was performed on both carotid blood fields by using standard techniques (Douglas and Williamson, 1980). Electromyography and cephalic arteriography also were performed on a normal uninfected adult deer.

Arteriograms were quantitatively evaluated by comparing measurements of the diameters of arteries of clinically affected deer to the control deer. Differences in body size between infected and control deer were accounted for by dividing all measurements by the diameters of the respective common carotid arteries at the level of the third cervical vertebra.

Following radiography, animals were killed by exsanguination and major arteries were examined for adult specimens of *E. schneideri*. Tissues and representative lesions were preserved in 10% neutral buffered formalin. Coronal sections of brain were cut at 2–5-mm intervals. Sections of buccinator, masseter, mylohyoideus, temporal, and tongue muscles were processed separately. Eyes were removed whole and sectioned vertically through the optic nerve. All other tissues were processed by standard methods and stained with hematoxylin and eosin.

In addition to the above deer, a retrospective study was made of necropsy records and tissues of 12 deer that previously had been found to be infected with *E. schneideri*. Three of the deer had been submitted by state and federal wildlife agency personnel to the Southeastern Cooperative Wildlife Disease Study diagnostic laboratory from 1977 through 1983 as single case accessions. Two of the three above deer originated from South Island, and one was from Okefenokee National Wildlife refuge, Charlton County, Georgia. Nine additional deer were collected by shooting from South Island. Necropsies and examinations for *E. schneideri* generally were done as described above. Since this part of the study was retrospective, there was no standard protocol for collection of tissues.

RESULTS

Two deer were captured alive on South Island. One was an aged doe with a food impaction in the intermandibular area. Prior to anesthesia the deer was alert and active, and did not exhibit other obvious external abnormalities. Abnormalities of oral or facial muscles were not noted on electromyography. Arteriograms revealed similar changes in arteries of both carotid blood fields. Compared to the normal deer (Fig. 1), there was delay in filling and diminished arborization of arteries rostral to the bifurcation of the common carotids (Fig. 2). Marked bilateral absence of perfusion of cerebral arteries was evident (Fig. 2). Compared to the control deer, carotid arteries were dilated 1.7- and 1.9-fold at their greatest diameters. At the least dilatation (bifurcation of rostral rete branch), the diameters of the maxillary arteries were 1 and 1.6 times normal. The bases of the superficial temporal arteries were enlarged 1.2- and 1.3-fold. Silhouettes of specimens of adult *E. schneideri* were noted in the left common and external carotid arteries in dilated areas and in the left maxillary artery (Fig. 2). On the right side, parasites were seen only in the common carotid artery. Also, there was retention of contrast media at the base of the right superficial temporal artery, and the vessel was only intermittently perfused. At necropsy, 14 adult nematodes were distributed bilaterally in cephalic arteries, and two nematodes were found in the right femoral artery.

The other deer captured alive on South Island was a 1-yr-old male with pronounced bilateral impactions of the buccal spaces. Several months prior to capture, field personnel had noted a small swelling beneath the right ear. Physical examination at capture revealed that the deer was alert and in good flesh. Electromyography revealed diffuse fibrillation potentials in masseter, mylohyoideus, and tongue muscles and repetitive motor units in mylohyoideus muscles. Cephalic arteriography revealed one nematode in the right maxillary artery at the level of the rostral rete branch, but there were no other radiographic changes in the cephalic

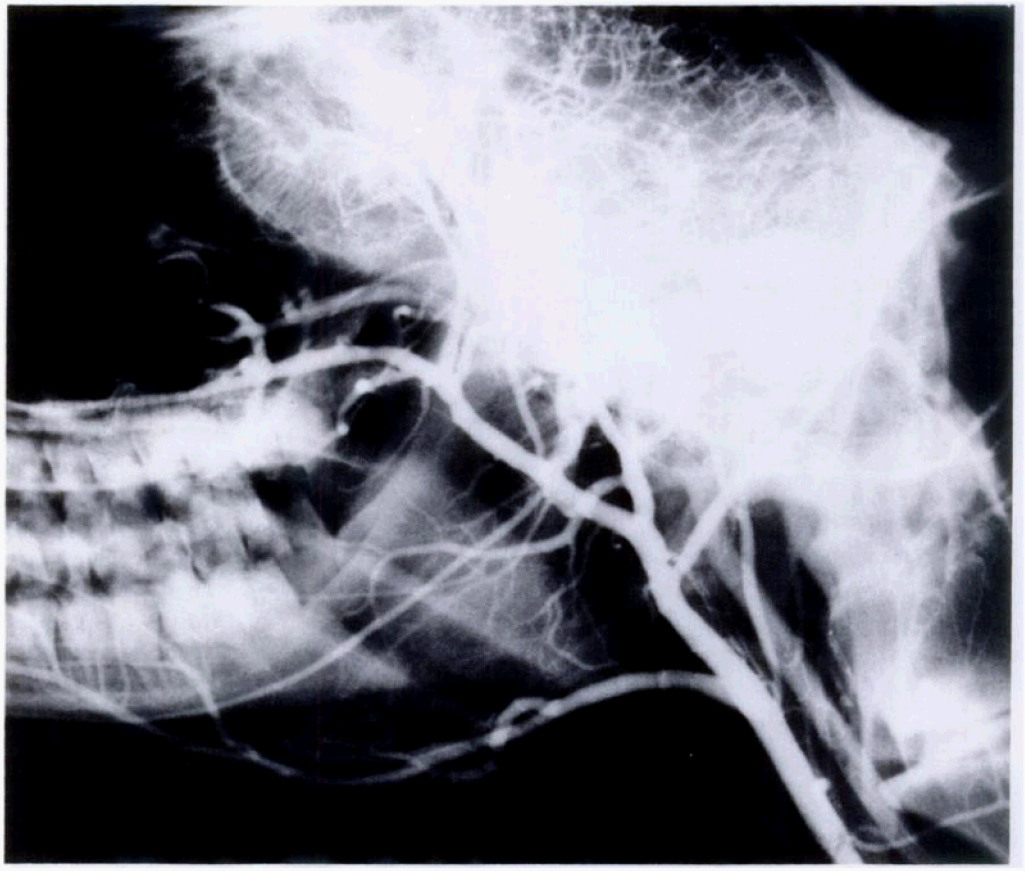

FIGURE 1. Cephalic arteriogram of right carotid blood field of a normal white-tailed deer.

arteries. At necropsy, one dead nematode was found partially attached to the wall of the right maxillary artery.

The results of pathologic investigations on the aforementioned deer were combined with those of 12 case accessions studied in retrospect. Of 14 naturally infected deer, 11 had oral food impactions. Of eight deer with sublingual food impactions, seven exhibited impactions on both sides of the frenulum, and one deer had an impaction on only one side. Buccal and salivary duct impactions each were noted in single deer. In another deer, a food impaction was not noted at necropsy, but resident biologists noted that the deer had a sublingual swelling for 1 yr prior to collection.

Externally, impactions appeared as swellings of the intermandibular or buccal regions (Figs. 3, 4); however, in one deer a smaller swelling in the maxillary region beneath the eye proved to be an impaction of the parotid salivary duct openings. Impactions consisted of dry, coarse ingesta, such as acorns, corn, grass, leaves, stems, and other partially masticated plant material (Fig. 5).

Intermandibular swellings were due to impaction of food in the sublingual spaces. Distinct masses of ingesta measuring 1–2 cm by 5–9 cm were separated by the frenulum and interfered with function of the tongue. Buccal impactions were approximately 6 cm by 10 cm and overdistended the spaces lateral to the cheek teeth (Fig.

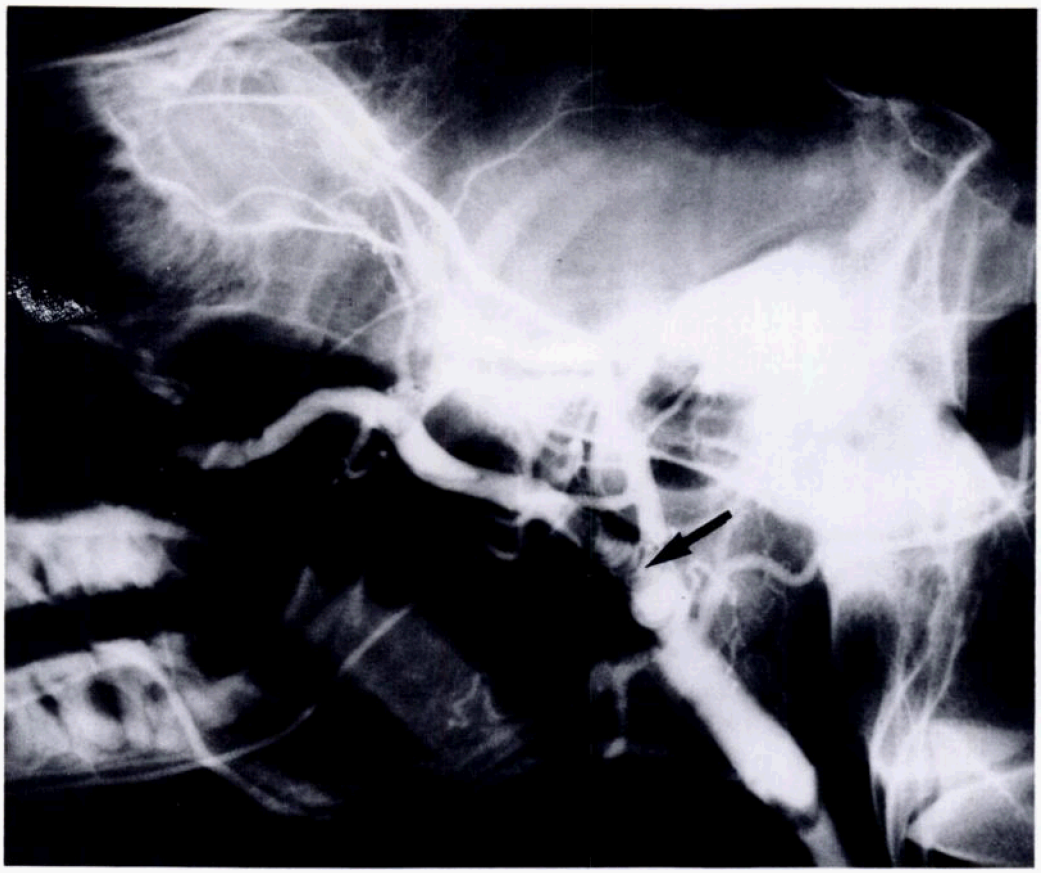

FIGURE 2. Cephalic arteriogram of right carotid blood field of a white-tailed deer infected with *Elaeophora schneideri*. (Note adult parasites within artery at arrow.)

4). In the deer with impaction of the salivary ducts, parotid and mandibular salivary duct openings were dilated (1–2.5 cm) by grass stems and hair. Ulcers on the gingiva and buccal mucosa were noted in most deer with impactions.

Six deer with food impactions had dental, mandibular, or maxillary abnormalities, including one or more of the following: gingivitis (n = 6); loose or absent premolar and/or molar teeth (n = 3); and remodeling/lysis of mandibular bone (n = 5). Mandibular conditions most often involved the region from the second premolar through the first molar (Fig. 6). Five deer with impactions had no other grossly visible oral pathologic conditions.

Deer also exhibited atrophy of mylohyoideus, buccal and masseter muscles (n = 3); unilateral blindness due to chronic pigmentary keratitis, corneal scars, and contraction of the globe (n = 2); and multiple unilateral or bilateral variably sized ($\leq$2.5-cm-diameter) cysts in the renal cortex and medulla (n = 4).

Infected deer ranged in age from 1.5 to 8.5 yr ($\bar{x}$ = 5.5 yr) and harbored one to 41 ($\bar{x}$ = 9) adult nematodes. Parasites usually were found at the bifurcation of the common carotid arteries. Nematodes also were lodged within the heart and in coronary, brachial, femoral, lingual, maxillary, and occipital arteries.

The intima of infected arteries was off-

FIGURE 3. External appearance of typical intermandibular food impaction in a white-tailed deer.

white or yellowish and had a villous or granular texture in areas where parasites were located. Five deer had thrombi in the following arteries: brachial, carotid, coronary, lingual, and maxillary. In two of these deer, dead nematodes were associated with thrombi; however, small (3–5-mm-diameter) thrombi with smooth surfaces were found in the carotid arteries of one deer along with live specimens of *E. schneideri*. Parasites were not associated with thrombi in two other deer. In two deer, the coronary arteries were thin-walled and tortuous, with multiple aneurysms. These deer also exhibited multifocal white streaks in heart muscle. The terminal common carotids, and portions of the maxillary arteries were dilated in one deer.

Microscopic lesions in arteries were noted in all deer from which arteries were collected. Focal, mild to moderate, smooth or villous, circumferential intimal thickening, disruption of the internal elastic lamina, and verminous thromboses (Fig. 7) were seen. The character and severity of arterial lesions varied depending upon whether parasites were alive or dead. In arteries with living parasites, the intima usually was uniformly thickened, the surface was smooth or villous, and often denuded of endothelium. Villi were composed of plump, round or ovoid cells in a network of collagen and reticulin fibers. The thickened intima was composed of a relatively hypocellular matrix of collagen, reticulin, and elastic fibers with sparse numbers of spindle-shaped cells, possibly myointimal cells. Cells usually were arranged circumferentially, although closer to the lumen they were oriented obliquely or radially. The internal elastic lamina was stretched, thickened, and reduplicated.

FIGURE 4. Bilaterial buccal food impaction in a white-tailed deer. (Courtesy Mr. Mark Epstein, South Carolina Wildlife and Marine Resources Department.)

Between segments of the lamina were large openings which contained migrating cells apparently from the tunica media.

Arterial lesions were more pronounced in arteries with dead specimens of *E. schneideri*. Verminous thrombi were composed of dead worms incorporated within an organized matrix characterized by an intense granulomatous reaction of fibrous connective tissue and multinucleated giant cells (Fig. 7). Multiple foci of necrosis and scattered focal infiltrates of polymorphonuclear leukocytes also were noted within the thrombi.

Three deer exhibited microscopic lesions in the heart as follows: (1) multiple irregularly shaped foci of cardiac fibrosis or necrosis with infiltration of lymphocytes, plasma cells, macrophages, and eosinophils (Fig. 8); (2) medial hypertrophy and degeneration of walls of small arteries; and (3) thrombosis of arteries within

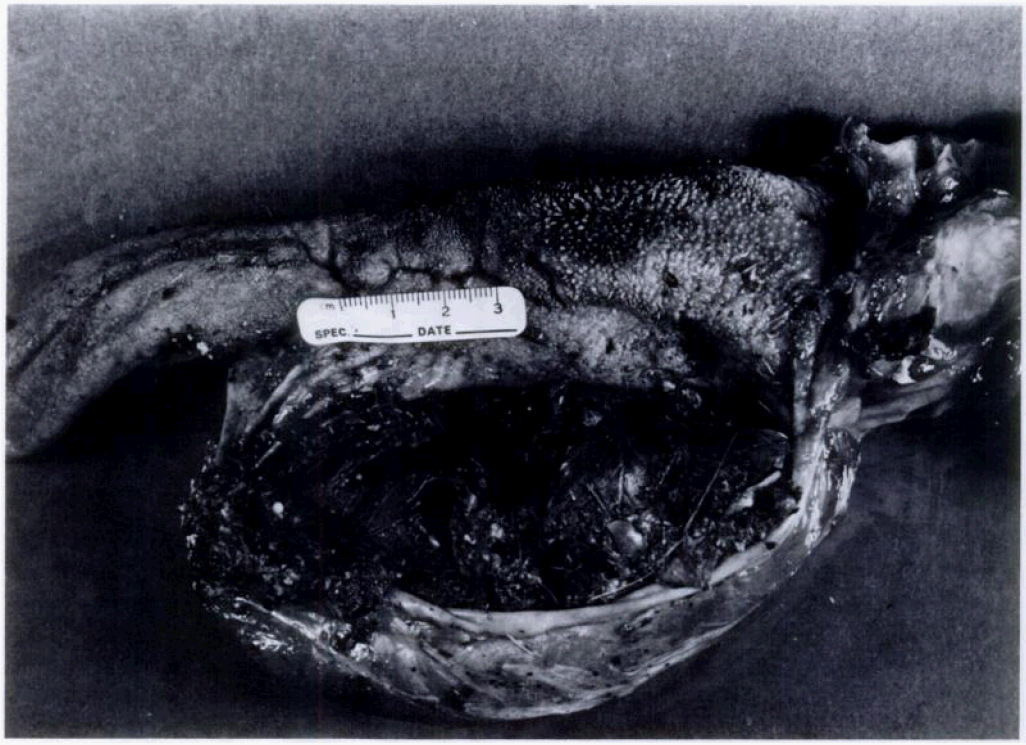

FIGURE 5. Impaction of coarse ingesta beneath tongue of a white-tailed deer.

areas of necrosis. Cross-sections of microfilariae of *E. schneideri* often were present in association with these lesions.

In forehead skin, there was multifocal to diffuse infiltration of lymphocytes, plasma cells, and eosinophils, primarily around small arteries in the superficial and deep dermis and hypodermis. Also noted were thrombosis of small arteries and degradation and inflammatory infiltration within connective tissue in the dermis. Sections of microfilariae of the size of *E. schneideri* were noted in capillaries and venous spaces in the middle to deep dermis and hypodermis, and infrequently in the superficial dermis. Microfilariae ranged from 6 to 12 μm in diameter, but the mean minimum and maximum diameters were 8.2 μm and 8.8 μm, respectively.

Thorough histologic examinations of brain and cephalic muscles were done in only three deer. Cross-sections of microfilariae of *E. schneideri* were noted within capillaries of some brain sections, but parenchymal lesions were not found. Lesions were not found in cephalic muscles, although cross-sections of microfilariae were noted in the mylohyoideus muscle of one deer. Microfilariae also were noted in the ciliary body of the eye of one deer.

DISCUSSION

The presence of nematodes and associated lesions in peripheral arteries, such as brachial and femoral, indicated that pathologic effects of *E. schneideri* are not limited to cephalic and coronary arteries as observed in experimental infections (Titche et al., 1979). Lesions in the arteries of naturally infected deer were similar to experimental cases, but were of greater severity. Artificially infected deer were necropsied on or before 30 wk post-infec-

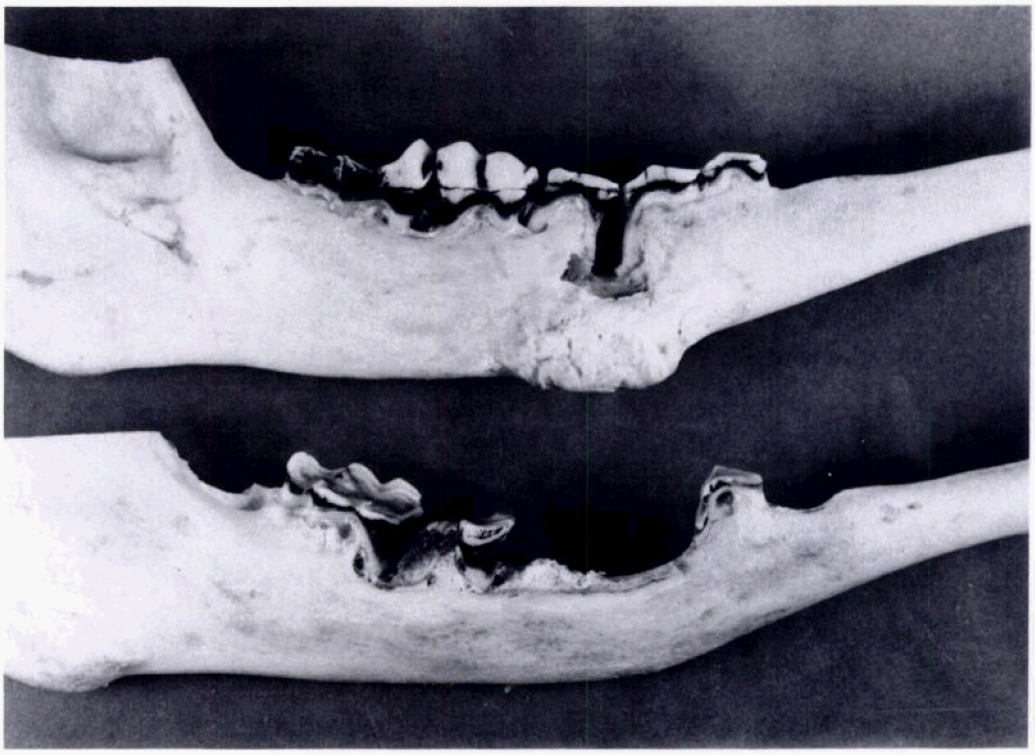

FIGURE 6. Mandibular and dental abnormalities of white-tailed deer with food impactions.

tion (Titche et al., 1979), and lesions probably had less time to develop than in natural cases, suggesting that duration of infection is related to severity of lesions. Also, Titche et al. (1979) did not have the opportunity to observe intra-arterial death of *E. schneideri*, which apparently produces pronounced granulomatous lesions within arteries (Fig. 7).

Studies both of experimental animals and of field cases have demonstrated that *E. schneideri* can produce coronary artery damage. Titche et al. (1979) previously reported that *E. schneideri* can produce occlusion of coronary arteries of deer. Coronary artery and cardiac muscle lesions in deer in this study also might be related to infection by adult parasites. The significance of myocardial lesions produced by microfilariae in natural cases is unknown. The presence of microfilariae in myocardium in the absence of adult parasites in heart or coronary arteries indicates that microfilariae deposited in arteries elsewhere in the body are small enough to move through capillaries and reach the venous circulation and be distributed to other sites.

Gross necropsy and radiographic studies indicated that *E. schneideri* can cause impairment of the cephalic arterial circulation in white-tailed deer; however, compromise of the circulation probably requires the presence of heavy bilateral infection with adult worms or bilateral lesions as a result of prior infection. There was no evidence of poor circulation on arteriograms of the deer with a unilateral infection with a single adult *E. schneideri*.

The relation between infections of *E. schneideri* and food impactions in white-tailed deer remains uncertain. On a circumstantial basis, the high number of infected deer with impactions on South

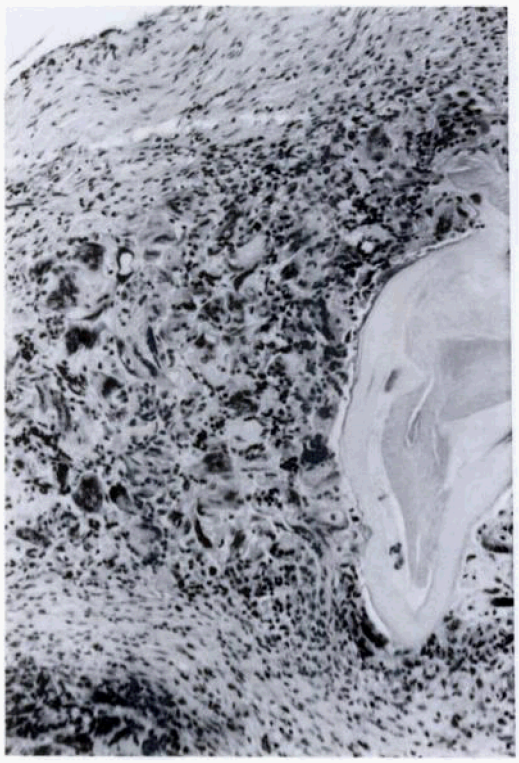

FIGURE 7. Organizing thrombus associated with a dead adult specimen of *Elaeophora schneideri*. (Note cross section of parasite at right.) H&E, ×100.

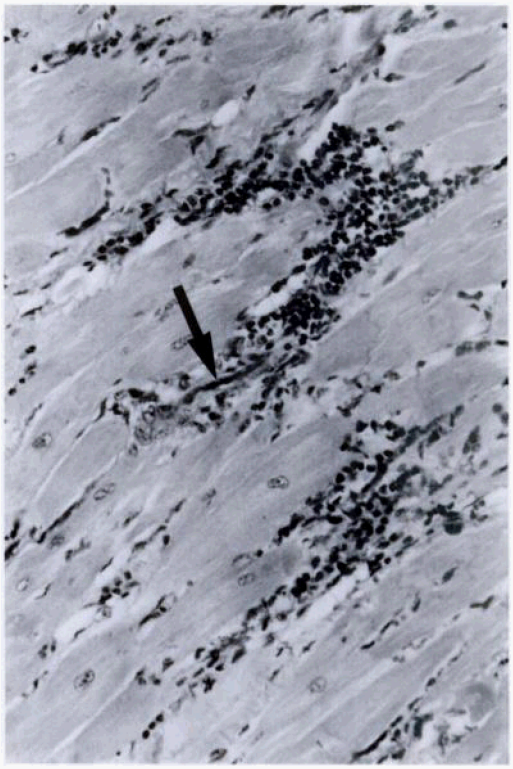

FIGURE 8. Mononuclear infiltration in myocardium associated with microfilariae of *Elaeophora schneideri*. (Note tangential section of microfilaria at arrow.) H&E, ×100.

Island suggests a cause and effect relationship. In the southeastern United States, sublingual impactions, which apparently are unique to white-tailed deer, have been reported only from areas where *E. schneideri* is enzootic in whitetails (Prestwood and Ridgeway, 1972; Hibler and Prestwood, 1981). Identical impactions also have been reported in white-tailed deer from Kerr County, Texas (Van Volkenberg and Taylor, 1943), and though the deer were not examined for arterial worms at the time of that study, more recent reports have indicated that the parasite is enzootic in Kerr County (Robinson et al., 1978; Waid et al., 1984). Neither of the latter workers reported food impactions in infected white-tailed deer, however.

Food impactions could be due to other causes that produce lesions in any cephalic structure important in mastication. Previous cases of buccal food impactions in goats (Williams, 1980), sheep (Shaw, 1981), and white-tailed deer (Cass, 1947) have been attributed to dental diseases. Furthermore, mandibular and dental abnormalities in deer in our study were similar in appearance and anatomic distribution to abnormalities which have been noted in other wild ruminants but ascribed to other causes. These cases include: mandibular lesions in caribou (*Rangifer tarandus*) (Doerr and Dieterich, 1979) and reindeer (*Rangifer tarandus*) (Leader-Williams, 1980); dental abnormalities in moose (*Alces americana*) (Ritcey and Edwards, 1958) and black-tailed deer (*O. hemionus columbianus*) (Cowan, 1946); and periodontal disease in chamois (*Rupicapra rupicapra*) (Pekelharing, 1974).

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