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STUDIES ON SPECIFIC OCULO-VASCULAR MYIASIS (UITPEULOOG) IN SHEEP. V. HISTOPATHOLOGY

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ABSTRACT

BASSON, P. A. Studies on specific oculo-vascular myiasis (uitpeuloog) in sheep. V. Histopathology. *Onderstepoort J. vet. Res.*, 36 (2), 217–232.

Specimens from 51 sheep and one goat were studied microscopically. Myiasis by *Gedoelstia* larvae occurred mainly in the organs of the head, neck and thorax, the eyes, brain, heart and blood vessels being most frequently involved. The fundamental lesion was thrombovasculitis which gave rise to glaucoma and other marked lesions in the eyes, infarction of the myocardium, lungs and kidneys and encephalomalacia in various parts of the brain. The migratory pattern of the larva appeared to be primarily intravascular, but extravascular routes such as those along the optic fasciculus and nerves of the head were also observed. The generic name, gedoelstiasis, is introduced for this specific type of myiasis.

INTRODUCTION

The first stage larvae of the oestrid flies, *Gedoelstia hässleri* Gedoelst, 1915, *Gedoelstia cristata* Rodhain & Bequaert, 1913, and their hybrids have been proved to be the cause of severe and fatal myiasis in some aberrant hosts such as the sheep (Basson, 1962a, b, c). In the blue wildebeest [*Connochaetes taurinus* (Burchell, 1823)] and red hartebeest [*Alcelaphus buselaphus* (Pallas, 1766)] which serve as natural hosts, only very mild or mild non-fatal lesions have been determined (Basson, 1966a). The present study deals mainly with the histopathology in sheep. Most of the macroscopical lesions have been described in the previous studies and only relevant additional features are recorded here. Although cattle and goats are also susceptible, mortality is usually rare. For this reason this study, with the exception of one goat, includes only sheep. As attempts to breed *Gedoelstia* larvae in captivity were unsuccessful, the production of experimental cases of the disease depended on the chance capture of a gravid female in the veld. This occurred once when two sheep were infested (Basson, 1962c). With the exception of these two cases, the pathological studies in this report are limited to natural cases.

MATERIALS AND METHODS

A total of 141 sheep and one goat suffering from the natural disease was examined, autopsies being performed either on animals which had died naturally or which were sacrificed at various stages of the disease. The two experimental cases (*vide supra*) were autopsied five and 17 days after infestation. After the migratory routes had been determined in each carcase, specimens of various tissues from 51 of the sheep and the goat were collected in 10 per cent formalin. The majority of the specimens were collected from macroscopically affected organs and were subsequently processed for histopathological study in a routine manner by paraffin embedding, sectioning with a sliding microtome at 3 to 6 μ thickness and staining with haematoxylin and eosin (HE). Some of the brains were fixed *in toto*, coronal sections of 4 mm in thickness being cut from them for the detection of gross lesions. Appropriate parts were then selected for sectioning. The eye sections varied from 6 to 10 μ in thickness and were stretched on a water bath at 50 °C before staining. Because of the technical problems in preparing suitable sections from eyes which contained intact lenses, the latter were removed in most cases before embedding and only a few were retained for study.

RESULTS

Macroscopical findings

The most significant findings were reported by Basson (1962c) to which the following observations, previously undescribed, are now added. The extensive thrombosis and vasculitis of the ophthalmic veins were frequently accompanied by similar lesions in the ophthalmic emissary vein and cavernous sinus. The ventral petrosal sinus was affected in some of the animals. The most frequent and most significant brain lesion was encephalomalacia, which was either pale or haemorrhagic in appearance. One or more areas in a brain were affected and, although the cerebellum proved to be the part most frequently involved, there appeared to be no specific predilection site. The majority of the lesions were confined to the cerebellar and cerebral cortex and to the roof nuclear area of the cerebellum, hippocampus, thalamus and midbrain, in more or less decreasing order of frequency. The size of the affected areas varied tremendously [Plate 1 (3 to 9)].

Microscopical findings

The histopathological findings are listed in Table 1 but, because most of the sections examined were prepared from selected areas, this table does not necessarily reflect the true picture of vulnerability of the tissues. In the survey reported previously (Basson, 1962c) the myocardium was affected in 5 to 22 per cent, the brain in 15 to 30 per cent and the eyes in 96 per cent of the cases examined.

Blood vessels

The most outstanding lesions in almost every organ proved to be of vascular nature. Thrombosis of some of the vessels was invariably present especially in the head and neck regions [Plate 1 (1 and 2)]. The veins, venous sinuses such as the cavernous sinus, and pulmonary artery were more frequently affected than the true arteries. The latter included those in

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TABLE 1. — *Distribution of microscopical lesions*

	Eyes	Lacrimal gland	Kidney	Liver	Spleen	Lymph nodes	Nasal mucosa	Lungs
No. examined	30	4	25	24	21	20	9	18
Pos. for lesions (%)	100	100	24	21	67*1	25*2	33	33
Pos. for thrombi or vasculitis (%)	80		16	13	—	5	33	28
Pos. for infarcts (%)			16	—	—	—	—	11
Pos. for larvae (%)	43	—	—	—	—	—	—	17

	Brain	Optic fasciculus + meninges	Periorbital nerves	Trigeminal nerve	Gasserian ganglion	Spinal cord	Hypophysis	Adrenal
No. examined	30	30	30	7	7	12	8	11
Pos. for lesions (%)	100	77	23	100	71	16	37	9
Pos. for thrombi or vasculitis (%)	53	3						9
Pos. for infarcts or encephalomalacia (%)	56	3				—		—
Pos. for larvae (%)	12	4	3	14	—	—	—	—

	Heart	Ophthalmic veins	Cavernous sinus	Jugular veins + tributaries	Pulmonary artery	Aorta	Arteries in brain	Veins in brain
No. examined	30	24	15	16	14	5	32	32
Pos. for lesions (%)	70	75	93	100	100	80	22	17
Pos. for thrombi or vasculitis (%)	27	75	93	100	100	80	22	17
Pos. for infarcts (%)	27							
Pos. for larvae (%)	7	17	33	25	29	—	—	3

*1 Secondary type lesions
*2 Both secondary and primary lesions

the periocular area, the internal carotid, aorta, coronary and renal arteries. Involvement of the pulmonary cusps of the heart was often seen in cases where the pulmonary artery was affected [Plate 7 (54)]. The size of the thrombi varied from very small disseminated parietal foci to extensive and even occlusive ones, and could be classified according to the stage and type of formation such as coral, laminated, fibrinoid, mixed, semi-organized and organized. In cases where reinfestation had occurred recent thrombi were superimposed on partially organized ones. Both eosinophil and neutrophil infiltrations were present in many thrombi. Various numbers of these leucocytes also infiltrated the walls of the thrombosed vessels adjacent to the affected parts and at a later stage were partially substituted or accompanied by round cells. Mild, localized karyorrhexis was sometimes observed in the intima. Localised fibrinoid necrosis of all the layers of the vessel wall was an infrequent observation. Mild or sometimes fairly prominent haemorrhages and mild perivascular leucocytic infiltrates of mixed nature were fairly regularly seen. Larvae (Table 1), located with difficulty, were found either between the thrombus and the intima or within the thrombus. Vascular parasitic microgranulomas were sometimes observed and in one case eosinophilic clubs surrounded the entrapped larva [Plate 10 (74)].

Brain

Sections from the affected brains of 30 sheep were examined microscopically. Lesions consisted of areas of encephalomalacia, meningo-encephalitis and oedema. The areas of encephalomalacia present in 56 per cent of the cases were either anaemic, haemorrhagic or of both types (Plate 1). Accompanying thromboses of the arteries and veins were found in sections from 22 and 17 per cent of the cases respectively [Plate 2 (12)].

All the various stages of encephalomalacia except the cystic stage were encountered (Plate 3). Lesions of an estimated 6 to 24 hours duration were swollen, pale, eosinophilic and well-demarcated with necrosis of all the elements except the blood vessels and some of the glial cells. The blood vessels were congested and small per-diapedetic haemorrhages were present. These haemorrhages were more profuse in haemorrhagic infarcts, but it could not be established with absolute certainty whether the veins only were involved in such cases. A more intense acidophilia of the tissue in both grey and white matter was apparently the next stage after the original state of pallor, and appeared to be more severe and of longer duration in the grey matter. The irregular demarcation zone became rarified and microcavitated and swollen axis cylinders appeared within and outside this zone. This was followed by a progressive disintegration of the necrotic tissue, that of the white matter occurring more rapidly and reaching a state of pallor and rarefaction before that of the grey matter. The small blood vessels and capillaries became more distinct because of endothelial hypertrophy and hyperplasia. The next stage noticed occurred after about two to four days when gitter cells formed at the periphery of the microcavitated demarcation zone. Various degrees of perivascular cuffing, usually

mild, or vasculitis of mixed cell type including lymphocytes, eosinophils and neutrophils were frequently present either within the demarcation or lytic zone. The changes in the tissues surrounding these areas of encephalomalacia consisted mainly of various degrees of oedema or brain swelling with swollen glial cells, congestion, haemorrhages, a mixed cell type of meningo-encephalitis, Wallerian and neuronal degeneration [Plate 2 (15) and Plate 3].

Progressive gitter cell formation towards the centre of the lesion eventually led to a complete or almost complete inundation with these foamy macrophages. Their formation seemed to extend more frequently alongside vessels and it appeared to be more rapid in the white matter. In the grey matter other types of macrophages, originating either from the perivascular elements or infiltrating mononuclear cells, were also present. Some of them contained calcium granules. Epithelioid and multinucleated giant cells were found in a few cases, one of which was the 17-day old experimental case [Plate 2 (16)]. These lesions evidently represented parasitic granulomas which had been provoked by the migratory larvae.

Mild, or occasionally somewhat more severe, focal disseminated or localized eosinophilic, lymphocytic or mixed cell meningo-encephalitis with accompanying congestion, and some haemorrhages in the absence of encephalomalacia, were found in 22 per cent of the brain specimens [Plate 2 (13 and 14)]. Either the leptomeninx or the pachymeninx, sometimes both, were affected. Nine per cent of the brains were only congested and oedematous with some small scattered haemorrhages, 6 per cent had only small areas of Wallerian degeneration and another 6 per cent just thrombosis in one or two small vessels. Most of these brains, however, were not collected *in toto* and consequently it was not always possible to exclude encephalomalacia.

Gedoelstia larvae were difficult to detect and were observed either intra- or extravascularly in only 12 per cent of the affected brains. The distribution of the lesions is given in Table 2.

Eyes

The affected eyes and periocular tissues of 30 sheep were studied and the most significant lesions are listed in Table 3. The primary lesions were traumatic in nature due to larval migration, and consisted of haemorrhages, thromboses, congestion, oedema and, depending on their severity, also degeneration and necrosis. The veins were frequently occluded by thrombi with resultant glaucoma development, but relatively few arteries were affected. These primary lesions were subsequently infiltrated by eosinophils, neutrophils and lymphocytes. With secondary bacterial infection the neutrophil reaction became more pronounced.

TABLE 2.—*Distribution of brain lesions*

Item	Pos. for lesions	Encephalomalacia
	(%)	(%)
Cerebral cortex	47	20
Thalamus	27	10
Corpus striatum	7	7
Hippocampus	13	13
Lateral geniculate	3	—
Amygdaloid nucleus	7	3
Hypothalamus	3	—
Corpora quadrigemina	23	10
Cerebellar cortex	57	30
Roof nuclear area	13	13
Medulla oblongata	17	3
Choroid plexus	3	—
Spinal cord	7	—

The larvae apparently migrated either via the blood vessels, along the optic nerve or through the tissues and were consequently found anywhere in the orbital tissues and its vessels [Plate 6 (51)]. Some were dead, sometimes calcified and surrounded by a granulomatous response [Plate 5 (43)].

Periocular tissues, optic nerve and conjunctiva (*Plate* 4)*:* All the lesions in the periocular tissues were of the above nature. Dacryoadenitis, blepharitis and conjunctivitis, together with primary lesions of thrombosis, haemorrhages and oedema, occurred in several of the cases where these tissues were examined. Haemorrhages, polymorphonuclear cell infiltrates and larvae were found around the optic fasciculus, within the meninges or subdural space. Demyelination and malacic lesions in the optic fasciculus were seen in 17 per cent of the cases and thrombosis of the central vein in one animal. Neuritis and Wallerian degeneration, sometimes in the presence of larvae, were also encountered in some of the periocular nerves.

Cornea and sclera: Thrombosis of the anterior ciliary and vortex veins was seen frequently, but scleritis was very rare [Plate 5 (39)]. Purulent keratitis occurred in most of the sheep. Corneal ulceration with perforation and rupture of Descemet's membrane was found in 17 per cent of the eyes examined [Plate 4(32)]. Precancerous melanosis in the corneal epithelium and substantia propria starting at the limbus and progressing towards the centre was occasionally encountered in cases of chronic kerato-conjunctivitis.

Uveal tract (*Plate* 5)*:* Haemorrhages, thrombosis and oedema were prominent and involved all three layers, namely the iris, ciliary body and choroid. The ciliary body, anterior portion of the choroid and iris, especially the areas around the filtration angle, were the parts most frequently injured by the migrating larvae found in these localities. Purulent iridocyclitis succeeded in most of the cases but choroiditis was rare. With increasing chronicity, round cell reactions and fibroplasia followed and eventually gave rise to anterior and posterior synechiae and iris bombé. Irregular proliferations of the injured uveal tract were noticed in one eye.

Retina (*Plate* 6)*:* Again, as in all the other tissues, haemorrhages, oedema and degenerative changes were the commonest, and could involve all the layers. A status spongiosus was noticed in both plexiform and nerve fibre layers as well as Wallerian degeneration in the latter. Vasculitis, mild retinitis, thinning and disappearance of the granular and ganglionic layers as a result of degeneration and necrosis and ultimate atrophy were not uncommon. An architectural disarray involving most of the layers and gliosis were also found. Foamy, lipid macrophages resembling gitter cells, as well as macrophages with phagocytozed erythrocytes and necrotic debris from

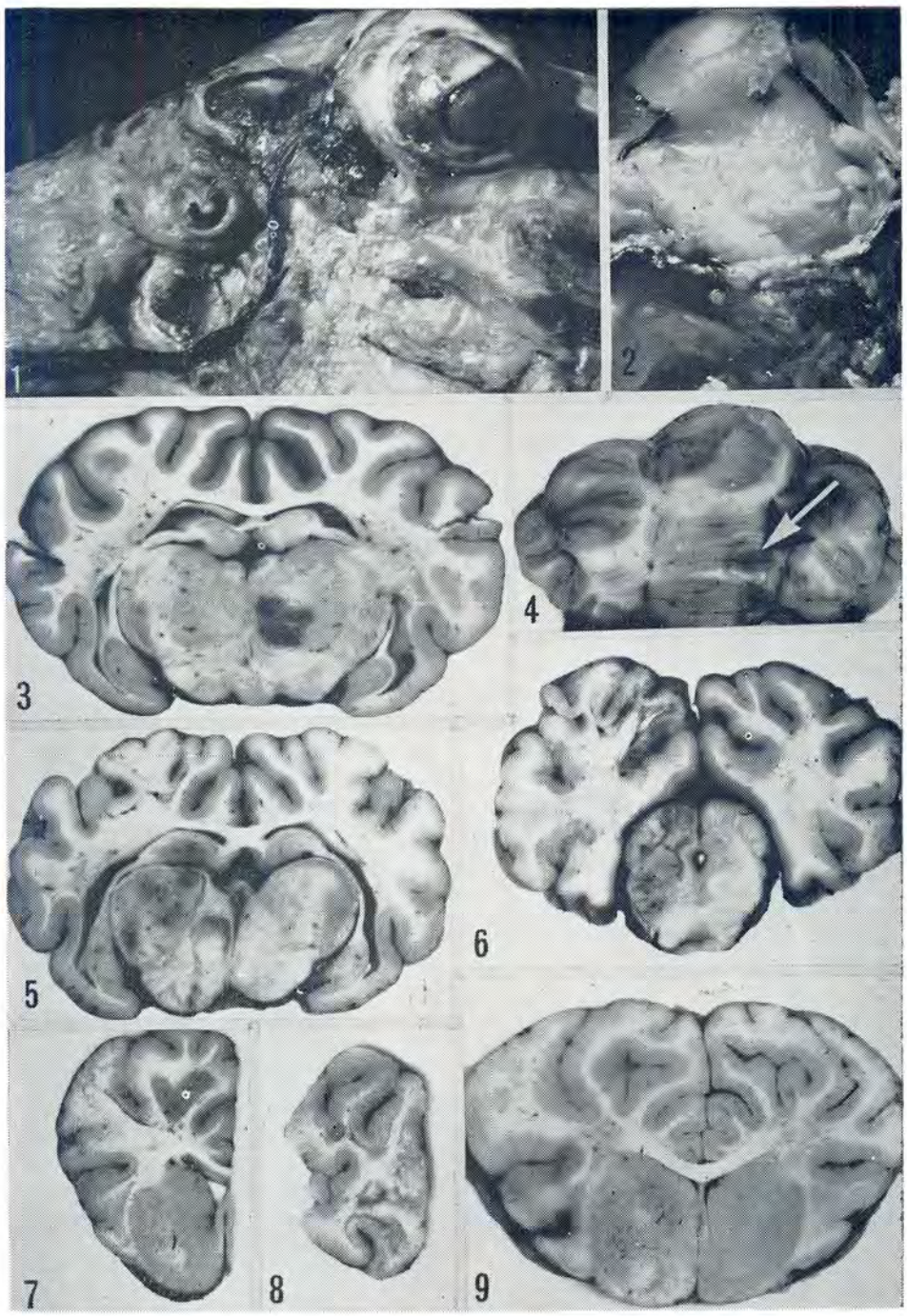

PLATE 1.—1. Dissected, thrombosed jugular vein and internal maxillary vein with its tributaries. Keratitis is also evident. 2. Dissected, thrombosed ophthalmic veins and ophthalmic emissary vein of an affected eye. 3. A haemorrhagic area of encephalomalacia in the right thalamus. 4. Encephalomalacic area in the cerebellum showing a clear zone of demarcation. 5. Encephalomalacia of the left thalamus, lateral geniculate body, hippocampus and upper cerebral cortex. A few haemorrhages are also present. 6. Encephalomalacia of the left midbrain and cerebral cortex. The demarcation zone with separation from the normal tissue is prominent in the latter. 7 & 8. Areas of encephalomalacia of the cerebral cortex. 9. Very early swollen stage of encephalomalacia on the left side involving part of the corpus striatum and cerebral cortex. The affected area is slightly darker and distinctly swollen. The latter is clearly noticeable if the cortical width of the two sides is compared

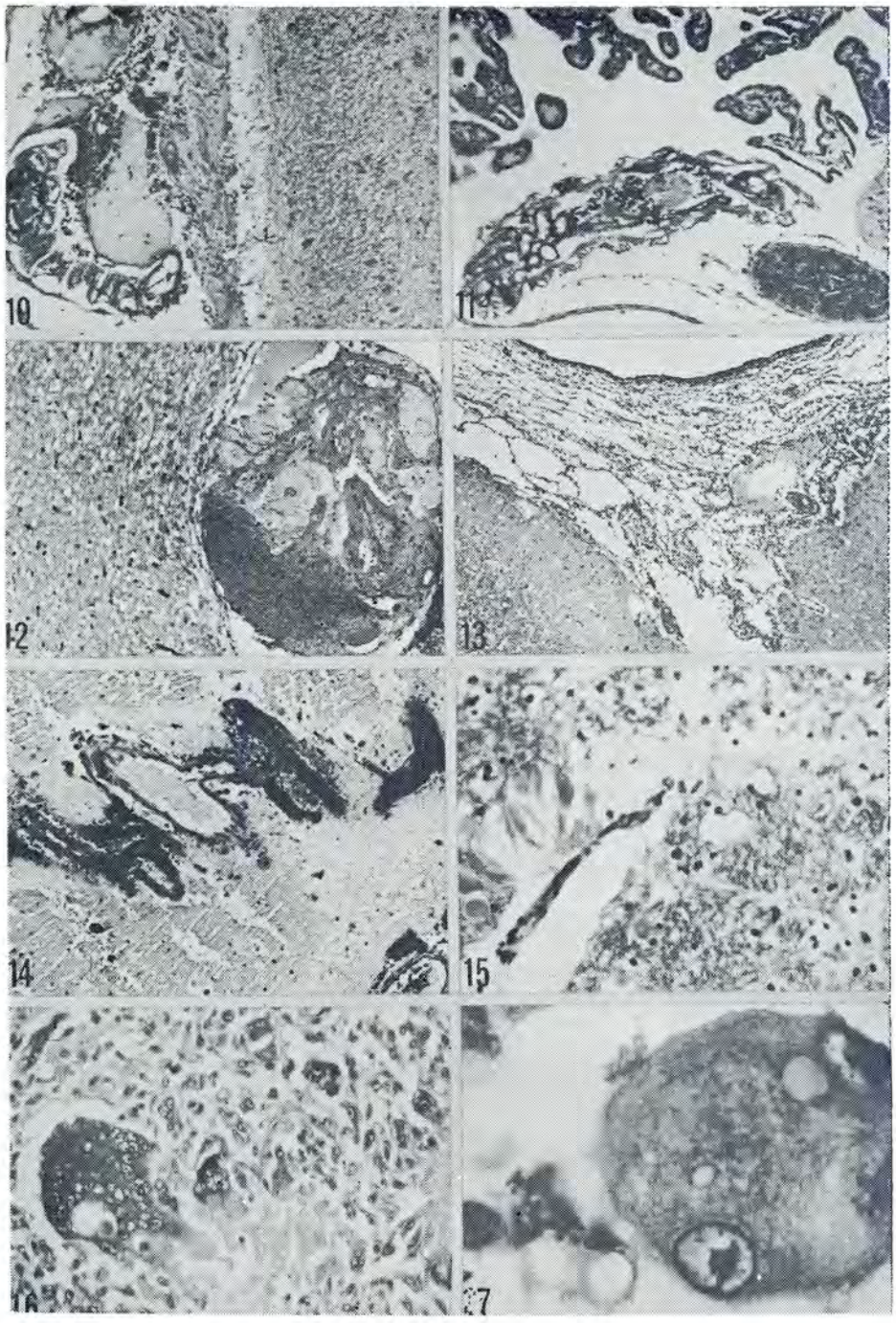

PLATE 2.—10. Brain: A *Gedoelstia* larva in a vein within the leptomeninx. HE × 75. 11. Cerebellum: A *Gedoelstia* larva in an extravascular position within the choroid plexus. HE × 75. 12. Brain: Venous thrombosis in the leptomeninx. HE × 75. 13. Cerebral cortex: Seropurulent leptomeningitis. HE × 30. 14. Cerebellar cortex: Haemorrhages, oedema and perivascular necrosis in the leptomeninx and cortical necrosis. HE × 75. 15. Brain: White matter outside an encephalomalitic area with Wallerian degeneration. HE × 200. 16. Cerebrum: Granulomatous reaction with a very large giant cell in the brain of a 17-day old experimental case. HE × 200. 17. Brain: Giant cell in another case containing some pigment granules which proved to be negative for iron. HE × 1200

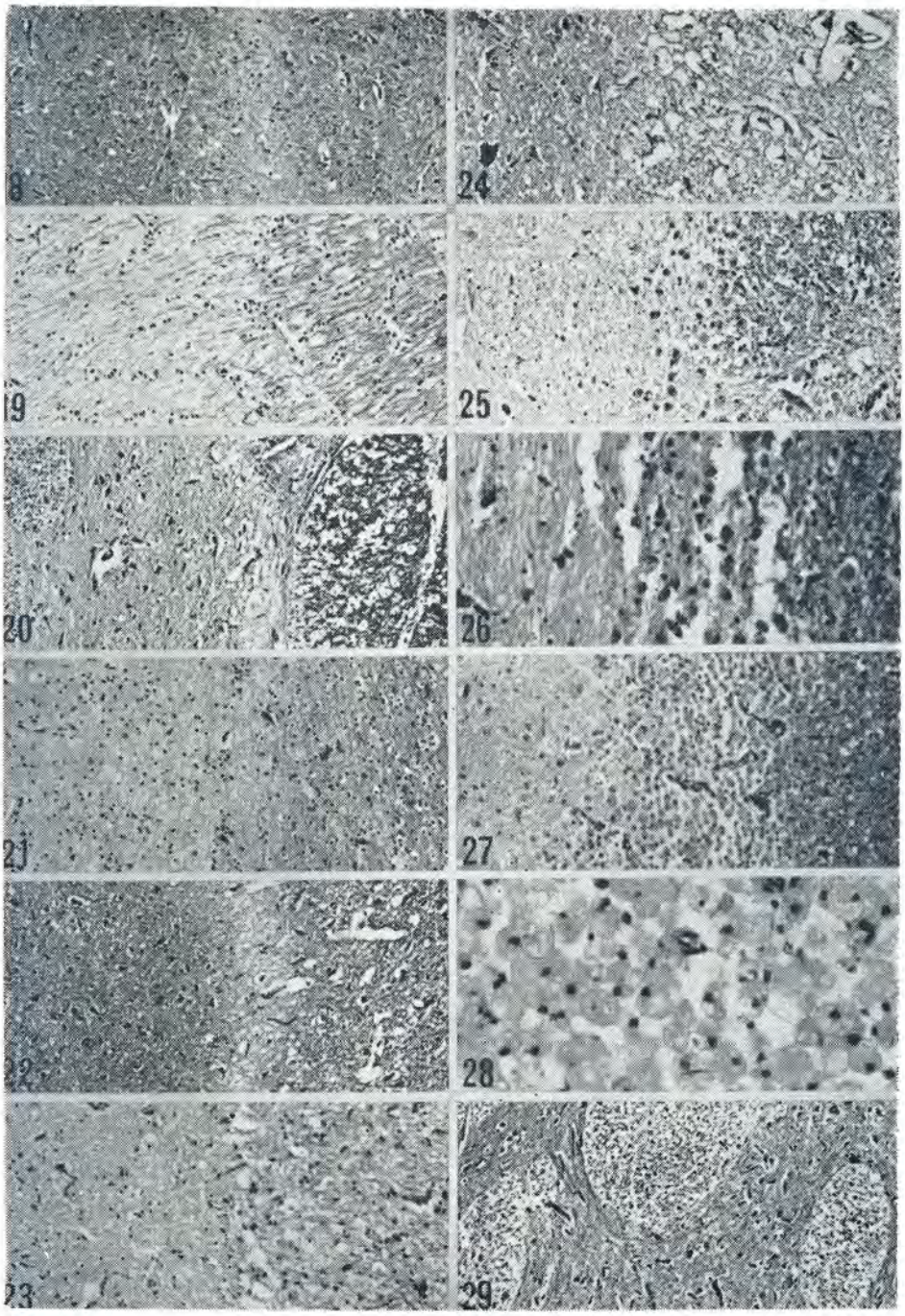

PLATE 3.—Various stages of encephalomalacia: The photomicrographs are arranged in such a way that the affected area is always on the left side and the demarcation zone in a paramedian position except in 28 and 29 which represent areas within the encephalomalitic areas. 18. Very early stage where the affected area is practically indistinguishable from the normal. The distinct demarcation zone in such cases, however, is of considerable assistance. Photomicrograph from a 5-day old experimental case. HE × 75. 19. Another early stage in the white matter showing the distinct pallor of the necrotic area. HE × 75. 20. An area within the corpus striatum with a faint demarcation zone and a normal tract on the right. HE × 75. 21. Slightly more advanced necrosis and pallor in the affected area. HE × 75. 22. Increased eosinophilia in the necrotic grey matter. HE × 75. 23. More advanced stage of necrosis and pallor, microcavitation of the demarcation zone and a few swollen axis cylinders in the surrounding area. HE × 75. 24. Increased eosinophilia of the affected area and microcavitation in the demarcation zone. HE × 75. 25. Onset of gitter cell formation in the demarcation zone. Distinct pallor of necrotic area as well as a few swollen axis cylinders in the surrounding zone. HE × 75. 26. Higher magnification of a slightly more advanced stage. HE × 95. 27. Progressive development of gitter cell zone and prominence of small vessels. HE × 50. 28. Photomicrograph from an area of complete gitter cell formation. HE × 200. 29. Corpus striatum: An area of early encephalomalacia showing the contrast between the grey matter (increased eosinophilia) and tracts of white matter (pallor). HE × 75

the rods and cones, were noticeable in one case. In one of the seriously injured and ruptured eyes gliosis of the retina was evident.

Lens: Most of the lenses were removed before embedding and only a few were sectioned. Some of these were necrotic and one contained a mild infiltration of eosinophils.

Heart

Thrombosis of the coronary arteries [Plate 7(53)] was responsible for the development of infarcts in the myocardium [Plate 7(52)]. Eight such cases (27 per cent) were studied microscopically and various stages of infarction revealed by necrosis, haemorrhages, eosinophil infiltration and fibroplasia were seen [Plate 7(55)]. Necrotic fibres were sometimes partially mineralized. Various degrees of localized eosinophilic myocarditis or one of a mixed cell type was found in 40 per cent and a similar endocarditis occurred in about 27 per cent of the cases. Various degrees of haemorrhages and oedema of the myocardium were frequent concomitant findings and parasitic microgranulomata occurred in some of the older lesions. The endocardial lesions resembled the thrombophlebitis elsewhere [Plate 8(57 and 58)]. Larvae were noticed in 7 per cent of the hearts examined.

Lungs

The pulmonary artery and its intrapulmonary branches were often thrombosed and inflamed [Plate 7(54), Plate 8(59, 60, 62 and 63)], but these changes only rarely gave rise to typical red pulmonary infarcts. Only two such cases with prominent congestion, haemorrhages, necrosis and eosinophil infiltration were encountered. Congestion, oedema, haemorrhages and emphysema were also concurrent features in other parts of the lung. Generalized oedema followed after cardiac insufficiency. Entrapped larvae were either found within the branches of the pulmonary artery or free within the lung tissue [Plate 7(56), Plate 8(61, 62 and 64)]. Eosinophil reactions in the surrounding areas were evidently provoked by either live or dead larvae. Parasitic microgranulomas were found in the lungs of three sheep.

Kidneys and liver

Four cases with renal thrombosis and infarcts were studied. Three of these were of an acute nature and one was subacute. Thrombosis of an artery [Plate 9(68)] was usually accompanied by vasculitis and sometimes by fibrinoid degeneration of the wall of the vessel. Mild nephrosis and localized areas of either eosinophilic or lymphocytic nephritis were occasionally present [Plate 9(67)]. In general the type of cell reaction varied from predominantly eosinophilic in the acute stages to either a mixed or lymphocytic type in subacute cases.

Eosinophilic vasculitis and perivasculitis of the portal and hepatic veins with adjoining areas of hepatitis were encountered in four sheep [Plate 9(66)], one of which had a thrombus in an intrahepatic branch of the portal vein. Disseminated necrotic areas with haemorrhages in one sheep were regarded as being probable migratory tracts. Small foci of purulent hepatitis were seen in another animal. Cloudy swelling, hydropic degeneration and fatty changes occurred in most of the sheep which had lesions in the pulmonary artery and myocardium or which were recumbent as a result of the encephalitic form of the disease.

Other organs and tissues

The marginal zones around the splenic corpuscles were usually prominent and contained many eosinophils, but thrombi and larvae were not found in the spleen [Plate 9(70)]. Twenty per cent of the lymph nodes examined revealed lesions, the commonest being a mild eosinophilic lymphadenitis. Haemorrhages, congestion and oedema occurred both within and around the nodes. Thrombosis, however, was seen only once. Eight pituitary glands were examined and 38 per cent had prominent and fairly large areas of degeneration, necrosis, haemorrhages and eosinophi reactions in the anterior pituitary [Plate 9(65), Plate

TABLE 3. — *Distribution of various ocular lesions*

Item	Total examined	Pos. for lesions	Pos. for larvae	Haemorrhages	Thrombosis	Advanced oedema	Degeneration or necrosis	Leucocytic reaction	Parasitic granulomas
		(%)	(%)	(%)	(%)	(%)	(%)	(%)	(%)
Periocular tissue	24	96	25	50		17		63	13
Veins	24	75			67			29	
Arteries	24	12.5			12.5			4	
Nerves.....	24	33					13	29	
Conjunctiva	19	74	—	11	10	5	5	68	—
Optic fasciculus + meninges	30	77	7	33	3	3	17	37	—
Cornea	30	70	—	—	—		33	60	—
Sclera	22	32	5	—			—	9	—
Vortex veins	22	18			18				
Anterior ciliary veins	22	14			14				
Uveal tract	29	83	14				17		3
Choroid	29	41		28	21	14		7	
Iris	29	52		14	7			34	
Ciliary body	29	76		31	41	10		45	3
Filtration angle	29	24		10				31	
Retina	22	45	—	14	—	5	31	14	—
Lens	2	50	—	—	—		50	50	—
Anterior eye chamber	22	59	—	31	—			14	—
Posterior eye chamber	22	36	—	21	—			5	—

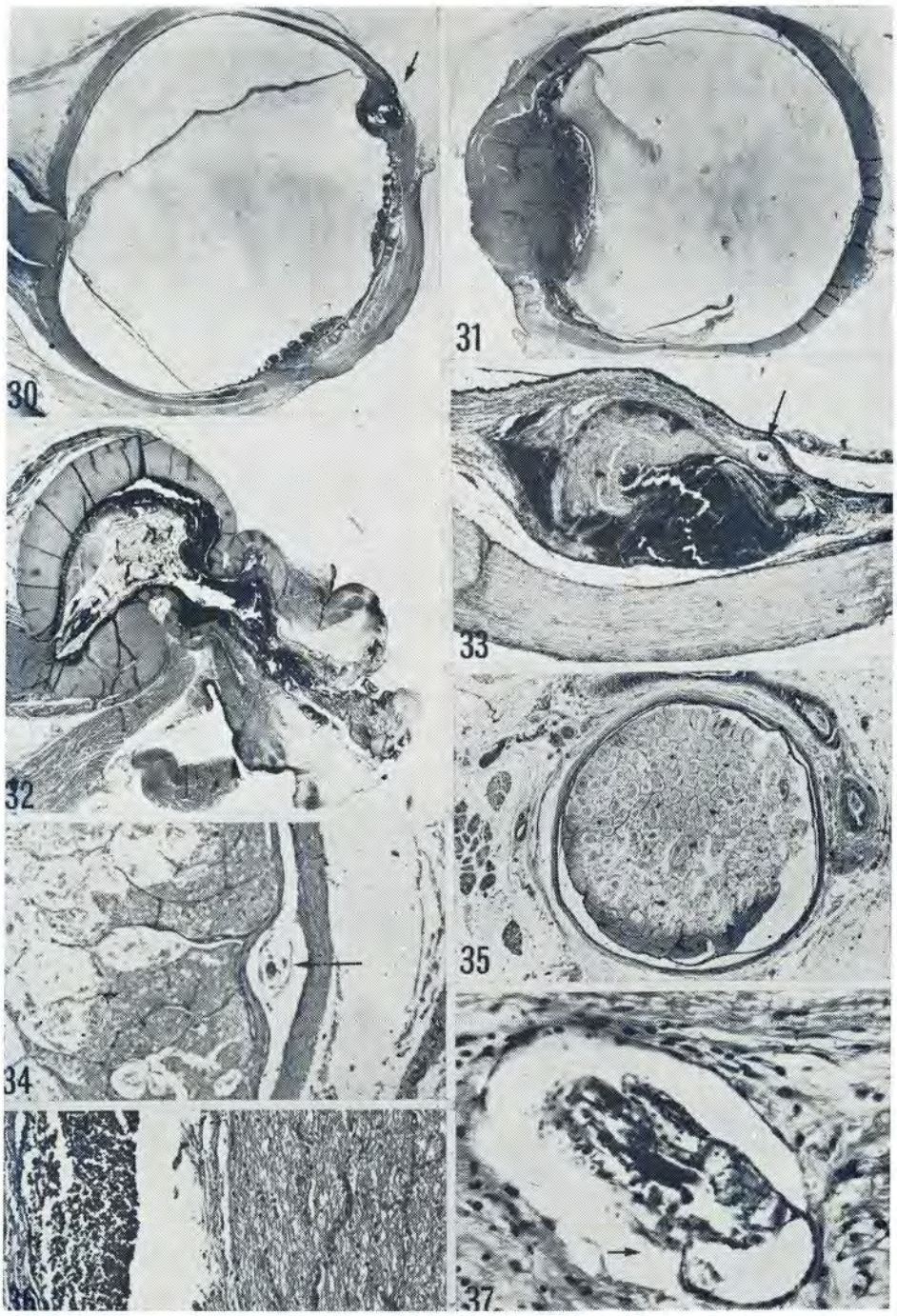

PLATE 4.—30. Affected eye with thrombosis and haemorrhages just anterior to the ora serrata. Haemorrhages in the anterior eye chamber and conjunctivitis are also noticeable. HE × 2.3. 31. Eye with anterior synechiae. HE × 2. 32. Ruptured eye. HE × 2.5. 33. Thrombosis at the ora serrata with a larva in cross section (arrow) just outside the affected vein. HE × 20. 34. Optic fasciculus showing partial malacia and a larva in cross section within the subdural space (arrow). HE × 30. 35. Optic fasciculus with almost entire necrosis. Only the lower darker zone is spared. HE × 9. 36. Purulent reaction in the subdural space of the optic fasciculus. HE × 75. 37. A larva within a periocular nerve. A few spines are noticeable (arrow). HE × 200

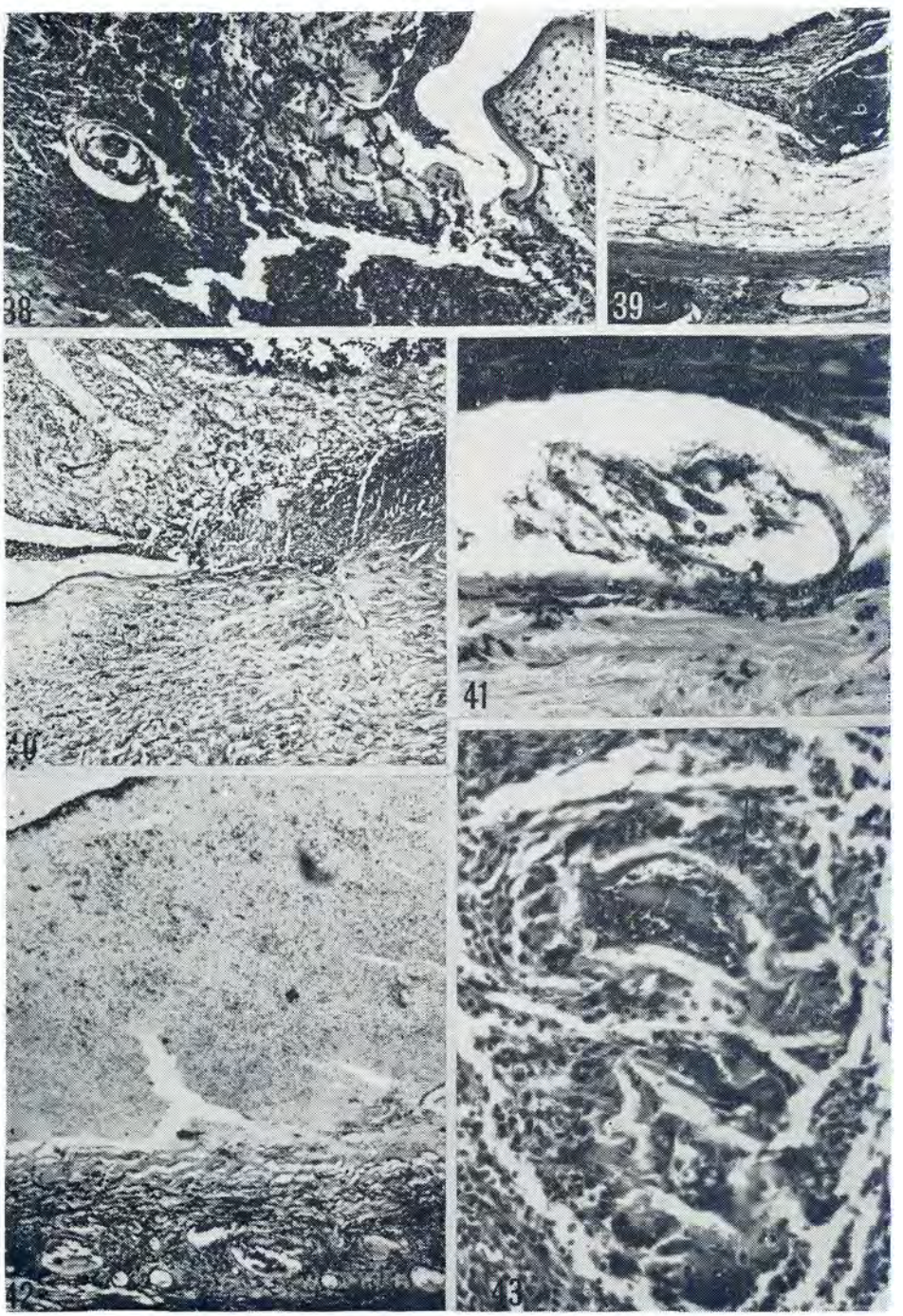

PLATE 5.—38. Filtration angle and adjoining area containing a purulent infiltrate and one entrapped larva on the left side in cross section. A piece of cornea with Descemet's membrane is noticeable on the right. HE × 50. 39. Oedema and thrombosis of the ciliary ring accompanied by thrombosis and scleritis. HE × 30. 40. Polymorphonuclear infiltrate in the region of the filtration angle and anterior eye chamber. HE × 30. 41. Larva within the outer vessel layer of the choroid. HE × 200. 42. Haemorrhages and oedema in the choroid of a ruptured eye. HE × 30. 43. Parasitic granuloma in the periocular tissue. The spines of the larva are noticeable (arrow). HE × 200

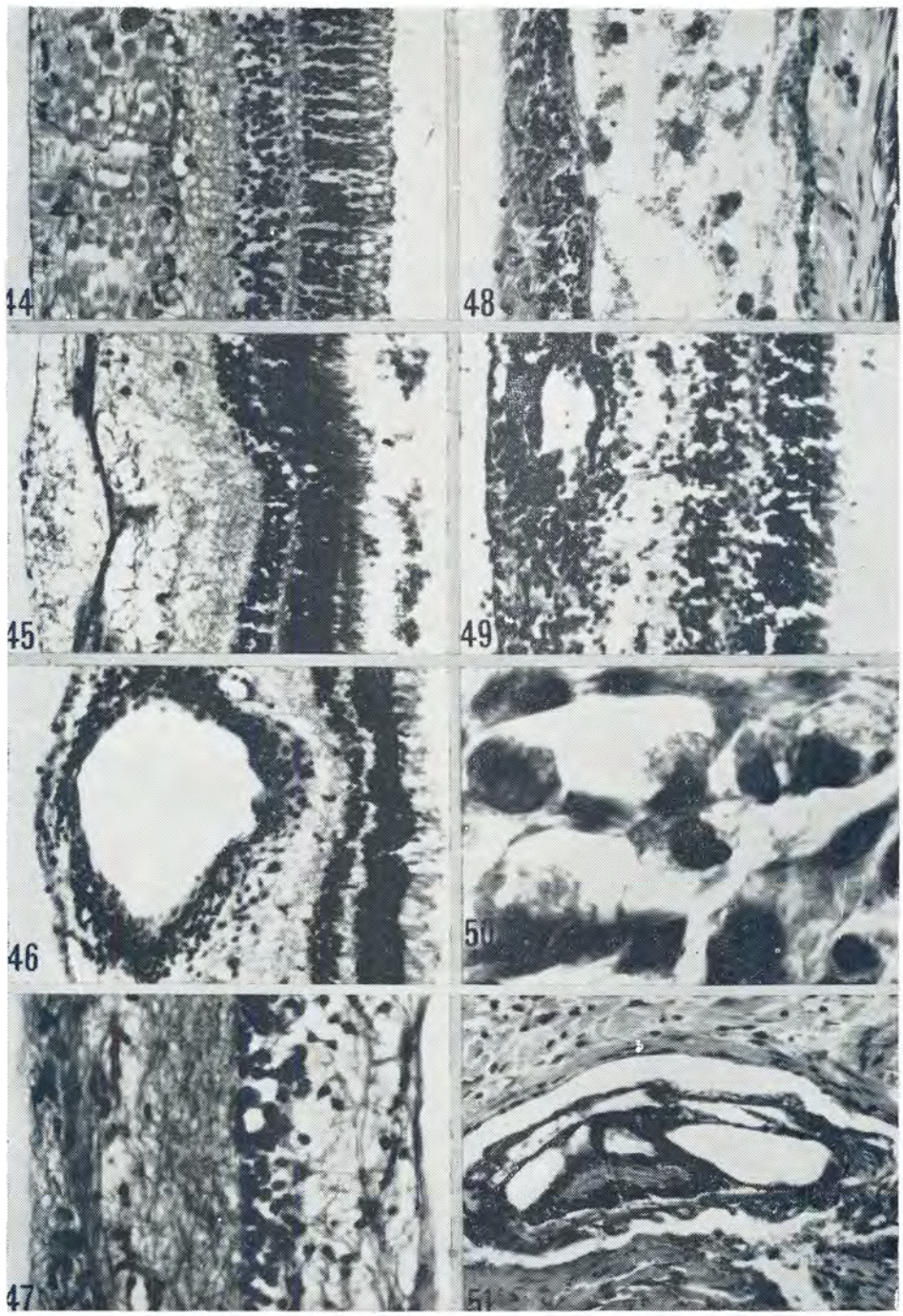

PLATE 6.—44. Retina: Wallerian degeneration of the optic nerve fibres and thinning of the ganglionic layer. HE × 200. 45. Retina: A status spongiosus of the inner layers and disappearance of the ganglionic layer. HE × 200. 46. Retina: Prominent vasculitis. HE × 200. 47. Retina: Disappearance and thinning of nuclear layers with rarefaction. HE × 200. 48. Retina: Atrophy and architectural disarray with haemorrhages and large macrophages in the disintegrated layer of rods and cones. HE × 200. 49. Retina: Retinitis. HE × 200. 50. Retina: Lipid macrophages within the inner plexiform layer. HE × 1200. 51. Periorbital vein: Larva in longitudinal section between the thrombus and the intima of the vein. Spines are noticeable ventrally. HE × 200

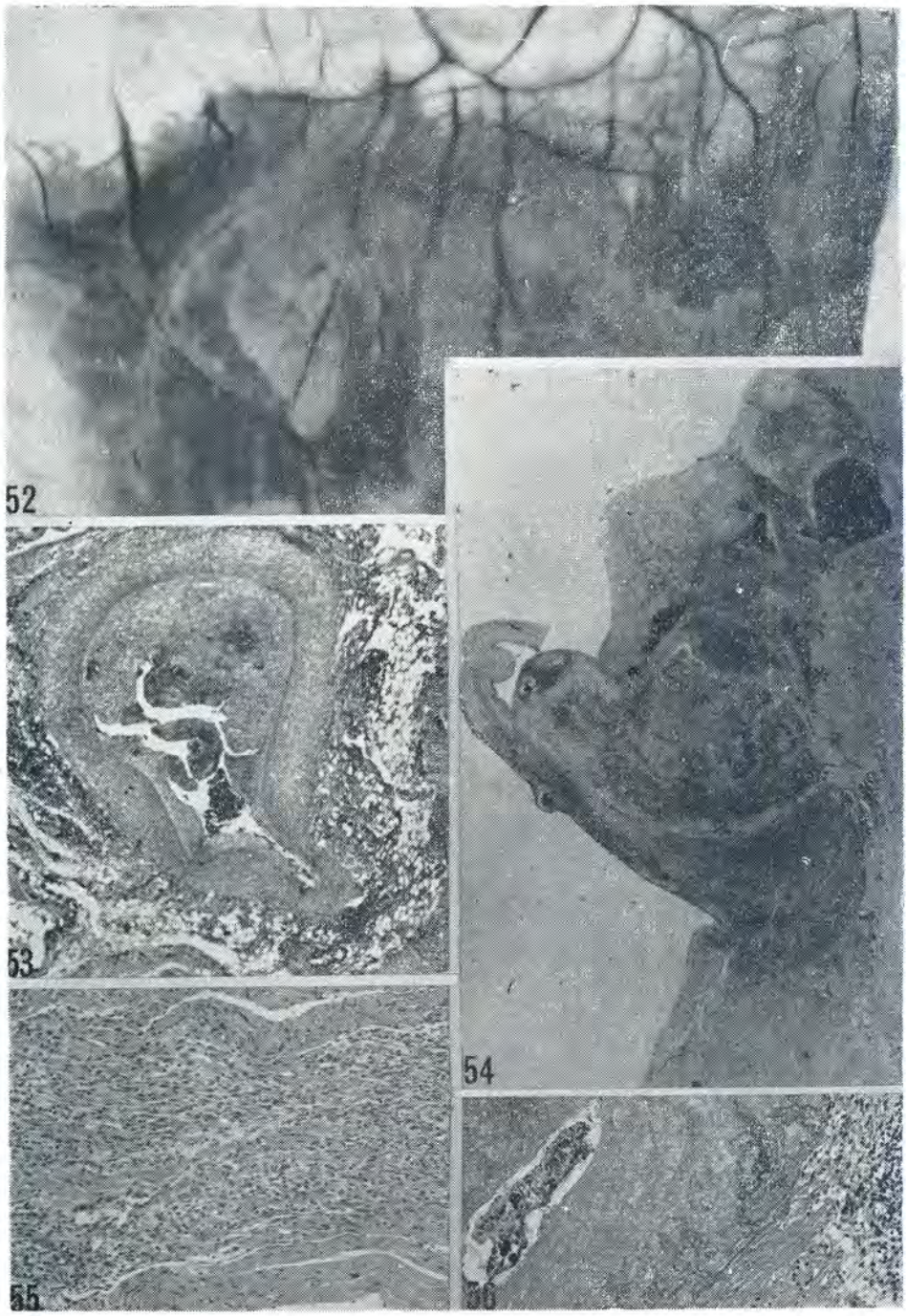

PLATE 7.—52. Myocardium with two infarcts. 53. Thrombosed coronary artery of the above myocardium. HE × 25. 54. Thrombosis of the pulmonary cusp and adjoining pulmonary artery. The intima is indicated by asterisks and a dead larva by the arrow. HE × 9. 55. Fibrosis in an area of infarcted myocardium. HE × 75. 56. Higher magnification of the dead larva in 54 which is surrounded by caseous material and a mixed cell infiltrate

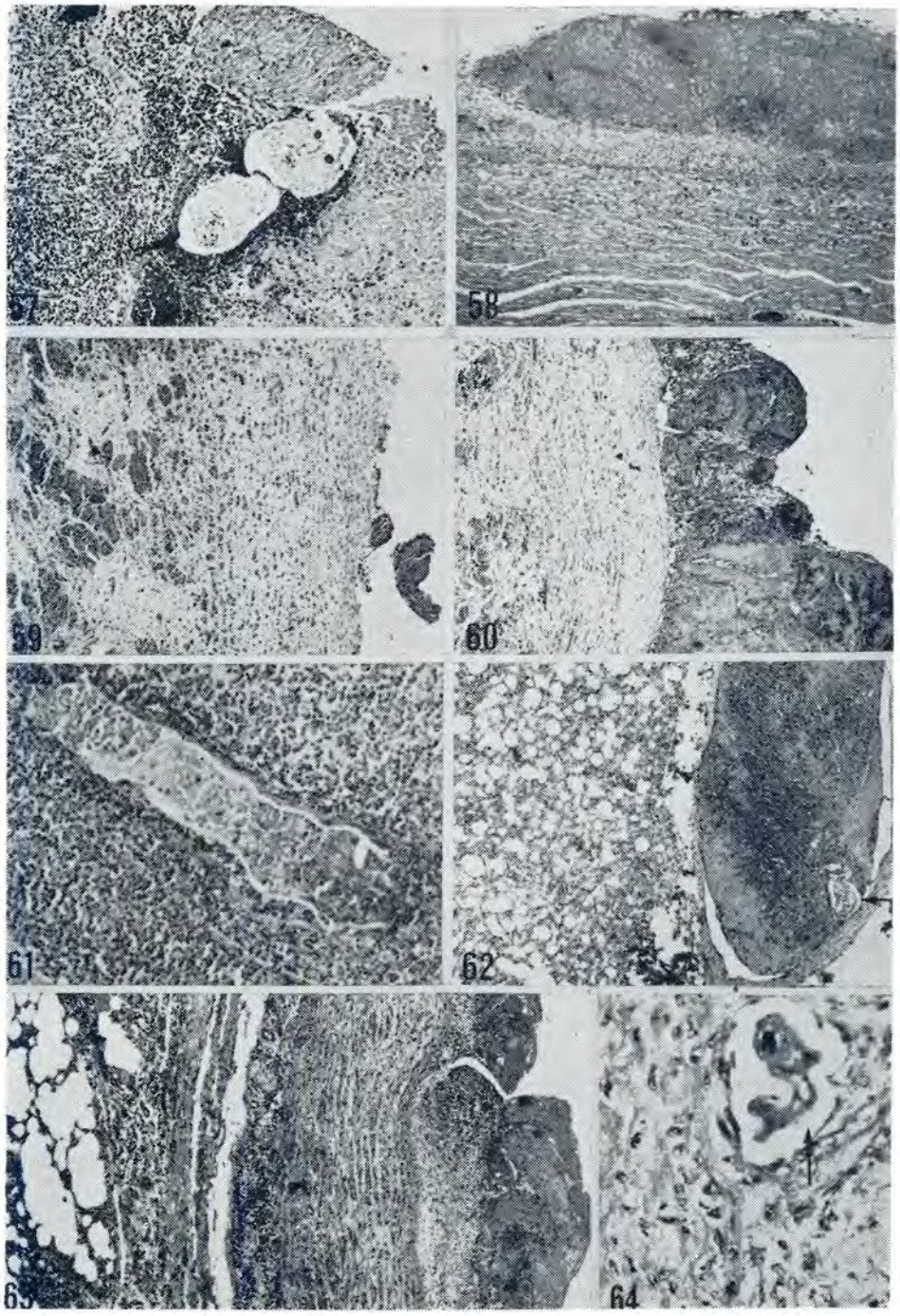

PLATE 8.—57. Photomicrograph showing marked endocarditis containing an entrapped larva and adjoining myocarditis. HE × 75
58. Fibrinoid endocarditis. HE × 30. 59. Pulmonary artery with very mild fibrinoid thrombo-endarteritis. HE × 30
60. Marked thrombosis of the pulmonary artery. HE × 30. 61. Lung: Eosinophil reaction surrounding a dead larva. HE × 75. 62. Lung: A larva in cross section (arrow) within a thrombus. HE × 35. 63. Lung: Thrombo-endarteritis in an intrapulmonary branch of the pulmonary artery. HE × 35. 64. Entrapped larva in the pulmonary artery surrounded by an intimal reaction. A few spines are present (arrow). HE × 200

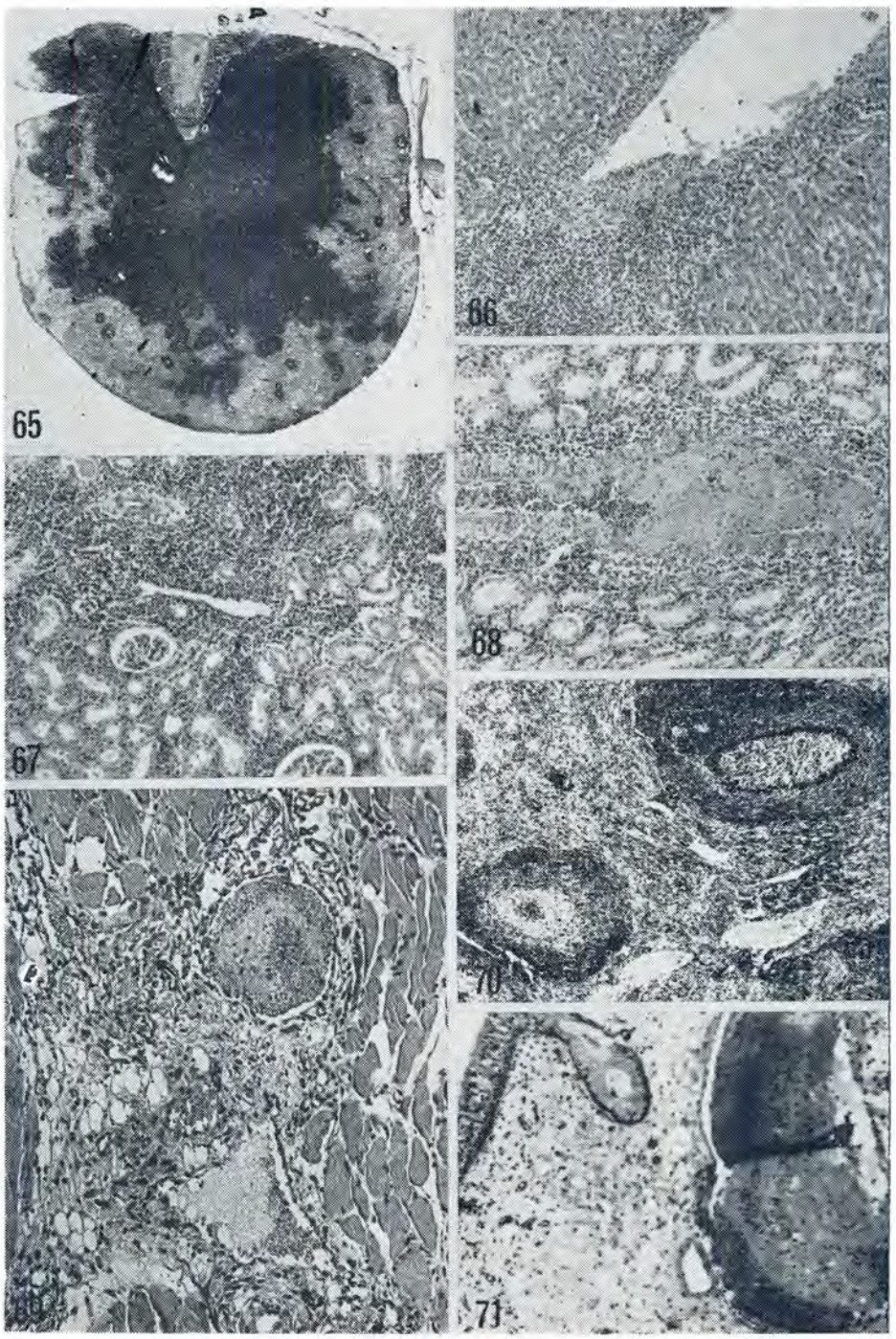

PLATE 9.—65. Hypophysis showing a lighter irregular peripheral zone of necrosis. HE × 9. 66. Liver: Eosinophilic phlebitis of a portal vein with adjoining hepatitis. HE × 75. 67. Kidney: Eosinophilic interstitial nephritis. HE × 75. 68. Kidney: Thrombosis of a renal artery with surrounding mild eosinophilic nephritis. HE × 75. 69. Muscle: Venous thrombosis along with mild haemorrhages and cell infiltration. HE × 95. 70. Spleen: The marginal zone around the splenic corpuscles containing many polymorphonuclear cells, especially eosinophils. HE × 30. 71. Nasal mucosa: Thrombosis in a vein. HE × 75

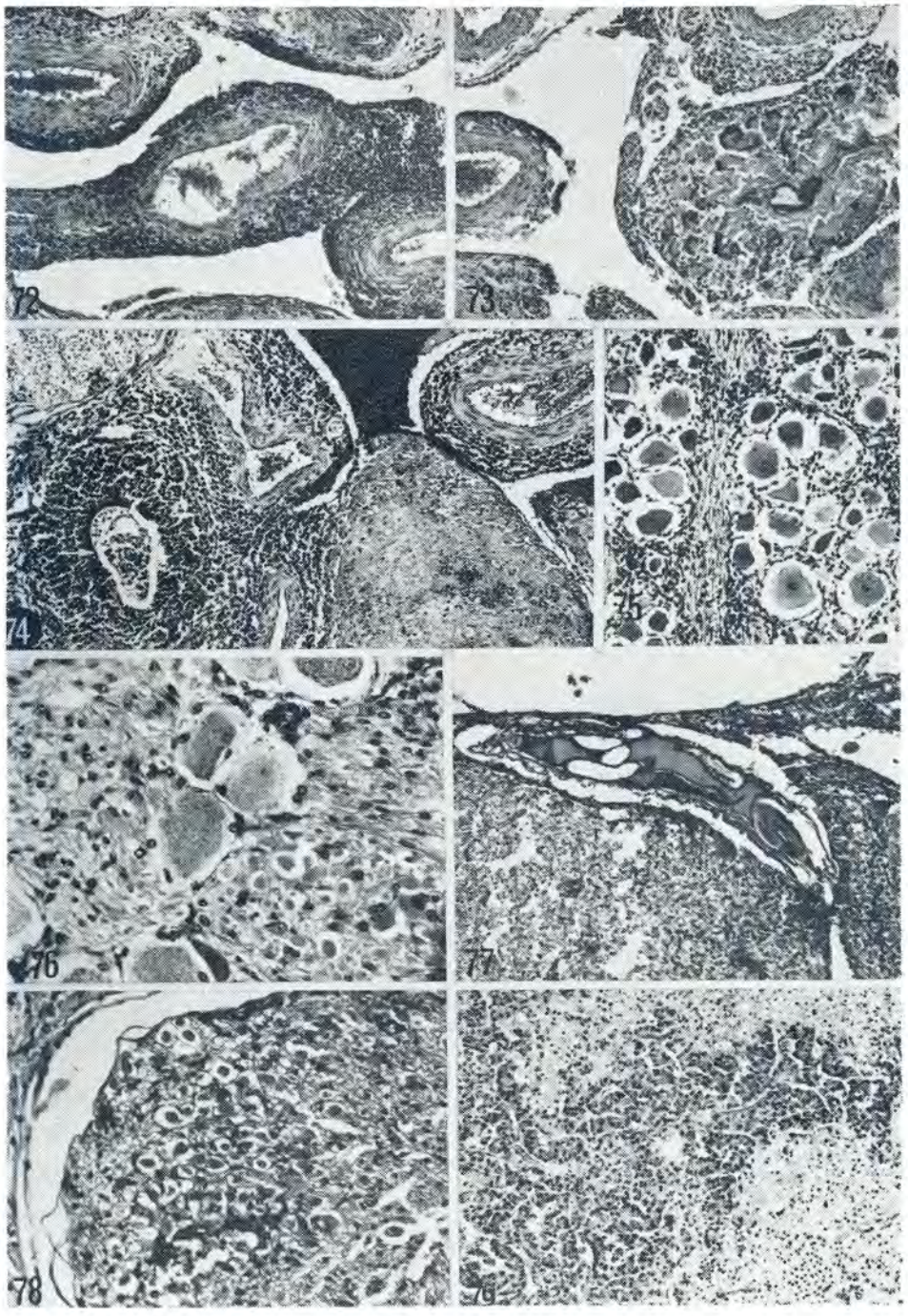

PLATE 10.—72. Rete mirabile cerebri and cavernous sinus: Mixed cell reaction in the walls of the cavernous sinus. HE × 75. 73. Rete mirabile cerebri and cavernous sinus: A parasitic granuloma within the cavernous sinus. HE × 75. 74. Rete mirabile cerebri and cavernous sinus: Phlebitis, thrombosis and an early parasitic granuloma. The larva (arrow) is immediately surrounded by a collar of eosinophilic clubs. HE × 75. 75. Gasserian ganglion: Ganglionitis. HE × 75. 76. Gasserian ganglion: Wallerian degeneration and mild cell reaction. HE × 200. 77. Root of trigeminus nerve: A larva in the act of penetration into the nerve. HE × 75. 78. Root of trigeminus nerve: Wallerian degeneration. HE × 200. 79. Hypophysis: Lighter staining necrotic area of the pituitary illustrated in 65. HE × 75

10(79)]. The adrenal medulla of one sheep was infiltrated with eosinophils and in a few, small haemorrhagic foci were present. A suspected gross lesion in a striated muscle of one animal proved microscopically to consist of haemorrhages, cloudy swelling and Zenker's necrosis together with a diffuse mild mixed cell infiltration and thrombosis of some of the smaller veins [Plate 9(69)]. Most of the gasserian ganglia and the adjoining root of the trigeminal nerve contained small haemorrhages, foci of Wallerian degeneration and mild eosinophil or mixed cell reactions [Plate 10(75, 76 and 78)]. Larvae were found in these regions in 17 per cent of the cases [Plate 10(77)]. The wall of the cavernous sinus was frequently inflamed and thrombosed with accompanying parasitic granulomas [Plate 10(72, 73 and 74)]. Mild purulent rhinitis in association with phlebitis, thrombosis and haemorrhages was present in a third of the nine specimens examined [Plate 9(71)].

In the goat, lesions were found in the blood vessels, eyes, brain, myocardium, optic nerve, hypophysis and cavernous sinus. They were identical to those present in sheep.

Discussion

Gedoelstia larvae were encountered either free within the tissues or in the blood vessels of the head, neck and thorax, the eyes, brain and heart being the organs that were most frequently parasitized. The fundamental primary lesions were thrombovasculitis and vasculitis which gave rise to the more serious and even fatal lesions such as infarction of the brain and myocardium and to glaucoma. Encephalomalacia was the most significant brain lesion and was due either to arterial or venous thrombosis or to parasitic embolism. Haemorrhages, oedema, degeneration and inflammatory reactions in the various layers of the eyeball and periocular tissues were common findings and corneal ulcerations and ruptures were frequent sequelae. The cavernous sinus, optic fasciculus, trigeminus nerve and pulmonary artery were also commonly parasitized, but the lungs, kidneys, liver, adrenals, nasal mucosa and muscles were either occasionally or rarely involved.

These findings indicate that the larvae, after deposition in the eyes, either migrate actively through the ocular and periocular tissues or enter blood vessels in these regions from whence they are passively transported to the heart and brain. It is also evident from the results obtained from the two experimental cases that the heart and brain may be reached within five days via the jugular veins and carotid arteries respectively. The commoner direct route to the brain, however, is apparently via the ophthalmic emissary vein and the cavernous sinus. The lesions around the optic fasciculus indicate that this route is also followed by the larvae. These observations on the migratory pattern confirm those made previously on both natural and aberrant hosts (Basson, 1962c; Basson, 1966a). The ultimate destination in the natural host is the nasal cavity or paranasal sinuses and it was stated in these earlier studies that the larva apparently does not complete its life cycle in sheep. The present histopathological studies, however, indicate that some of them at least reach the nasal mucosa via the bloodstream. The trigeminal nerve and gasserian ganglion are probably injured during their migration via the cavernous sinuses towards the brain, but the possibility that some of the larvae follow certain nerves towards the nasal mucosa cannot be disregarded entirely. Only a small number of the parasites seemed to reach the abdominal organs. This was well illustrated by the low percentage of organs affected in this area. In both the present and in previous studies the brain was found to be more frequently affected than the myocardium and the pulmonary artery. It appears therefore that the migratory route towards the brain is the commoner one.

In conclusion, the author wishes to introduce the generic name, gedoelstiasis, for this specific type of myiasis in both natural and unnatural hosts instead of the more cumbersome "specific oculo-vascular myiasis."

Summary

The histopathological changes in 51 sheep and one goat which showed evidence of myiasis by the first stage larvae of *G. hässleri*, *G. cristata* or *Gedoelstia* hybrids, are described. The fundamental and most significant lesion was thrombovasculitis which caused infarcts in several organs, particularly in the myocardium and brain. In this respect the brain in gedoelstiasis was a good model for studying encephalomalacia. The migratory routes were found to be bloodvascular, mainly from the eyes to the heart, lungs and brain, along the optic fasciculus to the brain and through the trigeminal nerve. The direction of migration along the latter route has not been clarified. Extravascular and extraneural migration was also evident.

The generic name gedoelstiasis is suggested for this disease.

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