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SOME PARASITIC AND OTHER NATURAL DISEASES OF THE AFRICAN ELEPHANT IN THE KRUGER NATIONAL PARK

P. A. BASSON[1], R. M. McCULLY[2], V. DE VOS[3], E. YOUNG[3] and S. P. KRUGER[4]

ABSTRACT

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Detailed descriptions are given of the lesions encountered at autopsy on a random selection of 32 free-living African elephants [*Loxodonta africana* (Blumenbach, 1797)]. Lymphoid nodules with inclusion-bearing syncytia caused by a herpes virus were found in many lungs and similar lesions occurred in the pancreas. Suspected viral lesions somewhat resembling bovine granular vaginitis were encountered in the genitalia. *Dipetalonema gossi* Baylis, 1923, which was originally recorded from the abdominal cavity, was recovered from the portal veins of several animals. They proved to be responsible for severe intrahepatic vasculitis and eosinophilic hepatitis in 50% of the elephants. *Grammocephalus clathratus* (Baird, 1868) Railliet & Henry 1910 was constantly present in the bile ducts where fairly marked cholangitis was produced. Even mild localized pancreatitis was sometimes caused by these parasites. *Parabronema africanum* Baylis, 1921 was found in large numbers in gastric ulcers. A new mite, *Loxanoetus bassoni* Fain, 1970 was obtained from the ears. The livers of two elephants contained a few suspected schistosome ova. Siderotic and haemangiomatoid splenic lesions occurred in several adult animals. Many of the cows had multiple periuterine papillomata. Arteriosclerosis of the aorta was occasionally encountered. Skin lesions resembling porcine lesions of zinc deficiency were observed. Mycotic lesions were seen once in the lungs and lymph nodes. The aetiology of focal disseminated cystitis in 39% of the cows was not established.

INTRODUCTION

A cropping programme of African elephant [*Loxodonta africana* (Blumenbach, 1797)] in the Kruger National Park during 1968 and 1969 afforded an opportunity to study the pathology of some parasitic and other natural disease entities of this mammal. The major object of the campaign was to relieve a steadily worsening ecological situation caused by the overpopulation of elephants. The meat and by-products were processed in as short a time as possible to prevent spoilage and the above studies were subordinate and consequently limited by these procedures. Hence, a complete and thorough survey of parasites and diseases was impossible and the following report deals only with the most significant observations obtained. The incidence of herpes nodules in the lungs of elephant has already been reported (McCully, Basson, Pienaar, Erasmus, Young & Pieterse, 1969; Basson, McCully, Kruger, van Niekerk, Young, de Vos, Keep & Ebedes, 1971; McCully, Basson, Pienaar, Erasmus & Young, 1971; Erasmus, McCully, Pienaar, Young, Pieterse & Els, 1971).

MATERIALS AND METHODS

A random selection of 32 elephants was either shot or immobilized with scoline and then shot. These animals were used for a survey of lesions and parasites. Some additional elephants which were cropped subsequently are also included in this study but do not form part of the survey. A macroscopic examination was done on most of the organs that were available. Blood smears were prepared and stained with Giemsa and specimens of various tissnes and organs were collected in 10% buffered glucose formalin. Some parasites for identification were preserved in 10% formalin, but a complete study of the intestinal helminths and bots was not made. The fixed tissue specimens were processed routinely by embedding in paraffin wax, sectioning with a sliding microtome and staining with haematoxylin and eosin (HE). Special staining techniques employed were Giemsa, Gram, Gomori's methenamine silver (GMS) (Anon. 1960); periodic acid-Schiff (PAS), Schmorl's method for lipofuscin (Schmorl's), Berliner Blue (BB) for ferric iron, Tirman & Schmeltzer's method for ferrous iron (T & S), Alizarin red (AR) for calcium (Pearse, 1961) and Ziehl-Neelsen (ZN) (Cruickshank, 1962).

RESULTS

The most important findings are quantitatively presented in Table 1.

Lung lesions

Herpes nodules

Comprehensive reports on this infection have been published and a note is included here merely for the sake of completeness. The main features of the disease were the development of lymphoid nodules in the lungs [Fig. 1 (1)], cuboidal metaplasia of the alveolar lining cells and the formation of giant and syncytial cells with intranuclear inclusions [Fig. 1 (2)]. The incidence was approximately 80%.

Lymphoid nodules with similar syncytial cells and inclusions were subsequently found in the pancreas of several animals [Fig. 5 (25 and 26)].

Mycotic pneumonia

One case was encountered in which several purulent granulomas resembling actinobacillosis were found [Fig. 1 (3)]. These lesions had an irregular central core of wavy, septate mycelia. They were slightly basophilic with HE, somewhat more basophilic with Giemsa,

(1)Section of Pathology, Veterinary Research Institute, Onderstepoort
(2)Lt. Col. U.S.A.F., V.C., Chief, Geographic Zoonoses Branch, Geographic Pathology Division, Armed Forces Institute of Pathology, Washington, D.C.
(3)State Veterinarian, Skukuza
(4)Formerly from the Section of Helminthology, Veterinary Research Institute, Onderstepoort

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argyrophilic with GMS [Fig. 1 (4)], negative or mildly positive with PAS, but completely negative with ZN and Gram stain. The fungal core was surrounded by eosinophilic clubs, a layer of neutrophils and epithelioid cells followed peripherally by round cells and fibrous elements. The regional lymph nodes were not affected.

These mycotic lesions were either secondary to or coincidental with a herpes infection and, as a diagnosis was only made microscopically on fixed specimens, the fungus could not be cultured and identified specifically.

Liver lesions

Hepatic dipetalonemiasis

Fifty per cent of the elephants had hepatic lesions which were apparently caused by filarids. Thirty per cent of these were mature animals and both sexes were equally affected. Macroscopically these lesions consisted mainly of vascular changes, which were most prominent on the cut surfaces of multiple incisions made along the edges of the liver (edge lesions) and of various numbers of pin-point yellowish-grey pustules. The vascular and pustular lesions were seen macroscopically in 28% and 37% of the elephants, respectively. The edge lesions comprised thickened, inflamed vessels, either early or organized thrombi [Fig. 1 (5 and 6)] and lymphoid nodules. Both of the latter often contained calcified material. The affected vessels which were dissected proved to be portal veins. The vascular lesions were more prominent in the terminal branches of the vessel but also occurred elsewhere in its course beginning at the point of entry into the liver. In some of the chronic cases either a polypoid or a very peculiar corrugated or criss-crossed proliferative endophelebitis was encountered [Fig. 2 (7 and 9)]. About 50% of the parasitized livers contained a few dead intravascular filarids, which were mineralized and semi-embedded in partially organized thrombi [Fig. 1 (6)]. In the largest portion of the portal vein in two cases, viable filarids of both sexes were found which were subsequently identified as *Dipetalonema gossi* Baylis, 1923. They were round, fairly slender and approximately 15 cm in length [Fig. 2 (8)]. Portions of the livers of a few elephants had a mottled greyish-brown pattern, sometimes with a sunken surface and increased consistency.

The earliest microscopic lesions consisted of fibrinous thrombosis and an eosinophilic inflammation of many portal [Fig. 3 (17)] and some central and hepatic veins. All the various layers of the vessel walls, especially the intima, were affected. These changes ultimately resulted in villous and polypoid proliferations of the intima and thickening of the vessel walls which were due mainly to smooth muscle hypertrophy and hyperplasia, fibrosis and cellular infiltration [Fig. 2 (10 and 11)]. The proportion between these elements varied considerably and depended to a large extent on the severity of the infestation and the duration of the disease. There was sometimes recent thrombosis superimposed on chronic lesions [Fig. 3 (18)]. Organization of thrombi and localized areas of partial or complete, well-organized lymphoid proliferation [Fig. 2 (12)] in the walls of the vessels, especially in the intima, were other prominent features. Some of these lesions contained dead and partially calcified nematodes [Fig. 3 (13, 14 and 15)].

There was also inflammation of the portal triads and surrounding parenchyma. It was frequently primarily eosinophilic but a mixed cell reaction was sometimes encountered. Many eosinophils were also found below Glisson's capsule. These evidently represented the pustular lesions seen grossly. Suspected migratory tracts filled with amorphous, necrotic debris surrounded by intense eosinophilic infiltrates and eosinophilic granulomas [Fig. 3 (16)] were encountered in some of the animals.

The periportal lymph nodes were frequently hyperplastic and contained many plasmocytes. Eosinophilic and granulomatous lymphadenitis were also found.

Grammocephalosis

This generic name is used to denote infestation with the bile duct hookworm *Grammocephalis clathratus* (Baird, 1868) Railliet & Henry, 1910. All the elephants were moderately to fairly severely infested with this parasite. The medium, large and main bile ducts were invariably thickened [Fig 4 (19 and 20)] and contained small disseminated haemorrhages, erosions, ulcers and necrotic foci. The terminal extrahepatic portion, however, was less severely affected. The microscopic study of the large bile ducts revealed epithelial hyperplasia and inflammatory infiltrates of eosinophils and round cells were found both subepithelially [Fig. 4 (22)] and in the deeper layers of the ducts. There was also ulceration [Fig. 4 (21)] and micro-abscessation of the thickened wall. Small eosinophilic or purulent microgranulomas with well-developed lymph follicles [Fig. 4 (23)], some containing suspected cuticles, calcified debris and/or bacteria, were formed. Secondary bacterial infections were frequently encountered, both cocci and bacilli being observed. Nodular lymphoid proliferation was often present in the bile duct wall and in the advanced cases there was chronic pericholangitis with extensive fibrosis [Fig. 4 (24)].

The small bile ducts in the portal triads were never directly involved, but mild inflammation and very mild bile duct proliferation in the immediate vicinity of the affected large ducts were frequently noticed in some of the livers. Eosinophils, lymphocytes and plasmocytes in various proportions were present in these adjacent areas.

In the elephant, as in some other mammals, the main pancreatic duct, which is very actively secretory, fuses with the main bile duct some distance (approx. 5 to 8 cm) before opening into the duodenum (Hill, 1953). It was evident that the initial unfused portion of the pancreatic duct was sometimes mildly affected by *G. clathratus*. Even the interlobular ducts, which are characterized by a marked mucous secretion, were affected in one case. A mixed cell reaction and one microabscess were found in the latter [Fig. 5 (27)]. Localized areas of the adjacent parenchyma revealed subacute pancreatitis [Fig. 5 (28)]. Lymphoid nodules were often encountered. Unfortunately, circumstances during the survey did not permit regular, proper examination of the pancreas. Ten additional elephants were therefore examined subsequently for pancreatic lesions, of which five had very mild or mild subacute reactions in the ducts, presumably due to grammocephalosis. However, no parasites were found. In the pancreas of six there were lymphoid nodules [Fig. 5 (25)], some of which were well organized in follicles. A third of these cases revealed giant cell syncytia [Fig. 5 (25 and 26)] with intranuclear inclusions which resembled the herpes virus inclusions in the lungs very closely (*vide supra*).

Suspected schistosomiasis

A few eosinophilic microgranulomas around ova which resembled those of *Schistosoma* spp. were observed in the livers of two elephants. No schistosomes, however, were found either on macroscopic or microscopic examination.

TABLE 1 *Incidence of pathological findings*

	Incidence (%)	No Sex Size	1 ♂Y	2 ♂A	3 ♀Y	4 ♀A	5 ♀Y	6 ♀Y	7 ♀A	8 ♀Y	9 ♀Y	10 ♀A	11 ♂Y	12 ♀A	13 ♂Y	14 ♀Y	15 ♂C	16 ♀Y	17 ♀Y	18 ♀Y	19 ♂Y	20 ♂Y	21 ♂Y	22 ♂Y	23 ♀A	24 ♂A	25 ♀Y	26 ♂Y	27 ♀A	28 ♀A	29 ♂Y	30 ♀A	31 ♂A	32 ♂Y
Herpes infection	80		+	+			-	+	+	–	+	+	+		+	+	+	+	–	+	+		·	+		+	–	+			+	+	+
Mycotic pneumonia	3									+																							
Hepatic dipetalonemiasis	50		+	+						:	+	+	+			+				+		–	+	+	+					+		+	
Grammocephalosis	100		+	+	+	+	+	+	+	:	+	+	–	+	+	+	+	+	+	+	--	,	+	+	+	–	+	+	+	+	--	+	+	+
Pancreatitis	3*												·																					
Parabronemiasis	47*		O	O	+	O	O	O	O	O	+	-	+	O	·	O	+	-	+	+	+	O			+			+	+	+			+
Splenic siderosis	16								+			+		+												+			+					
Lymphadenitis	22										+		+			+		-						+			·		+					
Cystitis	22				+	+		+	+											+										·		+		
Periuterine papillomata	33				–				-			+		+		·														–				
Granular vestibulitis	50				+		+		+		+	+				+		+	+	·														
Myocardial degeneration	6					+				+																								
Arteriosclerosis	3																												+					

C — Calf Y — Young animal A - Adult * — Inadequate survey O — Not examined specifically

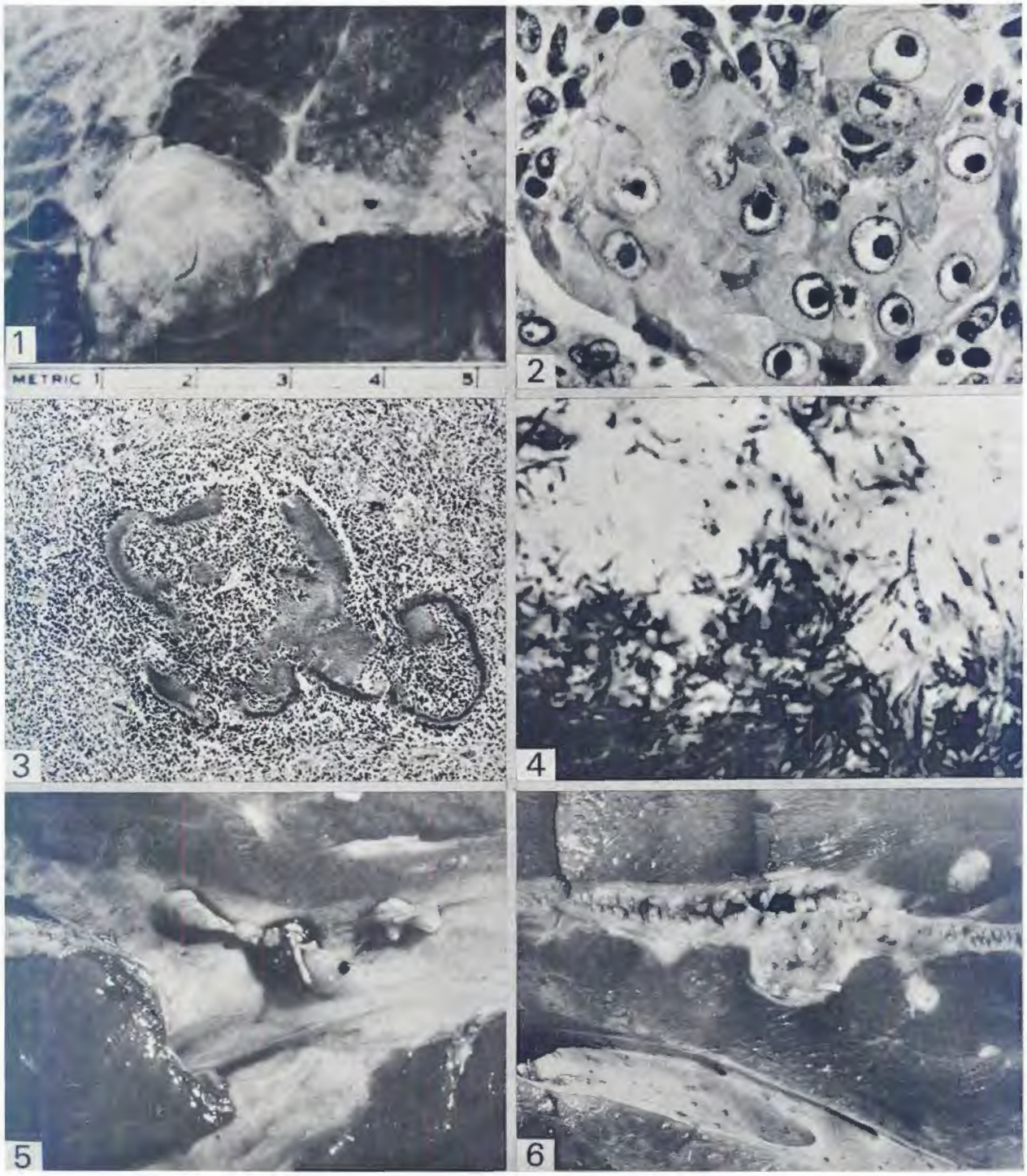

FIG. 1 1. Lung: A lymphoid nodule caused by a herpes virus. 2. Lung: A syncytial cell within a herpes nodule. Several intranuclear inclusions are present. HE × 500 3. Lung: Purulent, mycotic granuloma. The irregular dark grey material represents both the fungal core and eosinophilic clubs. HE × 75 4. Lung: Higher magnification of the fungi in (3) GMS × 1 200 5. Liver: Branch of the portal vein revealing semi-organized thrombi. Pieces of entrapped, mineralized filarids can be seen 6. Liver: An opened portal vein with some chronic proliferative lesions and an organized thrombus with mineralized filarids. An unaffected hepatic vein is seen below.

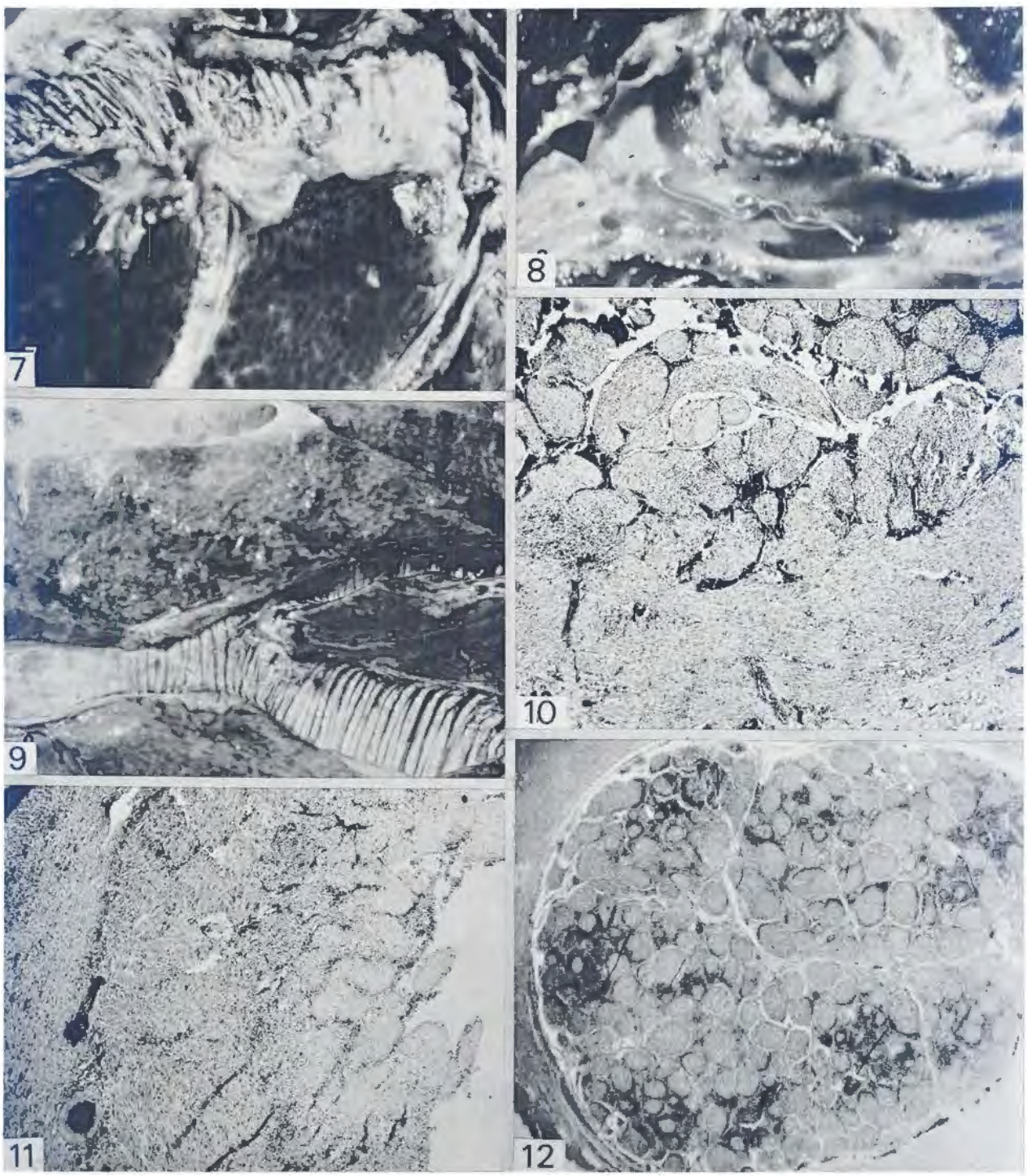

FIG. 2 7. Liver: Filariasis: Portal vein with a peculiar corrugated, criss-crossed proliferative endophlebitis and organization of thrombi 8. Liver: A filarid within an opened portal vein. 9. More marked corrugated, rugosal endophlebitis of a portal vein. A normal hepatic vein is seen dorsally. 10. Polypoid endophlebitis with prominent smooth muscle hyperplasia and fibroplasia. HE × 30 11. An earlier polypoid lesion in a portal vein. Very mild superficial fibrinoid thrombosis is present also. HE × 30 12. An intravascular lymphoid nodule with numerous secondary nodules. HE × 12

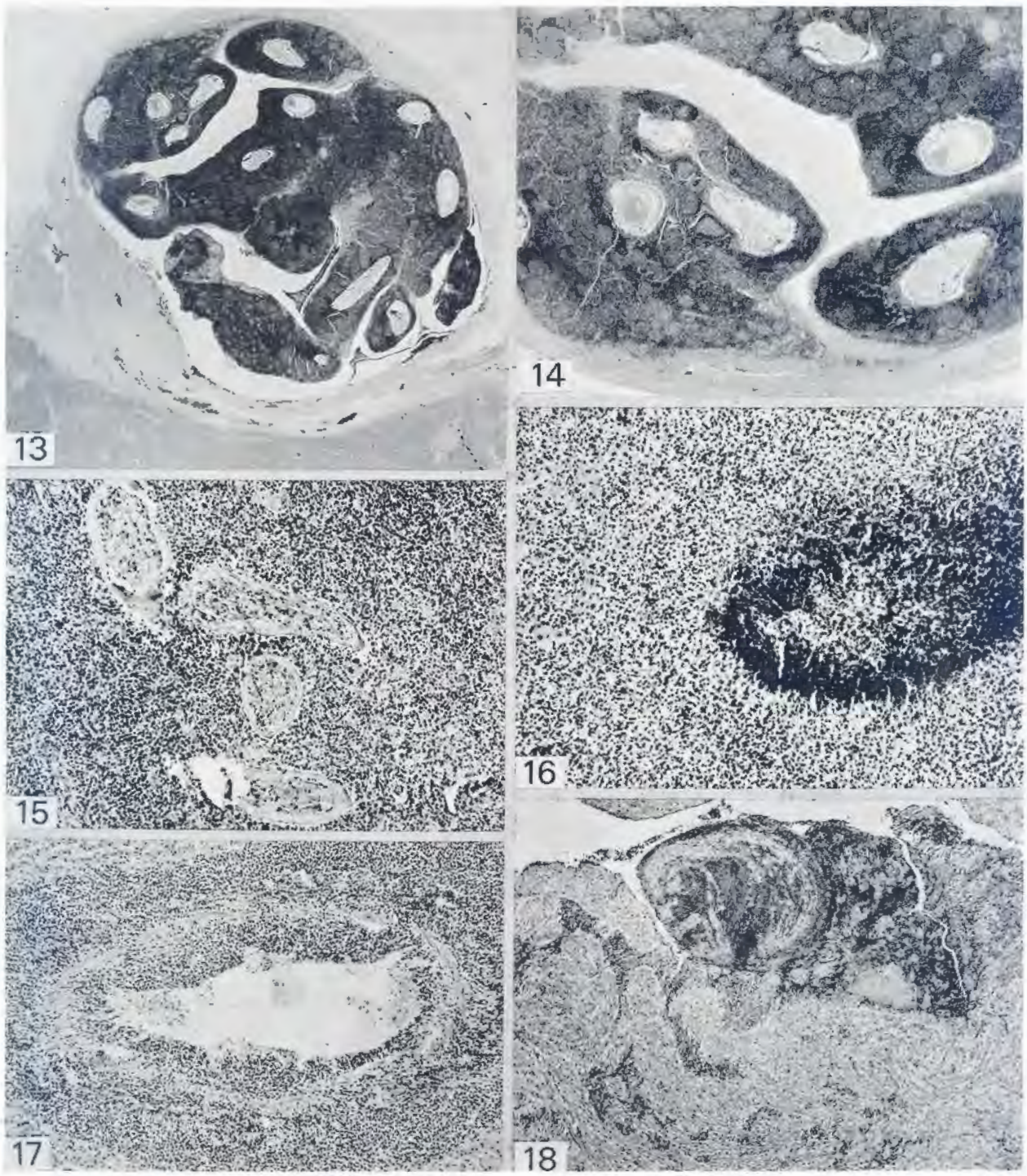

FIG. 3 13. Liver: Filariasis: A portal vein with well-organized lymphoid proliferation in the intima surrounding dead portions of filarids. HE × 18 14. Higher magnification of (13). The follicles of the lymphoid nodule are most distinct. HE × 30 15. Dead filarids in an unorganized lymphoid nodule. HE × 75 16. Eosinophilic granulomatous lesion surrounded by lymphocytes. HE × 75 17. Eosinophilic vasculitis and perivasculitis of a portal vein in a more acute case of portal filariasis. HE × 75 18. Acute thrombophlebitis superimposed on a chronic polypoid lesion in the portal vein. HE × 75

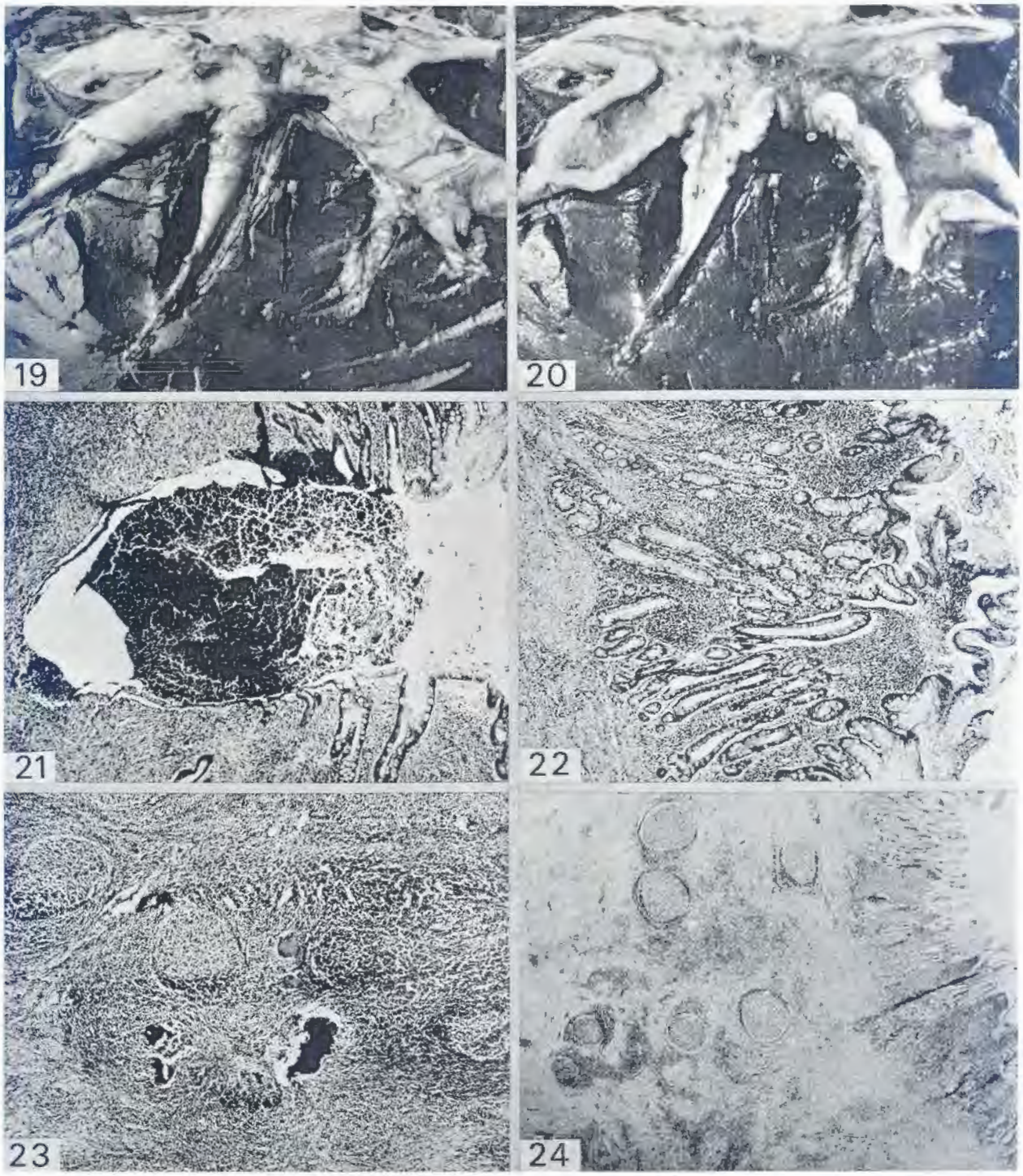

FIG. 4 19. Liver: Distended, enlarged bile ducts due to grammocephalosis 20. Liver: Opened bile ducts of (19) to show the presence of *G. clathratus* 21. Bile duct: Necrotic exudate in an area of ulcerative cholangitis. HE × 30 22. An area of cholangitis revealing sub-epithelial infiltration of leukocytes. HE × 30 23. Wall of the bile duct with granulomas and well-developed lymph follicles. Giant cells can also be seen. HE × 30 24. Portion of a bile duct with leukocytic infiltration and several lymph follicles. HE × 12

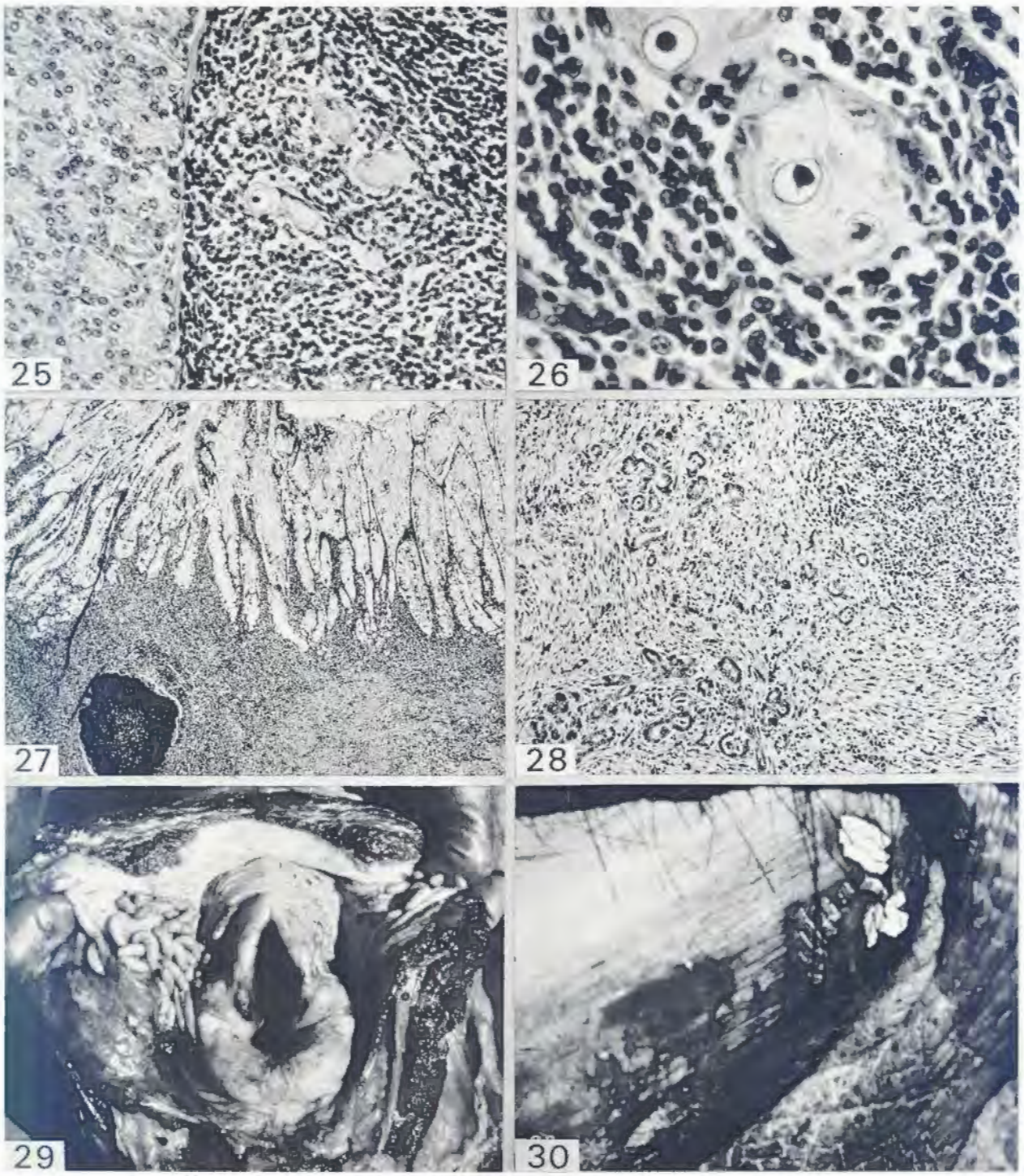

FIG. 5 25. Pancreas: Portion of a lymphoid nodule with a few syncytial cells. Notice one intranuclear inclusion. HE × 200 26. Pancreas: Syncytial cells with intranuclear inclusions found within lymphoid nodules. HE × 500 27. A large pancreatic duct with a subacute inflammation and an eosinophilic abscess caused by bile duct hookworm. HE × 30 28. A localized area of subacute pancreatitis. HE × 75 29. Pharyngeal area with bots 30. Rows of bot ova at the base of a tusk

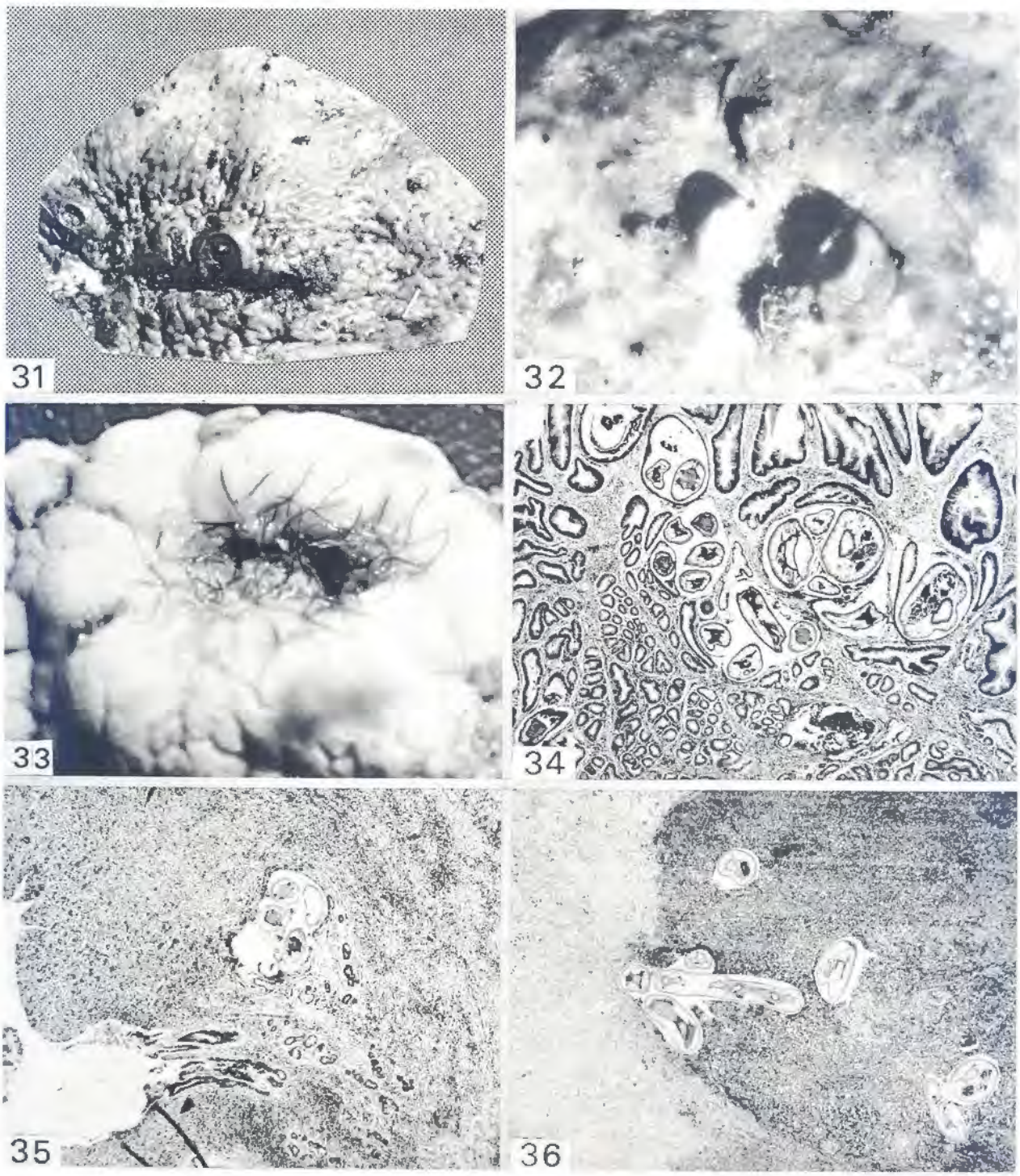

FIG. 6 31. Stomach: Parabronemiasis: Ulcers of various sizes caused by *Parabronema africanum* 32. An opened fistulous lesion with many of the helminths 33. A small stomach ulcer in which the parasites have been stained with iodine for greater contrast 34. Helminths in cross section in the gastric crypts and mucosa. HE × 30 35. Parasites within the stomach wall which shows a prominent subacute inflammation. HE × 30 36. Worms within and below necrotic debris in the ulcers. HE × 30

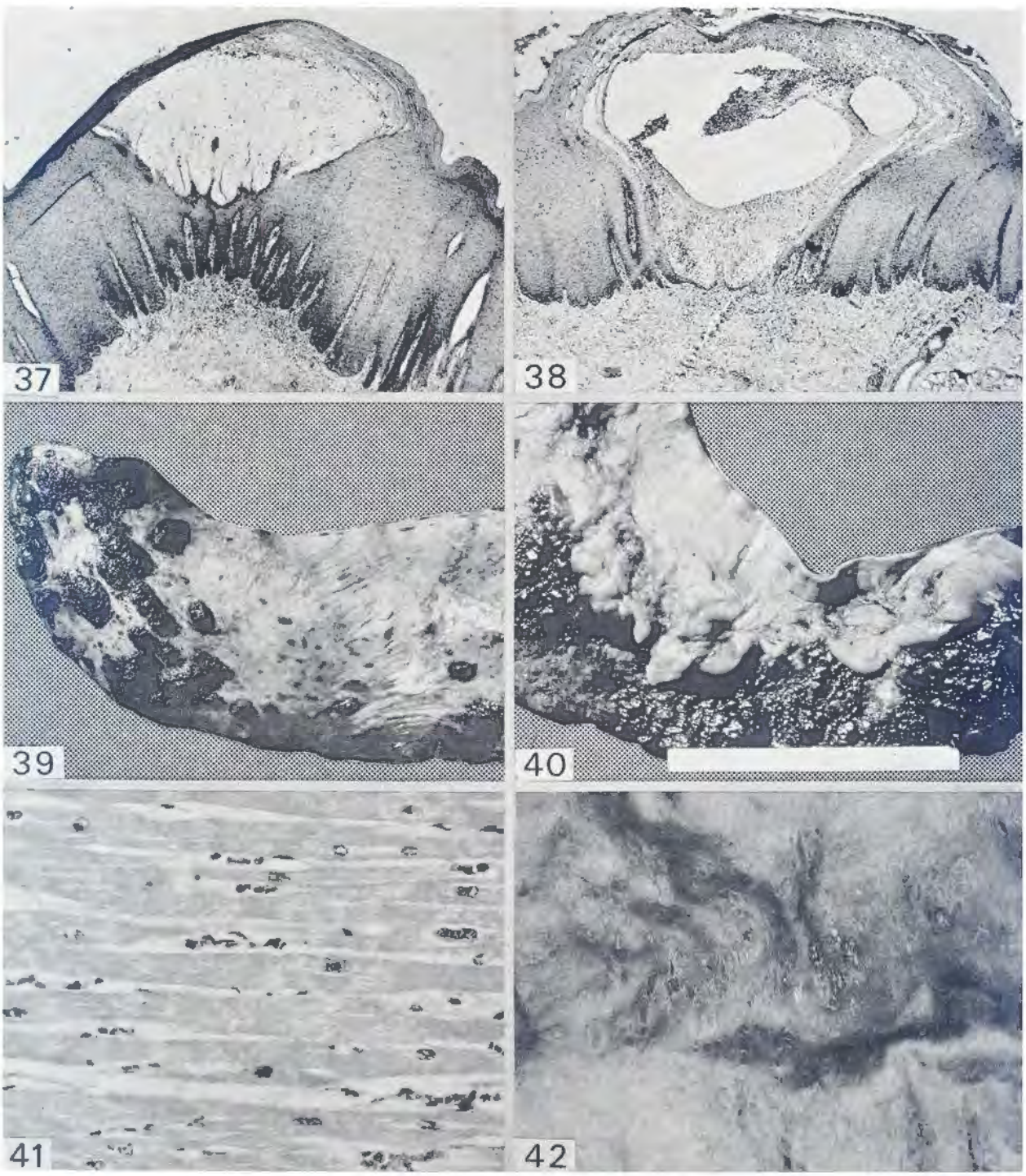

FIG. 7 37. A micro-mucocoele in the oesophagus. HE × 30 38. Another micro-mucocoele with some neutrophil infiltration and deeper extension into the mucosa. HE × 30 39. Spleen with several siderotic, haemangiomatoid lesions on the parietal surface 40. Spleen with similar lesions on the visceral surface 41. Myocardial degeneration fonnd in some of the immobilized elephants. HE × 200 42. Arteriosclerosis of the aorta

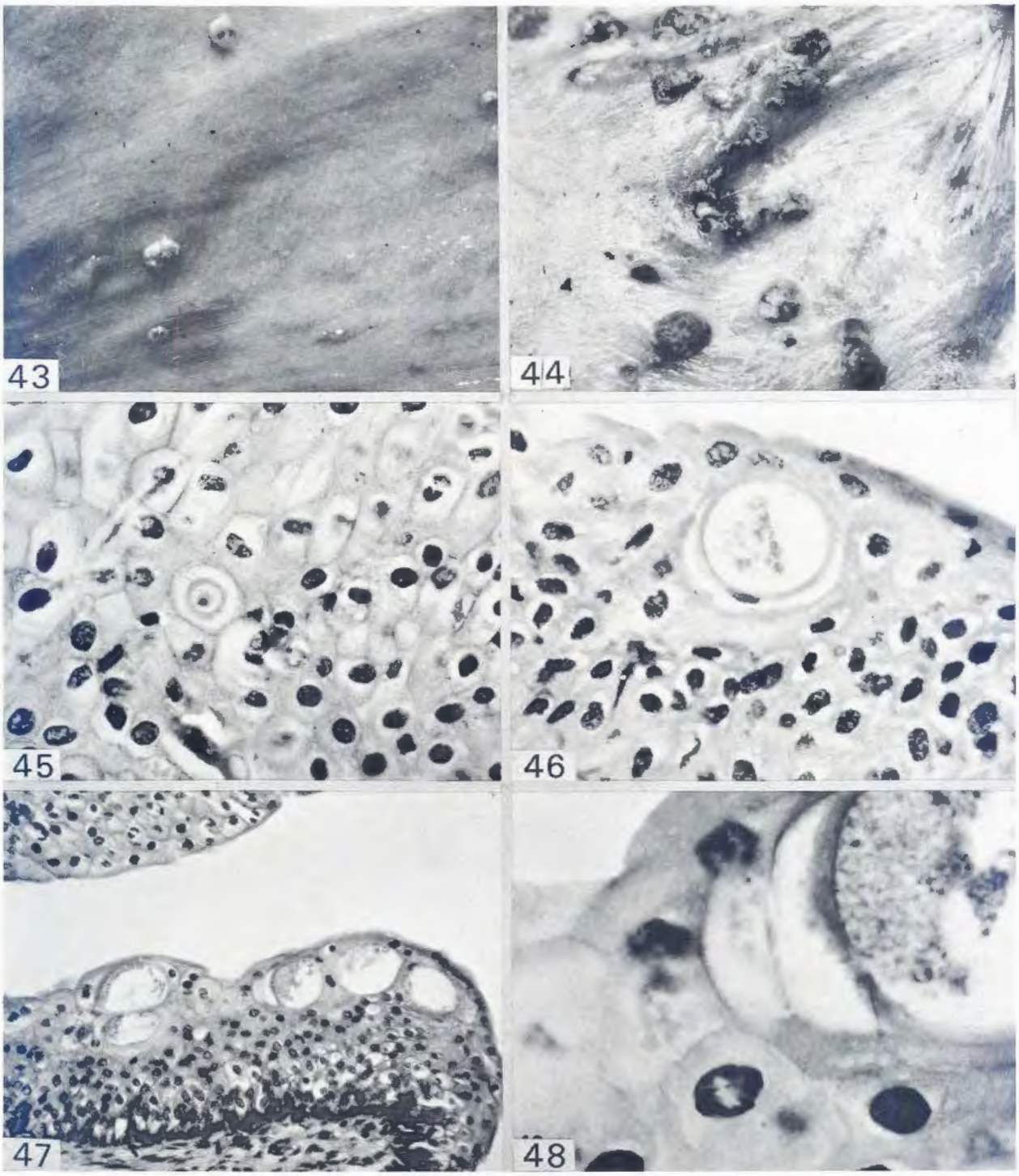

FIG. 8 43. Urinary bladder: Raised inflammatory and necrotic foci in the mucosa 44. Another case with large and more numerous foci of cystitis 45. An epithelial cell containing a circular eosinophilic rim with some more intensely eosinophilic substance in its centre. HE × 500 46. Another epithelial cell which has the shape of a signet ring. The nucleus is compressed and several eosinophilic granules are present. HE × 500 47. Ballooning changes in several epithelial cells which contain some mucoid substance and eosinophilic granules. HE × 200 48. Higher magnification of the central portion of the epithelium in (47) showing the mucoid substance and eosinophilic granules. HE × 1200

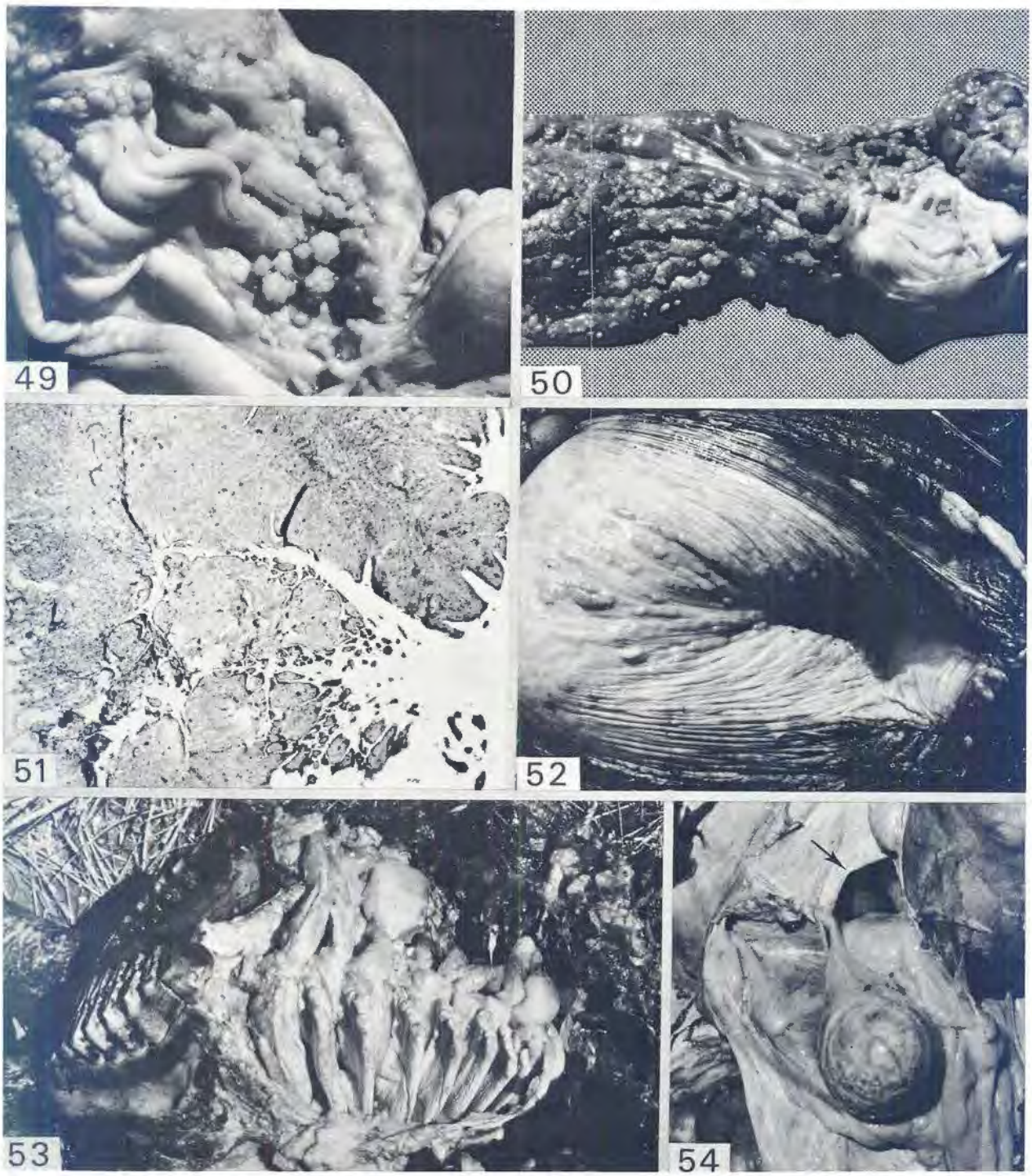

FIG. 9 49. Serosal surface of a uterine horn with many periuterine fibrous papillomata 50. Another case with more numerous fibrous papillomata 51. Section through some of the papillomata. HE × 30 52. A case of granular vulvitis 53. Jaw showing molar teeth in wear and exposed unerupted molars which are aligned like cartridges in a magazine 54. Exposure of intra-abdominally situated testis drawn through an incision (arrow) in the flank of a laterally recumbent elephant, after removal of the ribs. The back is at the bottom and the hind-quarters to the left

Digestive tract

Myiasis (cobboldiasis)

Bots were frequently found in the pharynx [Fig. 5 (29)], oesophagus and stomach but no survey of their incidence was made. Zumpt & Wetzel (1970) reported *Cobboldia loxodontis* Brauer, 1897 and *C. chrysidiformis* Rodhain & Bequaert, 1915 from the stomachs of some elephants from the same cropping programme. The only tissues collected for histopathological studies which might have revealed bot lesions were oesophageal specimens taken from small white spots and vesicles which were encountered along with the small erosive lesions that were obviously caused by these parasites. The vesicles resembled micro-mucocoeles histologically [Fig. 7 (37)]. Some were suppurating and contained varying numbers of neutrophils and cocci. The early stages of a few vesicles revealed hydropic or ballooning changes in the epithelial cells. Erosions were present and mild mixed cell reactions occurred in the propria. The ducts of some of the submucosal glands were dilated with accumulated mucous secretion. It is believed that some of the vesicles [Fig. 7 (38)] could have resulted from such occluded ducts. However, others probably developed from bot-induced erosions and ballooning changes in the epithelial cells.

As indicated by Zumpt (1965) the ova of the bot flies were recovered from the base of the tusks [Fig. 5 (30)] and on some of the teeth.

Gastric parabronemiasis

The stomachs of only 22 elephants could be examined carefully and 15 of these (68%) contained small pustules and one or more ulcers [Fig. 6 (31)] which were mainly confined to the anterior portion of the greater curvature of the stomach. The ulcers varied in size from approximately 3 to 50 mm in diameter. Their walls were elevated and some were connected to adjacent ulcers by fistulas [Fig. 6 (32)]. Their surfaces contained a yellowish exudate and numerous very slender nematodes [Fig. 6 (33)] identified as *Parabronema africanum* Baylis, 1921. These parasites were also recovered in smaller numbers from the surrounding mucosal surface.

Microscopically, the viviparous nematodes seemed to invade the crypts and eventually burrow into the mucosa and submucosa [Fig. 6 (23)]. Simple squamous metaplasia of the epithelium of the parasitized crypts, pustules and necrotic, suppurating ulcers were present. The helminths, which were all alive, were found within and below the necrotic debris [Fig. 6 (36)]. The cell reaction consisted mainly of eosinophils and neutrophils but many lymphocytes and plasmocytes were also present, especially in the deeper layers of the wall and around the blood vessels. Proliferation of capillaries and fibroplasia were other prominent features, a significant portion of the lesion being essentially granulation tissue. Many coccoid bacteria were found in the superficial detritus but fungi were never encountered.

Lymphatic organs

Splenic siderosis

Five elephants (16%), all adult or aged animals of both sexes, had elevated fairly firm dark red lesions in the capsule of the spleen. These lesions were roughly circular or ovoid with an irregular outline and uneven outer surface. They varied in size from approximately 1 to 40 mm, were focally disseminated and occurred on both parietal and visceral surfaces [Fig. 7 (39 and 40)]. They were frequently coalescent, forming large confluent patches with undulating surfaces. The surrounding capsule was irregularly thickened. On cut surface the lesions neither collapsed nor yielded much free blood. Microscopically they appeared to be partially in, but mostly bulging above, the surface of the normal capsule and were covered by flat to cuboidal epithelium which was recognizable as visceral peritoneum. They consisted of accumulations of blood in a stromal framework of connective tissue, with scattered capillaries and small vessels and amorphous pink material containing mineral elements. These were interpreted as areas of organization, but the primary lesions were probably being haemangiomatoid lesions, which might even be regarded as hamartomas. No evidence was found of invasion or metastasis to the pulp of the spleen, lymph nodes or other organs, nor was there any seeding of the parietal peritoneum. In these lesions greyish-blue (BB-positive and AR-positive) and bluish-grey (BB-positive) pigments were present in some collagen and elastic fibres of the blood vessels and surrounding connective tissue. Any, and sometimes more than one, layer of the vessel wall could be affected. The elastica interna of some of these vessels was frequently pigmented and fragmented. The fibres in the trabeculae were less affected and were impregnated with a different pigment, which stained greyish-brown to yellowish-brown with HE and positive with the Schmorl's technique for lipofuscins. This pigment, which proved negative with T & S for ferrous iron, was very abundant in the red pulp and occurred both as intracellular globules in numerous macrophages and as irregular extracellular patches. The latter form was probably deposited around reticulum fibres. A small number of macrophages with similar pigment was present in the capsule. Much brown BB positive pigment was found in the red pulp.

Lymphadenitis

Inflammatory lesions were encountered in some of the lymph nodes in seven elephants (22%). Two of the nodes showing localized foci of eosinophilic lymphadenitis were parietal but three were unidentified. A microgranuloma due to a pigmented fungus was found in one of the parietal nodes. One periportal lymph node had disseminated purulent foci in association with giant cells and macrophages which contained coccoid organisms thought to be due to secondary bacterial infection following hookworm infestation. Another animal had identical purulent lesions in a mesenteric lymph node.

Urogenital system

Cystitis

Seven of the 18 females examined (39%), of which 57% were mature cows, had small disseminated round to oval, dull, yellowish-grey or reddish-grey elevated or some slightly sunken foci in the urinary bladder [Fig. 8 (43 and 44)]. Their size varied from approximately 1 to 5 mm in diameter.

Microscopically the earliest lesions were found to contain many eosinophils and mild haemorrhages. These lesions developed into protruding, suppurative ulcers in which both eosinophils and neutrophils were recognized. A few gram negative bacilli were noticeable in the necrotic debris of one case. No helminths, parasitic ova or fungi could be demonstrated in the lesions.

Multiple or single, circular, eosinophilic, intracytoplasmic (IC) globules or granules were noticed in the epithelial cells of the urinary bladder of four females, of different ages, three of which had foci of disseminated cystitis. Some of these globules had an internal structure consisting of one or more tiny vesicles. In some animals a few of the more superficial epithelial cells exhibited

ballooning changes which resulted in very large intracellular vacuoles containing some amorphous, basophilic and mucoid substance as well as a few eosinophilic granules [Fig. 8 (45, 47 and 48)]. Only one vacuole was present per cell and it was invariably circular or ovoid, regular in outline and demarcated by a narrow rim of eosinophilic material that stained more intensely than the surrounding cytoplasm. Some of these cystic vacuoles were so large that signet cells with compressed nuclei were formed [Fig. 8 (46)]. A few polymorphonuclears were seen in some of these vacuoles.

The ureters of four elephants were specifically examined for similar structures, of which three were positive for both IC globules and intracellular vacuoles. The renal pelvis in two elephants that were studied contained IC globules only.

Periuterine and paraovarian fibrous papillomata

Six cows (33% of all the females), of which four were adults, had multiple raised villous and papillomatous nodules on the uterus and near the ovaries [Fig. 9 (49 and 50)]. These nodular lesions ranged from 1 to 20 mm in diameter, were very firm and appeared on cut surface to be confined to the subserosa. They proved microscopically to be fibrous papillomata [Fig. 9 (51)] covered by a simple squamous mesothelium which became cuboidal or columnar in some protected localized areas. The connective tissue consisted mainly of very dense mature collagenous fibres which were focally hyalinized. The lesions were not invasive and the myometrium and endometrium were not involved in any of the cases examined.

Granular vulvitis and vestibulitis

Lesions simulating those of granular vaginitis of cattle were noticed in the vulva and vestibulum of nine out of 18 female elephants (50%), two of which were adult cows. The lesions were round or oval, greyish papules approximately 1 to 15 mm in diameter [Fig. 9 (52)]. Exept in one case, where small suppurative ulcers were present, the mucosa was intact. Their microscopic appearance also resembled that of granular vaginitis. Well-organized, hyperplastic lymph nodules with germinal centres were located in the propria immediately below the epithelium. Some of the germinal centres had macrophages with necrotic debris giving them a typical "starry sky" appearance. A few suspected intranuclear inclusions were found in some reticulo-endothelial cells of one animal. Except for the migration of a few lymphocytes, the epithelium was usually intact. The ulcerative lesions seen in one cow revealed a purulent reaction and the presence of coccoid organisms in the superficial necrotic layers. Some of the surrounding vessels were infiltrated peripherally by round cells.

Erosive and ulcerative buccal lesions have recently also been noticed in a few elephant calves.

Cardiovascular system

Degeneration of the myocardium was noticed microscopically in two elephants in which small foci of rarefaction and vesiculation were fairly diffusely disseminated throughout the myocardium [Fig. 7 (41)]. Many myocardial nuclei, especially of the subendocardial fibres, were usually very large with conspicuously folded karyolemmas. This evidently resulted in engulfment of some of the sarcoplasm and the formation of eosinophilic "inclusions". Brown atrophy characterized by the prominence of lipofuscin around the nuclei was commonly seen.

Arteriosclerosis of the thoracic aorta was present, in one adult animal [Fig. 7 (42)]. Mineralization of the tunica media was noticed microscopically. Subsequent observations on 16 other animals which were not included in the original survey, revealed two more cases, thus increasing the incidence to 6%. One of these animals, a fairly aged male, had whitish, raised, irregular arteriosclerotic plaques measuring 2 to 30 mm in diameter in the aorta approximately 12 to 30 cm from the heart. Beyond this zone irregular depressed areas were found for another 30 cm along the aorta. The orifices of some of the efferent vessels were also affected. Microscopically the intima and adjacent media were very fibrous, with few cells, and stained mildly positive with ORO. The elastica interna was only very mildly eosinophilic and disintegrating in parts. The lower portion of the aorta contained mineralized areas in the tunica media. The third case revealed thickened areas in the aorta as a result mainly of the production of a myxomatous substance in the intima.

No specific changes or parasites were seen in the blood smears.

Skin

Acariasis (loxanoetosis)

Extremely small ear mites were recovered from the ears of some of the elephants. As these parasites were discovered at an advanced stage of the investigation their incidence could not be determined, but six of the eight animals that were properly examined were infested. They have since been described as a new genus and species, *Loxanoetus bassoni,* Fain 1970. Fixed tissue specimens from the ear which were examined microscopically did not reveal any lesions.

Other skin lesions

Skin lesions on the back resembling those of zinc deficiency in swine were found in approximately 4% of the 228 elephants from both the Tsende and Mahlangene areas. The lesions were characterized microscopically by hyperkeratosis, parakeratosis and acanthosis. Trace element analyses which were undertaken at that time unfortunately did not specifically include some of the affected elephants (Boyazoglu & Young, unpublished observations).

Anatomy

Only one or parts of two molars on each side of each jaw are visible and in wear at once in an elephant (Evans, 1910; Hill, 1953). As these are gradually worn they are pushed forward and eventually shed, and are replaced from behind by further growing teeth, which are aligned from front to rear like cartridges in a magazine. This feature, although well-known, is illustrated in Fig. 9 (53). Another characteristic anatomical feature, the intra-abdominal position of the testes (Evans, 1910; Hill, 1953) is shown in Fig. 9 (54).

Discussion

Many reports have been published previously on the diseases and parasites of elephants, mainly of animals in captivity (Evans, 1910; Bapu, 1936; Van der Westhuysen, 1938; Sutherland, O'Sullivan & Ohman, 1950; Carrington, 1958 and McGaughey, 1961 a, b and c, 1962 a and b) but these will not be reviewed here. The present study was confined to a limited, random number of apparently healthy elephants living under natural conditions. Consequently the results do not reflect the entire disease parameter of this mammal in the Kruger National Park. In fact, a few additional observations have already been made since the conclusion of this survey.

The most significant infectious diseases encountered were of viral nature. As noted previously, the herpes infection in the lungs has already been discussed in detail. The lymphoid nodules with inclusion-bearing syncytia in the pancreas are identical to those of the lungs, which suggests that they are caused either by the same herpes virus or by another closely related virus. The granular and ulcerative lesions in the vulva, vestibulum and buccal cavity are also thought be be of viral origin. These possibilities, however, await further investigation.

The aetiology of the inflammatory lesions in the urinary bladder was not determined and speculation about them at this stage is fruitless. The possibility that the mucinous changes could still be within the normal physiological range is considered. The IC granules are probably due either to degeneration or to a virus.

The other important group of diseases is caused by parasites such as the filarid from the portal veins in the liver. This parasite, formerly classified as *Filaria gossi* Baylis, 1923, was originally described from female specimens submitted to the British Museum (Natural History). The position in the host was given by the collector as the stomach but Baylis (1923) regarded it as the peritoneal cavity. Subsequently the parasite was put in a new genus, *Loxodontofilaria* by Van den Berghe & Gillain (1939), but Chabaud (1952) reclassified it again as a *Filaria* sp. and stated that *Loxodontofilaria* is synonymous with *Dipetalonema*. Yeh (1957) who did not mention *F. gossi* in his revision of the genus *Dipetalonema,* regarded *Loxodontofilaria* as a *genus inquirenda*. Yamaguti (1961) classified both *F. gossi* and another filarid *L. loxodontis* Van den Berghe & Gillain, 1939 as *Dipetalonema* spp. The filarids recovered from the portal veins during the present investigation included both females, which were identical with the description of *F. gossi,* and males, which will be described in another paper. However, following Yamaguti's classification they were identified as *D. gossi*. The parasites were associated with severe vascular lesions (including thrombophlebitis) and parenchymatous lesions. Their relatively high incidence and occasional severity make this filarid one of the most harmful parasites of the African elephant.

Attention has previously been called to lesions along the edges of an incised liver (edge lesions). In domestic ruminants with bilharziasis they are due to lymphoid proliferation around dead schistosomes in the branches of the portal vein at that level (McCully & Kruger, 1969). Identical lesions have been seen in cases of bilharziasis of the African buffalo [*Syncerus caffer* (Sparrman, 1779)] and also in response to pentastome nymphae (Basson, McCully, Kruger, Van Niekerk, Young & De Vos, 1970). They are evidently typical cellular immune responses. Lesions of thrombophlebitis caused by immature helminths were frequently observed macroscopically near the sharp edges of the liver of zebras with delafondiasis (McCully, Kruger, Basson, Ebedes & Van Niekerk, 1969). To this list of hepatic edge lesions in the portal veins the vascular lesions in the elephant produced in response to the filarid, *D. gossi*, can be added.

McCully, Van Niekerk & Basson (1967) compared some of the vascular lesions associated with various helminths in the vascular system. They noted that with dirofilariasis of the dog and fox, bilharziasis of the hippopotamus and to a lesser extent bilharziasis of domestic ruminants, as well as with cordophilosis of various antelopes there is a marked proliferative intimal response. Thrombosis in response to the live parasites causing these diseases is rare whereas it is common when some of them die. McCully *et al.* point out that, in contrast, thrombosis in the intrahepatic portions of the portal vein in cases of delafondiasis of the zebra is commonly associated with live parasites. These differences were tentatively explained on the basis that the former helminths are vascular parasites and do not attempt to penetrate the intima whereas *Delafondia vulgaris* (Looss, 1900) Skrjabin, 1933 [Syn. *Strongylus vulgaris* (Looss, 1900)] is an intestinal parasite which tries to penetrate the vessels during its migration towards its natural habitat.

The thrombophlebitis caused by *D. gossi* is probably mainly a response to the death of the filarids. The large number of dead filarids, however, could possibly indicate that the parasites are either in an aberrant location or host. Some of the peculiar, criss-crossed, proliferative, intimal reactions are probably due to the organization of thrombi but their extensiveness suggests that they are not caused by dead filarids only. It therefore seems possible that some of the thrombophlebitic and proliferative reactions are due to the live parasites.

Evans (1910) reported parasites in Burmese elephants which caused irritation and obstruction of the bile ducts, and sometimes even death. During the present survey, despite the consistent presence of *G. clathratus* and the lesions associated with them, obstruction or penetration of the duct was never observed, nor were the small, portal tributaries of the bile ducts ever directly involved. Secondary bacterial infection occurred frequently and it is possible that along with ulceration and the blood-sucking habits of the parasite, severe hepatic lesions may be caused. Evidently some of the parasites occasionally do go astray and enter the pancreatic ducts where mild lesions are produced. This latter finding adds considerably to the significance of this parasite.

P. africanum is another very harmful parasite which causes ulceration of the stomach. According to Evans (1910), Cobbold (1882) had associated *Parabronema indicum* Baylis, 1921 of the Indian elephant with small tumors in the stomach. Sutherland *et al.* (1950) also recorded a large abscess in the stomach of an elephant containing many *P. indicum*.

Arteriosclerosis of both captive elephants in zoological gardens and free-living elephants in Kenya has previously been reported by Zvetaera (1940), Lindsay *et al.* (1956) and Sikes (1967) (according to Sikes, 1971), and it is interesting that it also occurs in elephants in the Kruger National Park. Its incidence, however, appears to be very low.

The periuterine, fibrous papillomata occurred only in adult or aged animals. According to Sikes (1971), Zvetaera (1941) has reported a case of uterine fibroma in the Moscow Zoo. The ear mite, *L. bassoni,* did not appear to cause any lesions in the animals examined.

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