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PENTASTOME PATHOLOGY IN CAPTIVE REPTILES

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PENTASTOME PATHOLOGY IN CAPTIVE REPTILES

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PENTASTOME PATHOLOGY IN CAPTIVE REPTILES

CHAPTER I

INTRODUCTION

The Pentastomida, as adults, parasitize the respiratory tract of vertebrates and, except for two genera, are restricted to reptiles (Self, 1969). The pathology of pentastomes in man and animals has been studied by Cannon (1942), Fain (1960), Esslinger (1962), Self and Kuntz (1967), Self and Cosgrove (1968), Prathap et al. (1969), Cosgrove et al. (1970), Hopps et al. (1971), Self et al. (1972) and Self (1972). No such study of reptiles has been made.

From 1966 to 1970, while serving as parasitologist at the Oklahoma City Zoo, I encountered a number of reptiles infected with pentastomes. The relatively high percentage of reptilian pentastome infections associated with clinical disease suggests that pentastomes are more important as agents of reptilian disease than has been generally recognized.

As is the case with other potential pathogens, the identification of pentastomes in the host tissues is not, of itself, proof of a diseased state. It is the host response to the parasite which is indicative of disease. Even in the absence of a host cellular or humoral response, definite pathologic conditions, such as hemorrhage or mechanical trauma, may occur. The same basic pathophysiologic responses can be expected to pentastome

infections in reptiles as occur in other classes of vertebrates in response to a wide variety of disease agents, since the underlying similarities of the organisms far outweigh their differences. There is a limited number of reactive patterns to an infinite number of insults. The demonstration of a variety of morphologic lesions is to be expected in the case of a given parasite, depending upon the location, the numbers, the host immune state, and other factors. The same types of lesions are usually associated with similar parasites, and differentiation requires the demonstration of unique characteristics. In the case of pentastomes, the cuticle, the sclerotized mouth parts and hooks, and the penetration apparatus of the larvae provide excellent characteristics for identification, and these integumentary structures are often well-preserved in inflammatory foci (Figures 13, 20 and 27).

Reptiles are rarely infected with a single type of parasitic agent, and in captivity they are frequently afflicted with various diseases of metabolic and physiologic origin. The concomitant diseases greatly complicate the analysis of the role of pentastomes in reptilian pathology. Massive infections with Raillietiella orientalis in the Philippine Cobra, Naja naja philippinensis, was shown to be the result of auto-infection in an earlier study (Deakins, 1969 M.S. Thesis, The University of Oklahoma). It is the purpose of this dissertation to demonstrate that such massive infections are a major cause of pentastome pathology, and a significant cause of morbidity and mortality in captive reptiles infected with pentastomes.

CHAPTER II

HISTORICAL REVIEW

Except for a few observations, there are only passing references to pentastomes as agents of disease in reptiles. Hett (1924) attributed granulomata in snakes to the probable migration of larval raillietiellids. Weiss (1927) concluded that the pentastomes of North African lizards were inimical to their hosts, but did not elaborate. Heymons (1935) noted that females of Kiricephalus spp. and Armillifer spp. occasionally penetrated the lung of snakes. Rodhain and Vuylsteke (1932) encountered Leiperia sp. in the heart and aorta of crocodiles, and noted that this parasite also penetrated the mucosa of the bronchus, where the head occupied a cavity filled with lymphocytes. Fantham and Porter (1950) noted eosinophilia in helminth infections, including pentastomes in snakes, and found that in pentastome infections there was a hypersecretion of mucous, extravasation of blood, mucous congestion and partial perforation of the lung. Fain and Mortelmans (1960) described a tumor in the bronchus of a monitor lizard, Varanus komodoensis, attributed to a host response to Sambonia sp. An epithelioma, rich in eosinophils and metaplastic, is described, which almost totally occluded the inferior trachea. The presence of an adult female in the lung, and of nymphs in the trachea wall, was demonstrated. In 1964, Ms. Salazar reported deaths of cobras at the Manila Zoo caused by massive infections with Raillietiella sp., with no detailed descriptions.

Ardlie and Schwartz (1965) found that linguatulid larvae were associated with saccular aneurysms of the aorta in 31% of a series of Australian snakes, with as many as fifteen lesions in a single aorta. Most of the lesions were predominantly fibrocytic, with very sparse cellularity. Cellularity was considerable in some, with large foci of pigmented macrophages, numerous eosinophils and occasionally small, round mononuclear cells. Granulation tissue occurred in some lesions, and iron-laden macrophages were scattered throughout the lesions and within discrete foci in the adventitia. No identification of the genus or species was established, but Waddycephalus teretiusculus is the best known form from Australian snakes. Most of the Australian species of pentastomes are poorly known and belong to the family Sambonidae. Other pentastomes associated with Australian reptiles which do not belong to this family are Raillietiella sp. and Armillifer moniliformis.

Gray et al.(1966) reported parasitic granulomata in the stomach and colon of a Komodo dragon, Varanus komodoensis, as well as the parasite's eggs in the lungs. A small granuloma in one of the septa of the lung consisted of eosinophilic granulocytes, multinucleated giant cells, and an outer ring of fibrous tissue. Remnants of the parasite were found in the center of the lesion. Although no identification was established, it is likely that this was a case of pentastomiasis. Multiple granulomata were found in the stomach, the wall of the small intestine and colon, and were attributed to small nematodes. The autopsy report of the National Zoological Park (Smithsonian Year, 1967) indicates that pentastome larvae were found in characteristic granulomata in the stomach wall of an Agama stellio.

Nicoli and Nicoli (1966) reviewed the biology of the pentastomes, and

cited three references to pathology. Besides the tumor reported by Fain and Mortelmans (1960), they cite Noc and Curasson (1920) who encountered eggs and embryos in the cellular tissues of the coelom and intestinal wall of Python sebae, which Curasson (1929) hypothesized resulted from eggs entering the circulation. He postulated they became localized in the viscera and thus infected carnivores or scavengers feeding on the snakes. Fain (1961) also reported eggs and embryos in the coelom and various viscera of parasitized snakes. Giglioli (1923) and Faust (1927) suggested the portal system role in dissemination of pentastome larvae, and Haffner and Rack (1965) established this fact in Reighardia sternae infections of gulls.

Reichenbach-Klinke and Elkans (1965) treat the pathology of tuberculosis in reptiles, and review the diseases of cold-blooded animals, including pentastomiasis, but no description is given. The most recent and useful reviews of disease in captive reptiles are those of Page (1966) and Cowan (1968), but reflect how little is currently known of reptile pathology. Neither of these refer to pentastome pathology.

Several reviews of immunology of reptiles are available, including those of Evans et al.(1965) and Sprent (1968). None of these mention pentastome immunity in reptiles, but certain generalizations may be kept in mind. Snakes and other reptiles form antibodies to a wide variety of antigens; specific antibodies have been demonstrated for specific nematode infections (Timourian et al., 1961); the antibody response is temperature dependent; the development of immunity in amebiasis results in self-cure at proper temperatures; and anaphylaxis is easily provoked (Dessauer, 1970). The morphology of the reptilian circulating blood cells has been reviewed by Saint-Girons (1970).

Self and Kuntz (1967) have discussed host-parasite relationships of pentastomes in detail, and they found that in natural mammalian and reptilian hosts tissue-dwelling pentastomes elicit little if any pathologic response. They concluded that pentastomes occupying tissues of reptiles would be expected to be immature migrants destined for eventual maturity in the lung. They state:

> "It is interesting that so long as these parasites are in the natural host, whether mammal or reptile, they are capable of living in its tissues with little or no disturbance to the latter."

> "The tissue-dwelling habits of Porocephalus crotali and Kiricephalus pattoni described in this paper emphasize the ability of these pentastomes to live at random in their snake hosts with little apparent damage."

Self (1972) states that pentastomes live as free tissue nymphs in reptiles without producing pathological responses, but that visceral larva migrans effects may be produced by the migration of primary larvae. He notes that pre-sensitization may occur in both mammals and reptiles by an initial infection, with mononuclear cell response elicited by subsequent infections. He also notes that snakes have been observed at autopsy to have extensive lesions in the gut wall caused by primary larvae derived from auto-infection. He states:

> "In Kiricephalus pattoni infections of serpents, nymphs do not encapsulate but remain in the tissues until they are sexually mature and then migrate to the lung. There is little cellular response to the nymph, the normal tissue being in close proximity to it."

> "The adaptation of the host to the parasite is so complete as to result in little observable pathological effect."

> "Larva migrans syndromes would seldom if ever be associated with primary larvae, which are extremely difficult to detect."

Self (1972) also states:

> "It is not likely that the deteriorated nymph is often associated with the inflammation, because all that ultimately remains of it is a fragmented cuticle."

Self finds the most plausible explanation of the tissue responses is that inflammation associated with pentastomes always involves dead nymphs, which are relatively large and release large amounts of foreign protein into host tissues as they disintegrate. Inflammation, abscess formation, and ultimately granuloma formation occur if the host survives. He also suggests that the death of challenge nymphs in hosts that have been pre-sensitized is due to delayed hypersensitivity.

Self et al.(1972) studied experimental porocephaliasis in mice and the effect of challenge infection in sensitized mice. They report evidence that migration of nymphs follows natural passages such as the bile ducts, lymph channels and blood vessels. The tissue responses elicited by the host were typical of larva migrans except pronounced eosinophilia was not found. Extensive tissue damage and hemorrhage occurred, especially in sensitized mice. Presensitization was inferred from the lymphocytic response to challenge, and both the migration of the nymphs and their death were attributed to presensitization.

Life cycles

Inasmuch as auto-infection seems to be a major cause of pentastome pathology in reptiles, it is appropriate to review the life cycles. Although the life cycle of Linguatula serrata was one of the first parasite life cycles established (Leuckart, 1860), the life cycles of many pentastomes remain unknown. It is apparent from the literature that larval pentastomes are relatively non-host specific, while the adults are more spec-

ific, but ordinarily not confined to a single species of host (Sambon, 1922; Heymons, 1935; Fain, 1961; Nicoli and Nicoli, 1966; and Self, 1969). Accidental infections are relatively common.

The question of monoxenous versus heteroxenous development has been raised numerous times (Sambon, 1922; Giglioli, 1923; Heymons, 1935; Fain, 1961 and Self and Kuntz, 1967). In most, and probably all, genera, heteroxeny is the rule. It is difficult to prove that all stages of development are present in a single infection, and that those infections where several stages are represented do not result from multiple infection or parataenic transfer. Due to the frequency of massive infection in captive reptiles, it should be possible to demonstrate all stages of the pentastome in these cases if auto-infection occurs.

Fain and Mortelmans (1960) presented evidence of the direct development of Sambonia sp. in Varanus komodensis, but they did not demonstrate all stages of the life cycle. In 1964 Fain established the criteria he considered must be met to prove monoxeny: primarily the demonstration of the stage following the "primary larva." Haffner and Rack (1965) described the direct development of Reighardia sternae in gulls, demonstrating all stages of the pentastomes to be present, including the stage following the primary larva.

In an earlier study (Deakins, 1969, M.S. Thesis, The University of Oklahoma), I have demonstrated that a massive infection of Raillietiella orientalis in Naja naja philippinensis was due to auto-infection. In the present study, similar results are demonstrated in a number of Raillietiella species, two Kiricephalus species and in Sambonia species.

CHAPTER III

MATERIALS AND METHODS

Pentastomiasis was occasionally diagnosed in live reptiles at the Oklahoma City Zoo on routine fecal analysis, roentgenologic examination, or as a result of passage of adult pentastomes *per os* or *per nares*. With few exceptions, the materials on which this investigation is based were collected from reptiles which died in the collection. The animals were necropsied as soon as possible after death, or occasionally were euthanized, as post-mortem autolysis is usually rapid. Forty-eight pentastome infections occurring in two species of crocodylia, four species of lizards and twenty-three species of snakes are included in the study (see Appendix). The pentastomes belong to six genera and fifteen species.

Specimens for histological study were cold-fixed in buffered neutral 10% formalin, dehydrated via a standard alcohol series, cleared in xylene or chloroform, and embedded in Paraplast (melting point 57-58° C). Sections were cut at 5-20 micra, depending upon the tissue. Hematoxylin and esoin, and tissue Giemsa, were routinely employed; Gomori methenamine silver, periodic acid Schiff, Kinyoun acid fast, Gomori trichrome, and iron hematoxylin were employed for special purposes (Luna, 1968). Hematoxylin and eosin were used to detect evidence of degeneration and inflammatory lesions, and tissue Giemsa to detect parasites and bacteria. The special stains were employed in investigation of concomitant infections.

A single experimental life cycle was attempted during the course of this study, aimed at determining whether premunition was required to elicit a pathological response to pentastomes in reptiles. A single gravid female *Sambonia lohrmanni* obtained from a *Varanus* sp. at autopsy was administered by gavage to an *Elaphe obsoleta*. Nymphs recovered from this snake were later administered by gavage to each of five *Anolis carolinensis* and to a single *Natrix* sp.

CHAPTER IV

OBSERVATIONS

Histopathologic study of more than fifty cases of pentastome infection in reptiles reveals a variety of host responses, with lesions affecting virtually every organ system (see Appendix). The host response depends upon a number of factors, including the stage of pentastome present, the state of the pentastome (living or dead), the site of localization in the host, and whether the pentastome is encapsulated or unencapsulated, as well as the physiologic and immunologic state of the host. The host response includes inflammation, necrosis and other degenerative changes, repair and metaplasia. The inflammatory response is the process of primary concern, and it may be either acute or chronic in nature.

Acute inflammation

Eosinophils are the only abundant leucocytes of acute inflammation observed in reptiles in the course of this study (figures 16 and 17), and are the predominant leucocyte of the peripheral blood. These cells are the chief component of the dry pneumonitis frequently observed in reptiles and also in abscesses. The eosinophils are phagocytic and are often laden with bacteria. They are extremely abundant in the peripheral blood in some cases of pentastomiasis, but are not pathognomonic. They also show a localized infiltration about pentastomes in the tissues (Figures 24 and 25), around

larvae (Figures 16 and 17), the paths of migrating nymphs (Figure 22), around dead pentastomes (Figures 28 and 29) and both within and at the periphery of pentastome granulomata (Figures 19 and 23). Eosinophilia, however, is not a constant finding in reptilian pentastome infections, and eosinophil infiltration is not characteristic of all pentastome lesions. The role of the eosinophil in acute inflammation in reptiles is supported by its association with bacterial, mycotic and protozoal lesions, and as is frequently true in mammals, is probably linked to allergic manifestations.

Chronic inflammation

Chronic inflammation in reptiles, as in mammals, is characterized by the infiltration of round cells and mononuclear cells, with proliferation of connective tissue. The predominant cell types seen are lymphocytes, plasma cells, fibroblasts and multinucleate giant cells, primarily of the foreign body giant cell type. Langhans type giant cells are also encountered. Eosinophils are frequently encountered, particularly in the periphery of granulomata, but also within them (Figures 19 and 23). Calcification of granulomata is occasionally found (Figure 39). The extent and variety of the chronic inflammatory processes in pentastomid infections is shown in Figures 9, 10, 11, 13, 14, 19, 31 and 35.

The typical granuloma frequently contains the more sclerotized parts of the pentastome integument, the hooks and mouth parts (Figure 14). A series of granulomata in various stages, and presumably of different ages, illustrates the following sequence of development: the larvae are trapped and/or die (Figures 11, 30, 31, and 33), necrosis of the larvae occurs (Figures 13 and 39), and progressive degeneration is accompanied by the peripheral accumulation of epithelioid cells and fibroblastic proliferation.

Mononuclear cells infiltrate the surrounding tissue, and eosinophils may persist within the granulomata, especially at the periphery (Figure 19). The central area of the tubercle becomes increasingly eosinophilic (Figures 13, 19 and 21) and considerable hemosiderin may be present. Parenchymal degeneration is common, as evidenced by hydropic degeneration, fatty metamorphosis, changes in the tinctorial properties of the cells, and pyknosis. A stage of consolidation is found, and central necrosis is followed by resolution (Figures 13, 19, 20, 21 and 23). Ultimately a fibrous pseudotubercle or scar is all that remains (Figure 10). The granulomata result from larvae (Figures 11, 30, and 31), nymphs (Figures 19, 21, 23, 29 and 39), adults (Figure 28) and eggs (Figure 36). The formation of granulomata probably represents the tendency of the host to localize soluble antigens for pinocytosis, as well as the phagocytosis of particulate debris.

Granulomata are most commonly encountered in the intestine (Figures 10 and 13), where they are found in the mucosa, submucosa, muscularis and the serosa. The submucosa and serosa are the layers most likely to contain the primary larvae, nymphs and their remains, and the colon is the region most seriously involved. The intense granulomatous colitis is so severe in some cases as to almost occlude the intestinal lumen. There are marked changes in the muscularis (Figure 10), which has been seriously disrupted by cicatrization. The extensive fibrous proliferation results from migration of large numbers of pentastome larvae. The mucosa is ulcerated (Figure 10) and shows an increase inthe number of mucus-producing goblet cells (Figure 17). The serosa is also greatly altered (Figure 10).

Granulomata are also encountered in the liver (Figure 19), kidney, pancreas, heart and peritoneum. In the liver they are most frequent in the

portal areas (Figure 14 and 15) which are congested and occasionally show telangiectasis, fibrinoid degeneration and periportal inflammation. There are numerous parasitic plaques in the hepatic portal vein and its branches, and the smaller branches are frequently obliterated (Figure 14). The necrotic remains of pentastome larvae or nymphs are demonstrable in parasitic emboli in many instances, and in longitudinal sections of vessels in the liver they may have the appearance of a string of beads (Figure 14). Living nymphs may also be encountered in lumphatic vessels, veins and arteries (Figures 13 and 15). The bile ductules and hepatic arteries are usually spared, but occasionally the entire portal area may be obliterated.

Degenerative changes

A hemorrhagic necrosis of the lung was found in two cases, a water-moccasin (Agkistrodon piscivorus) infected with Porocephalus crotali and a Taiwan Beauty Snake (Elaphe taeniurus) infected with Kiricephalus pattoni (Figure 27). The significance of this finding is obscured by co-existing infections in both cases. Complicating lung infections in reptiles harboring pentastomes included hemosporozoan infections, secondary bacterial infections and infections with Aspergillus sp. and dematiaceous fungi, trematodes and nematodes. The death of adult pentastomes (Sambonia sp.) in the lung of Varanus sp. demonstrates the resulting intense inflammation (Figure 28).

The feeding sites of adult and juvenile pentastomes in the lung resemble those of many nematodes and trematodes, leaving a pedunculated papilla (Figure 12). This papilla is usually edematous, swollen at the end, and the epithelial cells are frequently disrupted, distorted, pale, with evidence of transudation and hemorrhage. Strands of mucus and fibrin are of-

ten present, and there is occasionally a lymphocytic infiltration. The epithelium may become denuded, giving a grazed-over effect, or may respond by becoming hyperplastic (Figure 36) or even metaplastic. Eosinophils are extremely abundant in a few cases, usually with a diffuse distribution suggestive of an allergic response. Frequently the adult and juvenile pentastomes in the lung occupy an emphysematous bulla or pseudocyst (Figure 12) created by the compression of the surrounding lung parenchyma. The alveolar wall immediately adjacent to the parasites is thin, while the surrounding alveoli are compressed and their walls are hyperemic.

The end result of infection may be such an accumulation of mucus, fibrin, hemorrhage, debris and inflammatory cells as to obliterate the lumen (Figure 38), predisposing to secondary infection and hemorrhagic diathesis. Hemosiderinophages are very prominent in the lung, liver and spleen, as well as being localized in granulomata.

Generally, the inflammatory response to living pentastomes, both nymphal and adult, is minimal. Many infections appear to be self-limiting. Dead pentastomes cause a marked infiltration of eosinophils and macrophages, and result in necrosis (Figures 28 and 29). The eggs of pentastomes may also elicit a marked granulomatous response (Figume 36), especially if there is pre-existing inflammation.

Adults in connective tissue elicit a lymphocytic response (Figure 35) and may create a hemorrhage-filled lesion in the adventitia of the lungs and intestine, or even feed on the liver. This may be due to the size of the females and an inability to obtain sufficient nourishment from the relatively small capillaries of the lung. It may also be due to development of delayed hypersensitivity by the host. The tissue propensity was observed

primarily in Sambonia sp. and Kiricephalus sp., where a lymphocytic response was noted to adults in ectopic sites in both cases. Inflammatory cells are common in the alimentary tract of the tissue-dwelling pentastomes and may be an important supplement to the erythrocytes which are the chief dietary component. Parenchymal cells are ingested by both nymphs and adults. The adults are efficient tissue penetrators as evidenced by the migration of adult Sebekia oxycephala in Alligator mississippiensis (Deakins, 1971).

Pentastome eggs are demonstrable in the feces of infected reptiles with gravid females in the lung, and they are encountered in sections of both lung and intestine (Figure 8). They are not demonstrated in this study in the coelom or other viscera (see pages 4 and 5). The larval exuviae are found in the lumen of the lung, in the alimentary tract, and are demonstrable in some granulomata (Figure 39). The larvae are demonstrated in the intestinal wall in infections with Raillietiella spp. (Figures 7, 9, and 11), Kiricephalus spp. (Figures 16, 17 and 40) and Sambonia lohrmanni (Figures 30 and 31). Presumptive identification of pentastome larvae is made in the intestinal wall of an Agkistrodon piscivorus infected with Porocephalus crotali in which eggs and hatched larvae are found in the intestinal lumen. This is also suspected in a Sebekia oxycephala infected Alligator mississippiensis with a chronic inflammatory process in the intestinal wall, but in which no larvae are demonstrated.

The larvae evoke an intense eosinophilia (Figures 16 and 17), mononuclear cell infiltration (Figures 7 and 9) or foreign body giant cell response (Figures 11, 30 and 31). They mechanically disrupt the intestinal epithelium, enter the lymphatics or vascular system, or migrate through

the tissues using their penetration apparatus and hooks (Figures 7 and 40).

The nymphs also cause considerable damage in their migrations, feeding on both parenchymal cells and blood, including inflammatory cells. Several hundreds of nymphs may be found, and they may cause considerable mechanical damage, and even fatal hemorrhage. In a newly imported Boa constrictor there were in excess of five hundred nymphs of three to five millimeters length, all migrating anteriorly from the area of the colon. There were numerous sites of hemorrhage in the mesenteries and evidence of use of the portal system by the nymphs. There were adults in the lung, but the origin of the nymphs was apparently from a heavily infected intermediate host fed to the snake after capture, or eaten soon before capture. The colon was extremely necrotic, and the snake died of apparent septicemic shock. There was hemorrhage from the mesenteric veins and liver, however. Similar hemorrhage was observed in experimentally infected snakes with very large doses of infective nymphs.

A marked eosinophilia is frequently seen in the vicinity of the nymphs (Figure 25) and in their migratory paths (Figure 22). One of the most striking features is the frequency of parasitic emboli in the liver (Figures 14 and 15). The more heavily sclerotized parts of the cuticle, the hooks and mouthparts, are often mecognizable and permit the precise identification of pentastomiasis. Peritonitis and septicemia are important complications of pentastome infections with larva migrans, as noted above.

Many of the subtle changes, such as staining quality of the cells, degeneration and necrosis, are probably due to the release of toxins by the degenerating parasites and necrotic tissues of the host, and to manifestations of delayed hypersensitivity. These include: diffuse perivasculitis

(Figure 33), fibrinoid arteritis (Figure 32), renal tubular degeneration and numerous glomerular changes, including mesangial proliferation and round cell infiltration, reticuloendothelial proliferation, hyperemia and hemosiderosis (Figure 14), and serous atrophy.

In addition, there is metaplasia in the regenerating livers of several snakes infected with Kiricephalus spp., with proliferation of ductal epithelium. Metaplasia is also encountered in the lungs of some snakes, and in the kidneys of two snakes. There are numerous pentastomid granulomata and marked degenerative changes in the kidneys of these snakes, with accumulation of large deposits of uric acid. The glomeruli are scarcely recognizable, consisting of a sparsely cellular fibrocytic scar, and large numbers of glomeruli have disappeared altogether. Uric acid deposits are also encountered in the intestinal wall and other foci in one of the snakes. No frank tumors are encountered in any of the reptiles of this study (see page 3).

Results of experimental infections

More than two hundred nymphs of Sambonia lohrmanni were recovered from an Elaphe obsoleta experimentally infected by gavage with a single gmavid female from autopsy of a Varanus sp. The experimental infection was interrupted at nine months, at which time the snake was in good health, feeding well, and showed no signs of infection externally. A single immature Sambonia lohrmanni was recovered from the lung, engorged with blood, and nymphs, both free and encapsulated, and were found in the musculature, mesenteries, subcutaneously, and throughout the viscera, including the myocardium, pericardium and adventitia of the aorta. There was no apparent inflammatory response to living nymphs. Microscopic examination of sections

of the tissues revealed numemous large eosinophilic granulomata, in various stages, especially in the submucosa of the intestine. The remains of Sambonia lohrmanni nymphs were demonstrable, and an eosinophilic infiltration was occasionally found about healthy unencapsulated nymphs. Larvae and larval granulomata were not demonstrated, suggesting that they had disappeared, since they must have been present at an earlier date, and in large numbers if the number of living nymphs is taken as an index. The intense reaction to the dead nymphs suggests that premunition has occurred.

Five encysted nymphs were administered by gavage to each of five Anolis carolinensis. Four of the lizards died in three to five days, and nymphs were recovered from the viscera. The fifth died on the tenth day and one immature Sambonia lohrmanni was recovered from the lung. Two other nymphs were located in the intestinal wall, still alive, but encysting, and another was dead, largely liquified, also in an early cyst in the intestinal wall. This was the only lizard suitable for histologic study, and it revealed extensive tissue migration damage, with extensive deposition of hemosiderin in all of the viscera, especially the lungs and liver. Twenty-five encysted nymphs were also administered to Natrix sp. at the same time, and autopsy at three months revealed no pentastomes or lesions.

CHAPTER V

DISCUSSION

Pentastomes may contribute to disease in reptiles in a variety of ways. One of the most important is the opening of portals of entry for micro-organisms, and provision of a rich substrate of exudate and hemorrhage on which they may grow. This is true of the larvae, nymphs and adults.

Adults are normally found in the lung, but occasionally undertake migrations, which is the general rule for the larvae and unencapsulated nymphs. Adult migrations are most often found in Kiricephalus spp., and in this genus the females frequently have their heads embedded deep in a crateriform lesion (Figure 2).

As with any other parasite, the size and numbers are an important index of pathogenicity, especially as related to metabolic impairment. These are also important factors in damage resulting from inflammation and deleterious immunological responses, as well as a measure of the mechanical damage compromising the function of vital organs. The number may range from one to hundreds, but the usual number of adults encountered is three to forty. The severity of adult infection may be visualized in some cases by roentgenological examination, but no effective treatment is known. Killing the adults would probably produce necrosis and serious immunologic sequelae. Judicious use of antibiotics might limit secondary infection.

In this study, pentastomes were not the sole or unequivocal cause of

death, although in several instances they were so numerous, and the damage they produced so severe, as to leave no doubt they were the original and probably the precipitating cause of death. Other disease processes were invariably present, including: bacterial and fungal infections, amebiasis, coccidiosis, trypanosomiasis, hemosporozoan infection, helminthiasis, and nutritional and environmental diseases.

Reptiles are the definitive hosts of all but two genera of pentastomes. Lizards and snakes may also serve as intermediate or parataenic hosts of Raillietiella spp., Sambonia spp., and Kiricephalus spp., and it is in these genera that auto-infection is most likely to occur.

The eggs of pentastomes normally pass from the lungs, through the alimentary tract, to reach the environment and infect intermediate hosts. The egg membranes provide an antigen-antibody barrier, but the eggs may be involved in granulomatous inflammation if pre-existing inflammation entraps the eggs, or if the eggs undergo degeneration.

The most serious pentastome infections are those in which large numbers of migrating larvae are present. This may occur in the natural intermediate host because of the sticky outer egg membrane, which promotes clumping and may thus insure multiple infection, both of the intermediate and definitive host. This could be a critical factor in a dioecious parasite with a relatively low frequency of infection, as is frequently true of the pentastomes. The role this may play in auto-infection is unknown. Certainly the most serious cases of pentastomiasis result from massive infections, and in captive reptiles usually from auto-infection. Similar cases have been observed in wild reptiles. Poor feeding, retention of feces and dehydration probably all contribute to the hatching of eggs in the intestine,

which is an active process on the part of the embryo, and the subsequent migration of the larvae. The larvae may become entrapped in the intestinal wall or migrate to other viscera, especially the liver. The circulatory system is commonly used by pentastome larvae as demonstrated by the parasitic plaques and emboli, and from nymphs found in vessels (Figures 13, 14 and 15).

The large eosinophilic abscesses encountered in pentastomid infections in reptiles are superficially similar to those sometimes encountered in fungal and other helminth infections, and other foreign body processes in mammals. Williams (1969) has studied the Splendore-Hoeppli phenomenon in fungal infections, and considers it a maximal host hypersensitivity, in which there is a lively local cell response and in situ antigen-antibody reaction. It is followed by varying degrees of central inflammatory cell necrosis to produce the various morphological variants seen in the different diseases. Such reactions are observed in pentastome infection response (Figures 13 and 21), in sharp distinction from the eosinophil infiltration noted earlier (Figures 17, 19 and 25).

Most of the pathologic changes associated with the adult pentastomes are due to mechanical trauma. In heavy infections anemia, anoxia and hypoproteinemia may occur, resulting in systemic impairment as fluid and electrolyte imbalance supervenes. Iron deficiency, vitamin deficiency, hypoglycemia and other changes may further complicate the clinical picture, and the hemopoietic and reticulo-endothelial systems are heavily burdened. Oxygen transfer may be further impaired by hypersecretion of mucus, accumulation of debris, hemosiderin-laden macrophages, and inflammatory cells in the lung (Figure 38). The traumatization of the lung epithelium, and resulting

exudation and hemorrhage all open the way for superimposed infection.

The cellular response of reptiles to pentastomes is similar to that of birds and mammals to a variety of foreign body reaction processes, and of reptiles to other types of infection. It is especially similar to schistosomiasis as regards the parasitic emboli which affect the liver, although in schistosomiasis these generally result from eggs and their subsequent degeneration.

The earlier work suggesting pentastome pathology (Fantham and Porter, 1950; Fain and Mortelmans, 1960; Salazar, 1964; and Ardlie and Schwartz, 1965) in reptiles is substantiated, and the studies of pentastome host-parasite relationships (Self and Kuntz, 1967; Self, 1972) are confirmed regarding the general lack of host reptilian response to the living adults and nymphs. Self et al. (1972) established the propensity of larvae and nymphs for utilizing natural avenues of dissemination, and reported acute cellular responses to second challenge infections in mice. Presensitized hosts may kill pentastome nymphs, and this is followed by allergic reactions to the proteins of the dead nymphs. Self (1972) has noted that the demonstration of the primary larvae in larva migrans syndromes is very difficult, but in the massive infections observed in captive reptiles it is much more likely. Furthermore, the deteriorated nymphs and larvae may frequently be identified in routine sections, because of the persistence of the sclerotized portions of the cuticle (mouth, hooks and penetration apparatus). The use of acid fuchsin or chromotrope 2R (trichrome stains) makes the identification of these parts easier. They are also PAS positive. Inflammation is not confined to dead nymphs, however, but can be demonstrated as a result of eggs, larvae, nymphs and adults, both living and dead.

Auto-infection does appear to be the major cause of pentastome pathology in captive reptiles, and it seems that the host immune response is an important part of the damage attributable to the pentastomes. The ease with which anaphylaxis is provoked in reptiles (page 5) may be an important factor. Due to the large size of pentastomes, and their migrations through tissues, they appear well-suited to the study of the immune response of reptiles to parasites.

The response of the experimentally infected Elaphe obsoleta to Sambonia lohrmanni indicates that a single dose can elicit the typical response as nymphs die over a period of months. The fact that no larvae or larval granulomata were encountered suggests that repair is fairly complete in the absence of continuing auto-infection. The syndrome is magnified in cases of auto-infection where eggs are continually released by adults in the lungs, hatch in the intestine, and larvae are constantly invading the gut wall, often in massive numbers. The experimental infections of Anolis carolinensis challenged with Sambonia lohrmanni nymphs emphasize the mechanical damage done by pentastome migrations.

CHAPTER VI

SUMMARY

The primary larva of three genera of pentastomes is reported and figured in the host intestinal epithelium for the first time. Varied pathologic changes are reported, including parasitic plaques of the blood vessels and parasitic emboli in the hepatic vein branches.

Definite tissue responses are demonstrated to eggs, larvae, nymphs and adults, both living and dead. The host responses include the acute inflammatory response, chronic inflammatory response, and granulomatous foreign body giant cell response. Certain resemblances are found to the Splendore-Hoeppli phenomenon, suggesting that hypersensitivity plays an important role in pentastome pathology in reptiles.

The underlying causes of auto-infection are not established, but it is auto-infection and constant exposure to increasing levels of antigen that cause the greatest problem with pentastomes in captive reptiles. Anaphylaxis may be the final result of auto-infection, but this has not been demonstrated experimentally.

The eosinophil of reptiles is probably a combination of the heterophil and eosinophil of mammals and birds, which would explain its presence in both acute inflammation and in presumed hypersensitivity or allergic states, as when it is diffusely distributed at the interfaces of the internal and external environments, such as the lung and intestine. The fact that there

are no neutrophils in the majority of reptiles tends to support the hypothesis that these characteristics are combined in a single type of heterophil in these animals.

The presence of lymphocytes and plasma cells in the chronic inflammatory response in reptiles is taken as evidence of an antibody response. There is probably premunition in reptiles, as these are the cells so engaged in birds and mammals. The study of these phenomena would appear to be the next logical step in investigations of pentastome host-parasite relationships in reptiles.

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APPENDIX

KEY: STAGES: Ad=Adult N=Nymph L=Larva E=Egg RESPONSE: A=Acute C=Chronic G=Granulomatous D=Degenerative changes N=Necrosis SYSTEMS MOST AFFECTED: Al=Alimentary R=Respiratory Ci=Circulatory Ex=Excretory M=Muscular N=Nemvous I=Integumentary OTHER DISEASES: Nu=Nutritional Am=Amebiasis Hg=Hemogregarines Hm=Helminths Fu=Fungi B=Bacterial OTHER: Exp=Experimental infection NP=Not processed 1/2 = 1 infected/2 examined

HOST	PENTASTOME	STAGES	RESPONSE	SYSTEMS AFFECTED	NOTES
CROCODYLIA:					
Alligator mississippiensis	Sebekia oxycephala	Ad-E	C-G-D	Al-R-Ci-Ex	7/7; Nu-Hm
Osteolaemus tomistomi	Sebekia sp.	Ad-E	A-C-G-D	Al-R-Ci-Ex	1/1; Hm
LACERTILIA:					
Anolis carolinensis	Sambonia lohrmanni	N	A-D-N	Al-R-Ci-Ex-M	5/5; Exp.
Chamaeleo dilepis	Raillietiella sp.	Ad			1/1; NP
Gecko gecko	Raillietiella sp.	Ad-N-E		Al-R	1/1; NP
Varanus niloticus	Sambonia lohrmanni	Ad-N-L-E	A-C-G-D-N	Al-R-Ci-Ex-M	1/2; Hm

HOST	PENTASTOME	STAGES	RESPONSE	SYSTEMS AFFECTED	NOTES
OPHIDIA:					
Agkistrodon piscivorus	Porocephalus crotali	Ad-N-L-E	C-D-N	Al-R-Ci	2/5; Am-Hg-Hm
Bitis arietans	Armillifer sp.	Ad			1/1; NP
Bitis gabonica	Porocephalus subulifer	Ad	C-D-N	Al-R	1/2; B (acid-fast)
Bitis nasicornis	Raillietiella boulengeri	Ad-N-L-E	C-G-D-N	Al-R-Ci-Ex	1/2; Hm
Boa canina	Raillietiella sp.	Ad			1/1; NP
Boa constrictor	Porocephalus clavatus	Ad, N	A-C-D-N	Al-R-Ci-Ex-M	3/6; B-Fu-Am-Hg-Hm
Boa hortulina	Raillietiella sp.	Ad-N-	A-C-D	Al-R-Ci-Ex	1/1; Fu-Hg
Bothrops atrox	Raillietiella furcocerca	Ad-L-E	A-C-G-D-N	Al-R-Ex	1/1; Hg
Chrysopelea ornata	Raillietiella sp.	Ad	A-C-D	Al-R-Ci-Ex	1/1; B-Am
Crotalus atrox	Porocephalus crotali	Ad-N-E	A-C-D-N	Al-R-Ci-Ex-M-I	1-3% average; Am-Hg-Hm
Drymarchon corais	Kiricephalus coarctatus	Ad-N-L-E	A-C-D	Al-R-Ex-	2/3; B-Hg

HOST	PENTASTOME	STAGES	RESPONSE	SYSTEMS AFFECTED	NOTES
Drymarchon corais	Raillietiella furcocerca	Ad-N-L-E	A-C-G-D	Al-R-Ex	1/3; B-Hg
Dryophis nasuta	Raillietiella sp.	Ad	A	R	1/1; NP
Elaphe obsoleta	Sambonia lohrmanni	N-L	A-C-G	Al-R-Ci-Ex-M	1/1; Exp
Elaphe taeniurus	Kiricephalus pattoni	Ad-N-L-E	C-G-D-N	Al-R-Ci-Ex-M-I	1/1; Hg
Eunectes murinus	Kiricephalus coarctatus	N		Al	1/1; Hg-Hm-Trypanosomes
Farancia abacura	Kiricephalus coarctatus	Ad-N-L-E	A-C-G-D-N	Al-R-Ci-Ex-M-I	1/1; Hm
Lachesis muta	Porocephalus stilesi	Ad-L	C-G-D-N	R-Ci-Ex	1/1; Hg
Lampropeltis getulus	Kiricephalus coarctatus	Ad		R	1/1; NP-Hm
Naja naja	Raillietiella orientalis	Ad-N-L-E	A-C-G-D-N	Al-R-Ci-Ex-M	3/5; Hm
Natrix sp.	Kiricephalus coarctatus	Ad-N-L-E	A-C-G-D-N	Al-R-Ci-Ex-M-I	2/5; Hm
Oxybelis acuminata	Raillietiella sp.	Ad-N-E	C-G	Al-R-Ci-Ex	1/1; Hm

HOST	PENTASTOME	STAGES	RESPONSE	SYSTEMS AFFECTED	NOTES
Python regius	Armillifer sp.	Ad			1/1; NP-Hm
Thamnophis sirtalis	Kiricephalus coarctatus	Ad-N-L-E	A-C-G-D-N	Al-R-Ci-Ex-M-I	1/1; Hm

PLATE I

Figure 1. Kiricephalus coarctatus in the lung of Farancia abacura, one-half actual size. An adult female and two males are seen in the opened lung. Note the spiral shape of the female, which is probably due to growth in a confined space. This character was once used to establish a new species, K. spiralis.

Figure 2. Kiricephalus coarctatus in the lung of Thamnophis sirtalis, one-half actual size. An adult female and two males are seen in the membranous lung. The female has penetrated the lung and her head is buried in the adventitia of the lung and the hypaxial musculature.

Figure 3. Kiricephalus coarctatus nymph in liver of Farancia abacura, one-half actual size. The nymph has penetrated the capsule of the liver with considerable hemorrhage into the coelom. Note another site of hemorrhage beneath the capsule is to the right.

Figure 4. Kiricephalus coarctatus nymph in the ventral musculature of Farancia abacura and associated petechia. One-half actual size.

Figure 5. Large number of Kiricephalus coarctatus nymphs beneath the renal capsule, with sub-capsular hemorrhage, in the kidney of Farancia abacura. One-half actual size.

Figure 6. Nymphs of Raillietiella orientalis in a portion of the vascular lung of Naja naja philippinensis. One-half actual size. Adult females were approximately 30-60 mm., males 10-25 mm. long (not shown). This lung contained 500+ pentastomes.

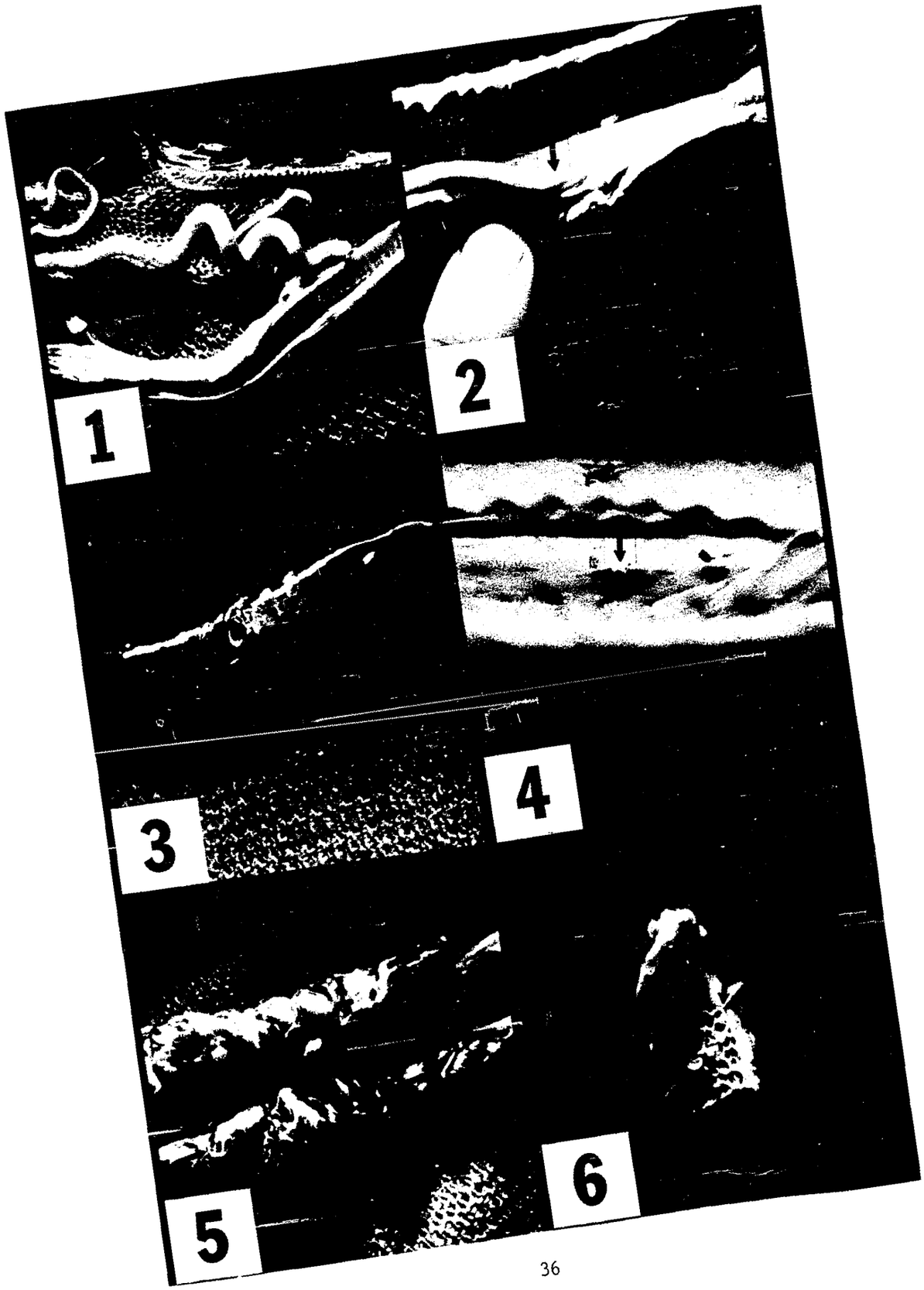
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PLATE II

Figure 7. Larva of Raillietiella orientalis migrating in the serosa of the colon of Naja naja philippinensis. The larva has an extended parapodium which bears a sclerotized hook, and the mouth-ring may be distinguished, as well as the large subesophageal ganglion. There is a prominent round cell infiltration and numerous pyknotic nuclei. H and E. 320 X.

Figure 8. Egg of Raillietiella boulengeri in the intestine of Bitis nasicornis. H and E. 250 X.

Figure 9. Larva of Raillietiella orientalis in serosa of colon of Naja naja philippinensis adjacent to atypical foreign body giant cell granulomata. Round cell infiltration. H and E. 80 X.

Figure 10. Section of the colon of Naja naja philippinensis showing extent of fibrosis. The mucosa is eroded, hypermic and infiltrated with eosinophils. The submucosa and serosa are markedly fibrotic with large numbers of granulomata, readily identified as Raillietiella larvae. H and E. 40 X.

Figure 11. Early granuloma formation with intact Raillietiella nymph in serosa of colon of Naja naja philippinensis. A necrotic nymph is seen below. H and E. 250 X.

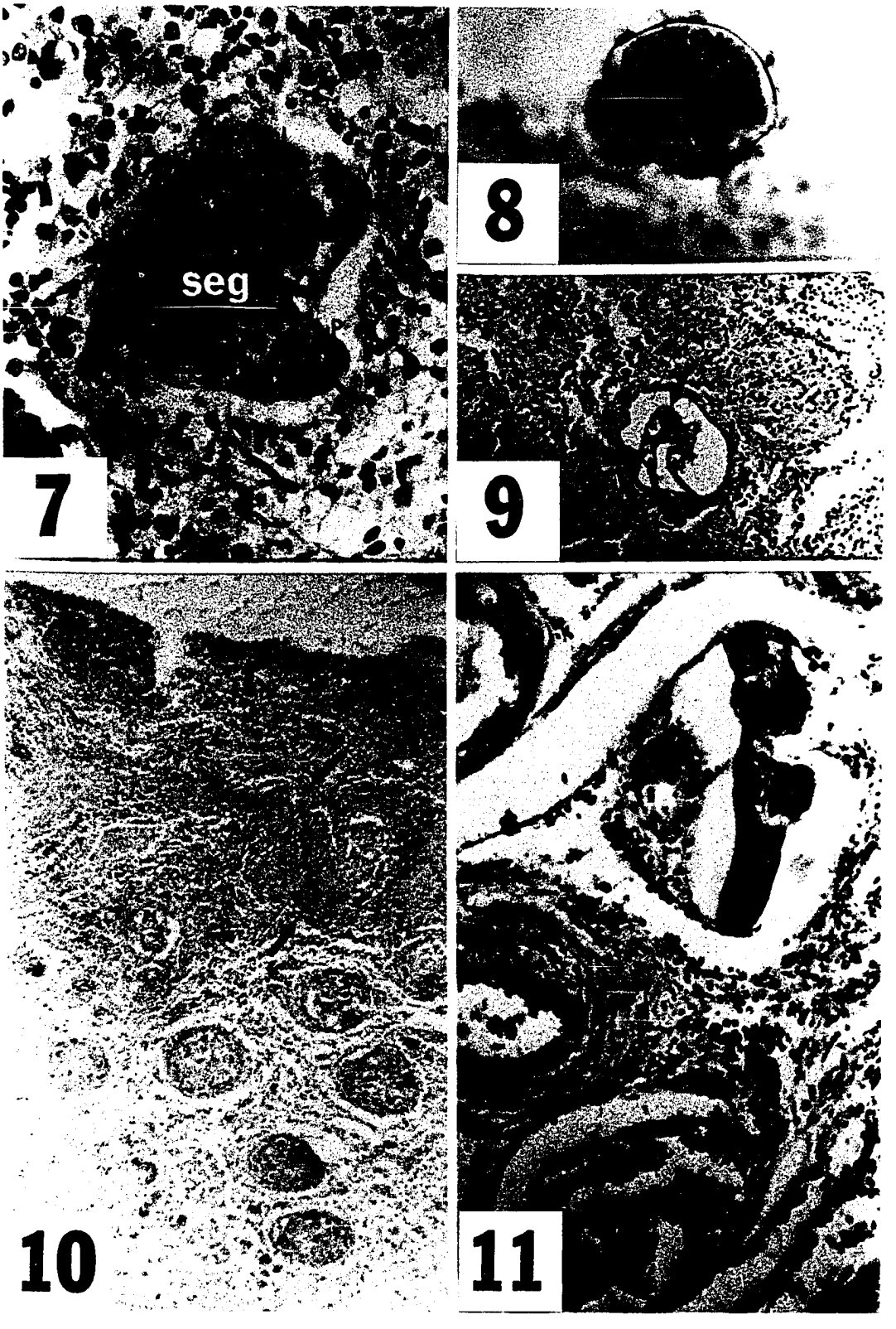
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PLATE III

Figure 12. Raillietiella orientalis adults and nymph in the lung of Naja naja philippinensis. Note the characteristic feeding sites (arrows), the bullous pseudocyst occupied by the pentastomes, and the accumulation of mucus and debris in the lung lumen. H and E. 40 X.

Figure 13. Raillietiella orientalis granulomata in the serosa and muscularis of Naja naja philippinensis colon. Note the two viable nymphs (arrows) and various stages of necrosis and granuloma formation. Note also the extent of fibrosis, altering the appearance of the muscularis. H and E. 40 X.

Figure 14. Three Raillietiella larval granulomata within a branch of the portal vein (arrows), indicating the vascular route of migration. The two larger granulomata contain identifiable chitinous remains. H and E. 40 X.

Figure 15. A viable Raillietiella nymph in the hepatic portal vein of Naja naja philippinensis (upper arrow), and a thin fibroelastic membrane partitioning the vein. Below is a granuloma resulting from a pentastome embolus in the vein, infiltrated by granulation tissue. H and E. 100 X.

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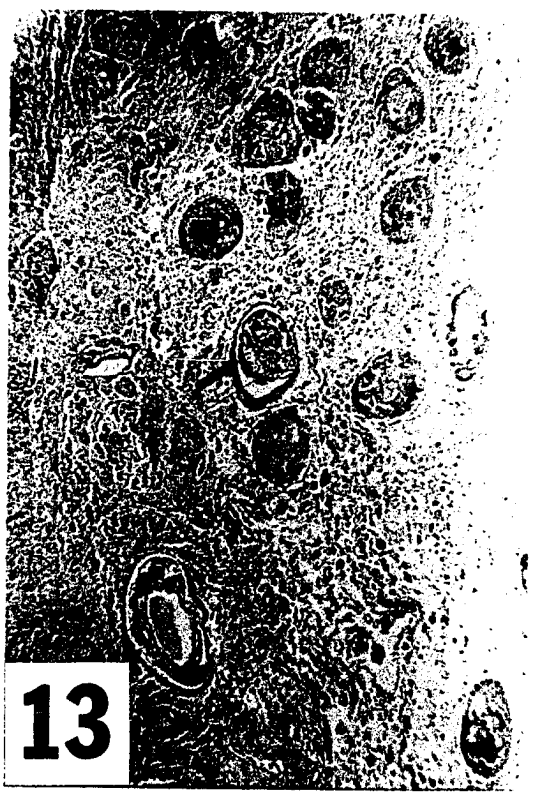

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PLATE IV

Figure 16. "Primary larva" of Kiricephalus coarctatus in intestinal mucosa of Farancia abacura. H and E. 400 X.

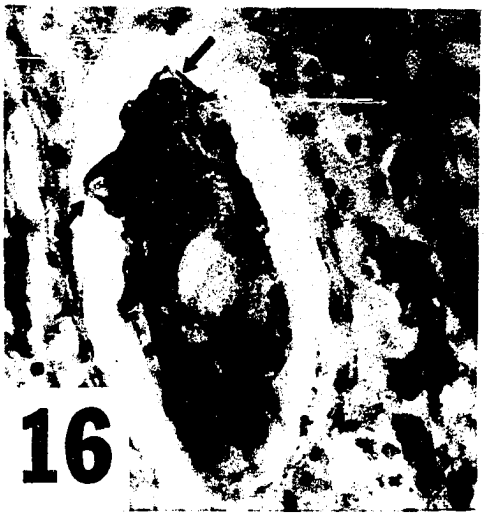
Figure 17. Same. 125 X. Note the intense eosinophil infiltration.

Figure 18. Obliteration of portal triad and lymphocytic infiltration following pentastome embolus in liver of Elaphe taeniurus infected with Kiricephalus pattoni. H and E. 125 X.

Figure 19. Atypical foreign body giant cell response to Kiricephalus coarctatus in kidney of Natrix. Note eosinophils peripherally. H and E. 250 X.

Figure 20. Necrotic remains of Kiricephalus coarctatus in serosa of intestine of Drymarchon corais invaded by granulation tissue. The hooks are clearly discerned and there is no marked inflammatory response evidenced at this time. H and E. 125 X.

Figure 21. Numerous pentastomes (Kiricephalus coarctatus) in serosa of intestine of Thamnophis sirtalis. Large eosinophilic granuloma is suggestive of Splendore-Hoeppli phenomenon. H and E. 40 X.



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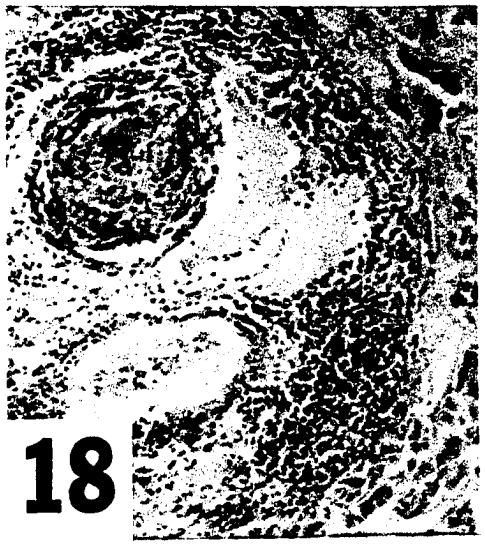

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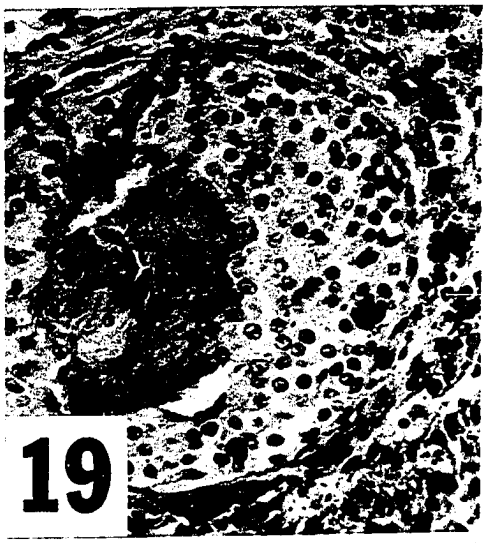

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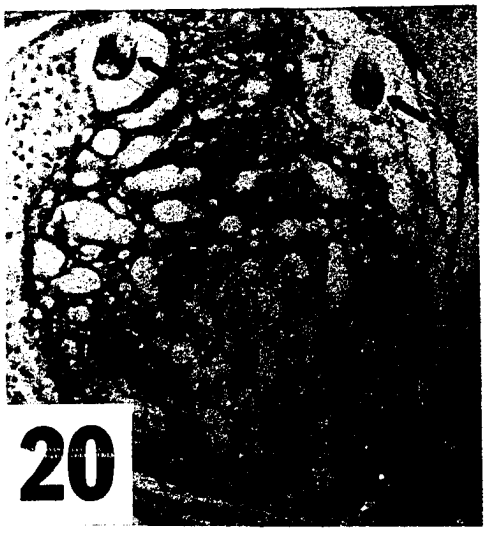

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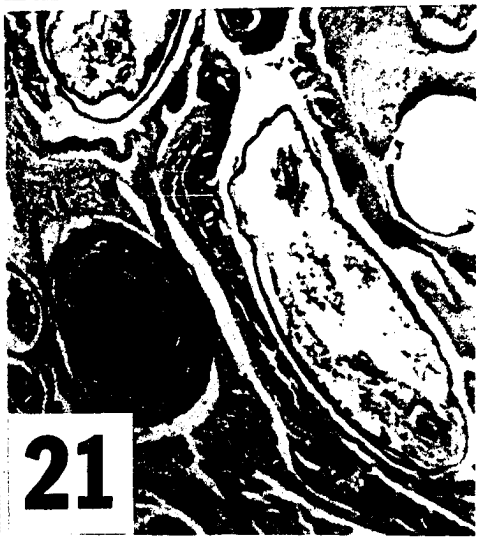

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PLATE V

Figure 22. Migratory path of Kiricephalus coarctatus nymph in cross-section, in kidney of Farancia abacura. Note eosinophil infiltration. H and E. 125 X.

Figure 23. Granulomata in kidney of Farancia abacura with eosinophil infiltration. 250 X.

Figure 24. Kiricephalus coarctatus in liver of Natrix sp. with mild eosinophil infiltration. Note regeneration of the damaged parenchyma, which may be quite metaplastic. H and E. 40 X.

Figure 25. Kiricephalus coarctatus in kidney of Natrix sp. with eosinophil infiltration. H and E. 40 X.

Figure 26. Adult Kiricephalus coarctatus gravid female in connective tissue between lung and digestive tract. No evident inflammatory response in this section. H and E. 40 X.

Figure 27. Adult Kiricephalus pattoni female in lung of Elaphe taeniurus. The lung is so fibrotic and filled with hemorrhage as to be scarcely recognizable. H and E. 40 X.

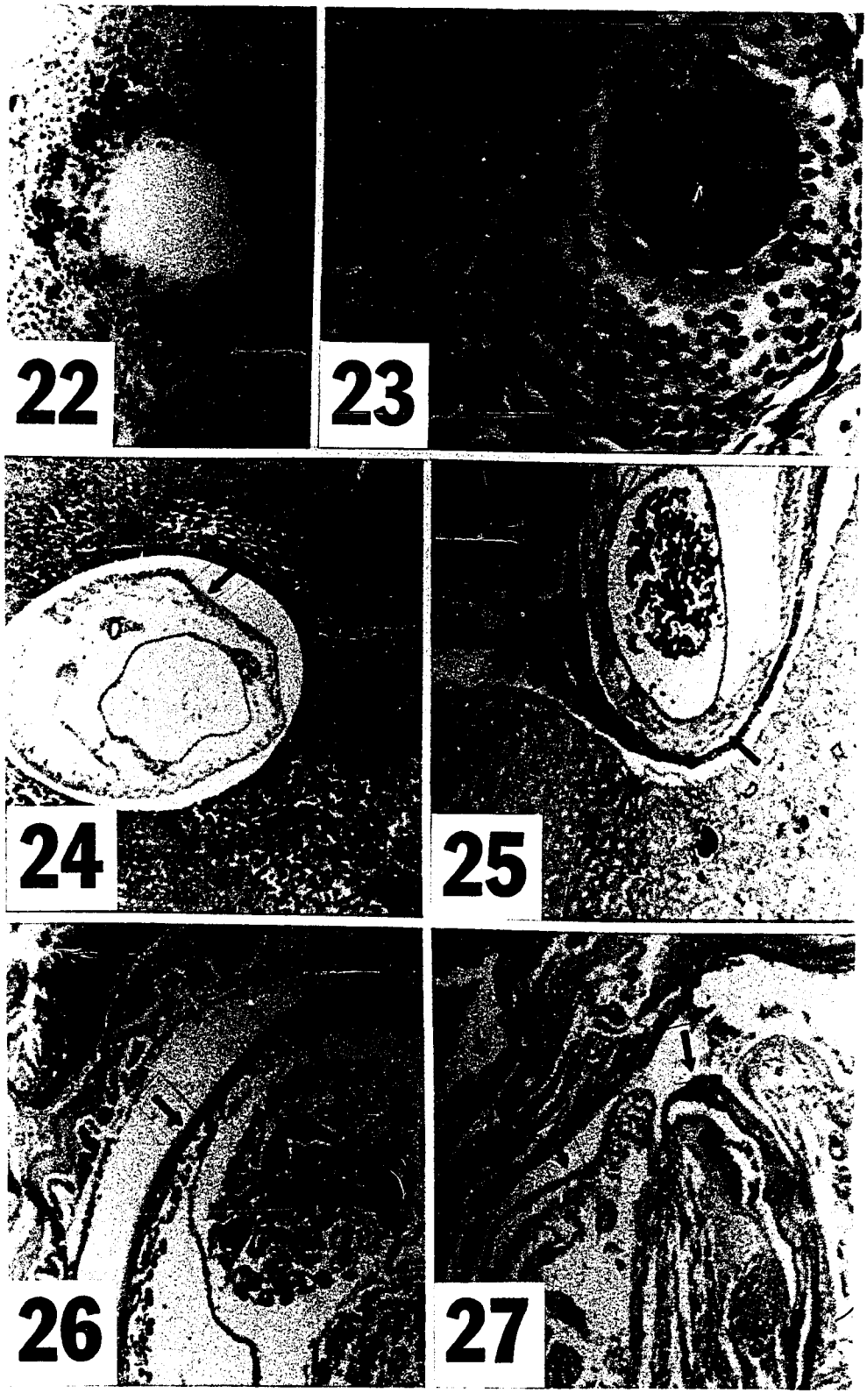
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PLATE VI

Figure 28. Degenerating adult and nymph of Sambonia lohrmanni (arrows) in the lung of Varanus niloticus, showing the marked necrosis and eosinophilic response found throughout the lungs of this lizard. H and E. 40 X.

Figure 29. Tubercles in the lung of Varanus niloticus, the result of degeneration of Sambonia lohrmanni. H and E. 40 X.

Figure 30. Granuloma in the intestinal submucosa of Varanus niloticus containing a larva of Sambonia lohrmanni (arrow). H and E. 40 X.

Figure 31. Same. 250 X. Showing the intense mononuclear infiltration and early formation of an atypical foreign body giant cell granuloma about an intact Sambonia lohrmanni larva which shows no signs of death or degeneration.

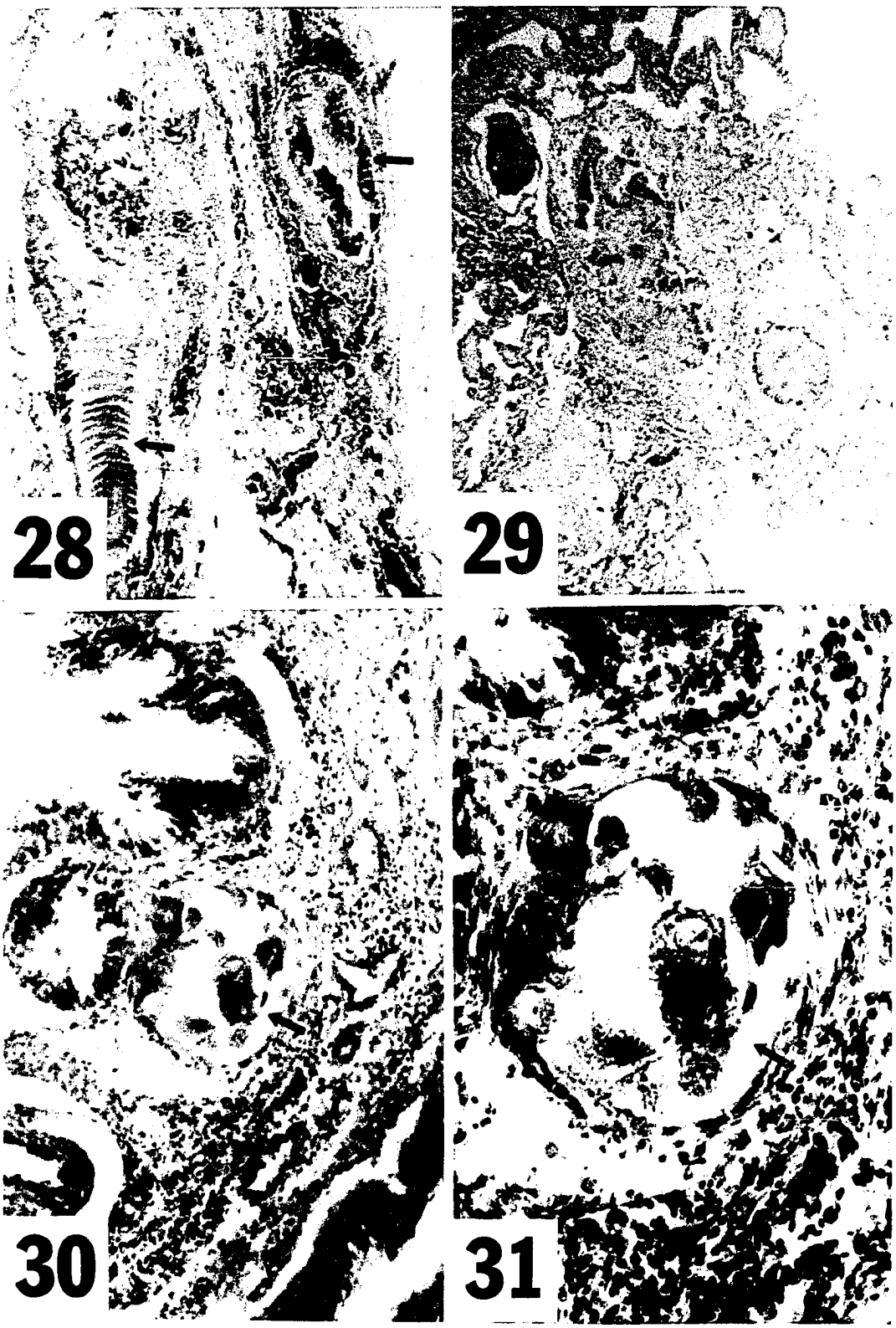
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PLATE VII

Figure 32. Intimal thickening of arteriole in liver of Varanus niloticus and congestion of venule. This lizard was massively infected with Sambonia lohrmanni by auto-infection. H and E. 400 X.

Figure 33. Nymphs and larvae of Sambonia lohrmanni (arrows) in the intestinal adventitia of Varanus niloticus. One is fibrosed, typical of the plaques in the adventitia of blood vessels. Gomori trichrome. 80 X.

Figure 34. Pigmentation and fatty degeneration of kidney, with pronounced proliferative changes in the glomeruli, in Varanus niloticus heavily infected with Sambonia lohrmanni. H and E. 400 X.

Figure 35. Lymphocytic infiltration about hook of adult Sambonia lohrmanni (arrows) in adventitia of lung and intestine of Varanus niloticus. The hook is cut in two sections. H and E. 125 X.

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PLATE VIII.

Figure 36. Lung of Osteolaemus tomistomi showing egg granulomata (arrows), hypersecretion of mucus and hemorrhage in Sebekia oxycephala infection. H and E. 40 X.

Figure 37. Feeding site of Sebekia oxycephala in lung of Osteolaemus tomistomi. A section of the pentastome is below (arrow). H and E. 250 X.

Figure 38. Eggs of Sebekia oxycephala in the lumen of the lung of Osteolaemus tomistomi, with inflammatory cells and debris. H and E. 250 X.

Figure 39. Granuloma in the connective tissue of lung and intestine of Lachesis muta which contains the necrotic remains of a Porocephalus sp. nymph. The refractory pores of "stigmata" of the cuticle are a helpful diagnostic character in identifying porocephalid nymphs. H and E. 250 X.

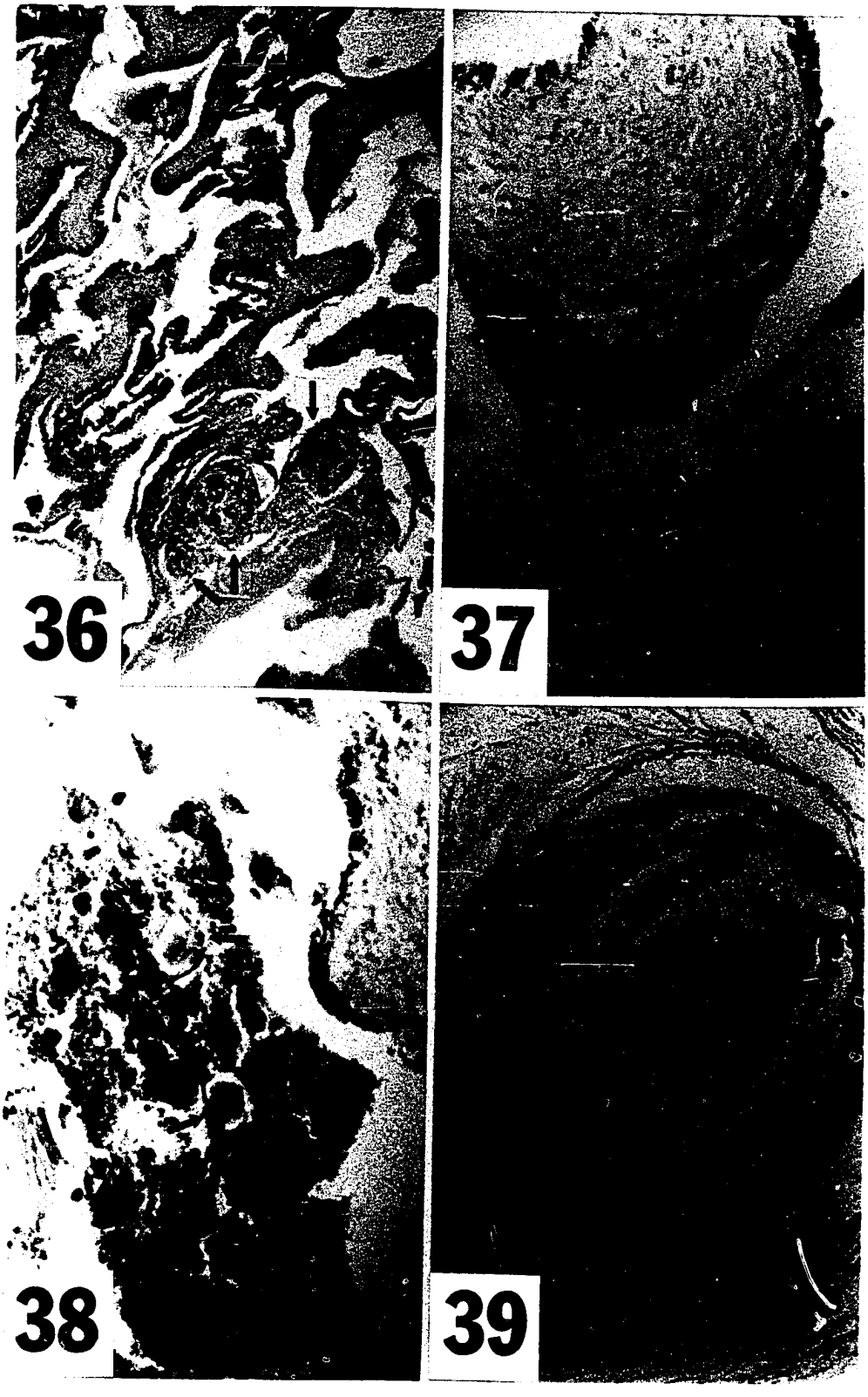
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PLATE IX.

Figure 40. A Kiricephalus pattoni larva is shown migrating through the submucosa of the intestine of Elaphe taeniurus, illustrating auto-infection in a captive reptile. H and E. 450 X.


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PENTASTOME PATHOLOGY IN CAPTIVE REPTILES

By Dennis Eugene Deakins

Major Professor: J. Teague Self

Abstract. Auto-infection is shown to be a major cause of pentastome pathology in reptiles, and to occur in three genera: Raillietiella, Sambonia and Kiricephalus. A variety of host responses are elicited by pentastomes including an acute inflammatory response characterized by eosinophil infiltration, chronic inflammatory response, and granulomatous response. Pentastomes cause numerous types of trauma, open the way to secondary invaders, and the larvae cause parasitic emboli. The larval migration of the intestinal wall is described and illustrated for the first time in reptiles, as is the utilization of the hepatic portal vein. It is demonstrated that the identification of pentastomes is possible long after death and degeneration of the parasites due to the persistence of the sclerotized portions of the cuticle (mouth, hooks and penetrations apparatus).