THE PATHOLOGY OF INFECTIOUS SEROSITIS OF DUCKS *

E. DOUGHERTY, 3RD, V.M.D.; L. Z. SAUNDERS, D.V.M., and E. H. PARSONS, JR., B.S.
From the New York State Veterinary College Duck Disease Research Laboratory, Eastport, N.Y. (Dr. Dougherty and Mr. Parsons), and the Brookhaven National Laboratory, Upton, N.Y. (Dr. Saunders)

During the past 4 years, 7,155 ducks have been necropsied at the Duck Disease Research Laboratory. Of these, 2,216 were affected with a pathologic entity which we have designated infectious serositis. This disease represented 55 per cent of the accessions of birds 2 to 8 weeks of age. It is economically the most serious disease affecting domestic ducks in the United States. Several duck ranchers on Long Island were questioned in an attempt to establish when the disease was first observed. Some could recall its occurrence on their farms over a decade ago, but these recollections were based only on clinical symptoms.

Two diseases are recorded in the literature which are clinically indistinguishable from infectious serositis. The first is "anatipestifer infection" (new duck disease), described by Hendrickson and Hilbert[1] from Long Island in 1932. The second is the "duck septicemia" described by Graham, Brandly, and Dunlap[2] from Illinois in 1938. Both of these may be the same disease described here; however, since neither report contains a detailed description of the lesions, anatomical comparisons cannot be made.

CLINICAL FEATURES

During the growing season, February to November, most Long Island duck farms have a series of flocks ranging from 1 through 8 weeks of age on the premises. Infectious serositis usually breaks out first in one of the older groups (6 to 8 weeks of age). The disease eventually descends through the various flocks until birds about 10 days of age are affected. The assembly line system of moving the ducks every 4 to 7 days into the quarters vacated by the next older group accelerates the spread of the infection.

A mild cough accompanying white or greenish white diarrheal discharge are the first symptoms. The feathers around the vent are frequently stained green. Older birds develop a slow tremor of the head, locomotor incoordination, and finally lie on their backs and paddle

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convulsively with their legs. Many of the birds so affected die of inanition. The course of the disease in a flock may vary from 1 to 4 weeks. Individual birds die in 1 to 7 days after symptoms appear. In flocks 2 weeks of age birds die after only 1 or 2 days of illness. In older flocks the birds may be ill 6 to 7 days before dying.

Morbidity as indicated by anorexia and diarrhea is very high in flocks affected with serositis, often approaching 100 per cent. Mortality is highest in younger flocks (under 4 weeks of age). Mortality rates in these age groups vary from 20 to 65 per cent. In older ducks mortality is usually under 20 per cent.

Adverse environmental conditions often predispose to outbreaks of the disease. Severe climatic changes or sea tides backing up into the ducks' fresh water streams have been noted to precede some of the outbreaks.

Pathologic Findings

The distribution of gross lesions in 100 consecutive necropsies on birds affected with serositis is recorded in Table I. The following is a description of the pathologic changes in each organ.

Table I

Distribution of Gross Lesions in 100 Ducklings (74 Females and 26 Males)

Splenomegaly	89
Air sac inflammation	87
Pericarditis	85
Perihepatitis	75
Salpingitis	24*

* Represents 32.4% of females.

Heart. Fibrinous pericarditis was one of the more common and striking lesions (Fig. 1). The pericardium was often adherent to the heart and in some cases also to the thoracic wall. Histologically, the inflammation in most cases did not extend beyond the epicardium (Fig. 5). In some cases the superficial portion of the myocardium was involved also, and inflammatory cells infiltrated the spaces between the myocardial fibrils (Fig. 6). The cellular infiltrate consisted predominantly of large mononuclear cells, with a small percentage of heterophilic leukocytes.

Liver. The liver was enlarged and covered by a yellowish white membrane (Fig. 4). Histologically, this membrane contained fibrin and numerous inflammatory cells, similar to those in the pericardium (Fig. 7). In addition there were numerous fibroblasts, which tended to organize the exudate early in the course of the disease. At necropsy the exudate was often sufficiently organized to slip off as a membranous cover when the liver was handled. The liver was sometimes affected by periportal infiltration of inflammatory cells. The blood vessels were congested.

Spleen. The spleen was enlarged to three to five times the normal volume. Its color was pale and the surface mottled, giving it a lobular appearance somewhat resembling a pig's liver (Fig. 3). Fibrin was often present on the serous surface.

Kidneys. The blood vessels in the kidneys were unduly prominent (Fig. 2). Fibrinous exudate was present on the ventral surface. Histologically, passive congestion was the only change seen. All of the veins were distended, the congestion being particularly prominent in the smaller vessels.

Oviducts. One or both oviducts were distended throughout their length by caseous exudate in about one third of the females (Fig. 9). Histologically, the submucosa was infiltrated by inflammatory cells which extended into the mucosal folds (Fig. 10). The lumen was distended by exudate which was predominantly cellular and contained little fibrin. The cells were almost all large mononuclear phagocytes. The salpingitis was considered a part of the pathologic picture, because pericardial or hepatic lesions were present in all birds with salpingitis. Salpingitis without accompanying lesions in other organs has not been found in this laboratory in young ducks.

Alimentary Canal. The blood vessels in the serosa of the intestine were unduly prominent. Mucopurulent exudate sometimes was present in the lumen of the intestine.

Nasal Sinuses. Mucopurulent exudate often was present in the nasal sinuses.

Air Sacs. Grossly, the air sac membranes were thickened and opaque. Histologically, there was distention by fibrinous and cellular exudate to approximately twice the normal thickness (Fig. 8). The inflammatory cells were predominantly large mononuclear phagocytes. In birds in which the disease had apparently run a less acute course, some of the mononuclear cells had coalesced to form giant cells. Fibroblastic proliferation also was present in the more chronic cases. The gross appearance was similar to that described in chronic respiratory disease of chickens; however, the lymphofollicular lesions of chronic respiratory disease were not seen.[3]

Lungs. No significant changes were found in the lung sections, nor were there any in the trachea and bronchi. The inflammation of the air sacs did not extend to the pulmonary parenchyma.

Central Nervous System. Fibrinous cerebrospinal meningitis was present in all birds with nervous symptoms, and in some which died rapidly without well defined symptoms (Figs. 11 and 12). In a few specimens the exudate in the leptomeninges was thick enough to be

recognizable grossly as soon as the dura mater was incised. The meningitis extended from the forebrain to the end of the spinal cord. The inflammatory cells were studied in smears of cerebrospinal fluid taken at necropsy from freshly killed birds, as well as in tissue sections. Large mononuclear phagocytes predominated, but heterophils were always present in numbers estimated at 5 to 25 per cent of the inflammatory cells. Inflammatory changes in the brain were slight, and were seen only in the peripheral tissue adjacent to the meninges, where a few vessels with perivascular cuffing were found (Fig. 13). Pyknosis of some of the motor neurons was seen in several spinal cords (Fig. 14). No neuronophagia or myelin sheath degeneration was seen in the central nervous system. Inflammatory changes were present in a number of lumbar spinal nerve roots, but the inflammation did not extend to the sciatic nerves.

The extensive meningitis in our birds distinguishes serositis from the virus encephalitis of wild ducks described by Rosenow.[4] In addition, none of the visceral lesions described here were present in the wild ducks.

Peripheral Blood. Wright-stained blood smears were examined from 120 birds and spleen impression smears from over 1,000 birds. No duck plasmodium, leukocytozoon, or other blood-cell parasite was ever encountered.

Interpretation of Lesions

The pathologic picture was one of a generalized fibrinous inflammation of the serous membranes. This attribute was shared alike by the meningeal, pleural, pericardial, and peritoneal surfaces. The inflammation showed little or no tendency to extend from the membranes to the underlying parenchymatous organs. It is likely that the nervous symptoms were referable to the lesions in the central nervous system. The congestion observed in the liver, spleen, intestine, and kidneys was probably due to cardiac insufficiency.

Although there were a few differences, the pathologic picture as a whole was similar in many respects to that of sporadic bovine encephalitis.[5]

Differential Diagnosis

Infectious serositis can be distinguished from other diseases of ducks by the following features:

Virus Hepatitis of Ducks. Petechial and ecchymotic hemorrhages on the surface of the liver are characteristic of virus hepatitis. These were not seen in infectious serositis. On the other hand, the lesions of infectious serositis were not seen in uncomplicated hepatitis.

Fowl Cholera. Differentiation between the peritoneal form of cholera and infectious serositis must be made by cultural identification of the *Pasteurella multocida* organism; however, the serosal exudate of fowl cholera is yellow, while that of serositis is yellowish white. The petechial hemorrhages on the epicardium and the focal necrosis of the liver which characterize acute fowl cholera were not seen in infectious serositis.

Duck Plague. In duck plague, as in serositis, there is pericarditis, peritonitis, and salpingitis. However, the widespread hemorrhages described in plague have not been seen in serositis, and there is no mention of neurologic lesions in the description of plague.[6]

Discussion

Because of the nature of the lesions, which affected chiefly the serous membranes, the name serositis was chosen for this disease. Hjäre and Wramby[7] have previously applied this term to a disease of swine with similar widespread exudative changes. In the absence of etiologic information we are naming the disease on the basis of its morbid anatomical features.

We have been able to transmit the disease to ducks by the administration of suspensions of ground spleen, liver, and serosal exudates, thus establishing its infectious nature. Intratracheal and intraperitoneal inoculations have been successful in 95 of 120 attempts. The etiologic agent has not yet been determined. The non-suppurative character of the inflammation is suggestive of a non-bacterial etiology. The similarity of the pathologic picture to that of sporadic bovine encephalitis suggests that a psittacoid organism may be involved.

Summary

Infectious serositis of ducks is an epizootic disease with a high morbidity and a mortality of 5 to 60 per cent. It is characterized anatomically by cerebrospinal meningitis, pericarditis, and perihepatitis. The infection can be transmitted by inoculation of diseased tissue, but the nature of the etiologic agent is not known.

We are indebted to Robert F. Smith for the photographic illustrations.

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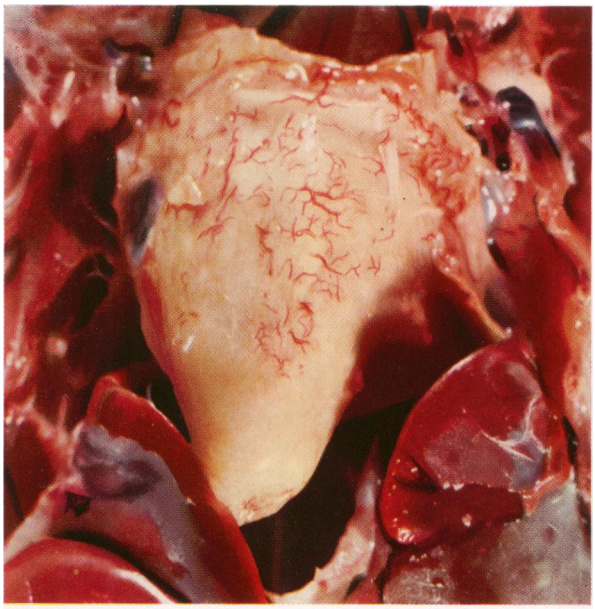
LEGENDS FOR FIGURES

FIG. 1. Fibrinous pericarditis in a 3-weeks-old duckling.

FIG. 2. Ventral surface of the kidneys, with passive congestion.

FIG. 3. Dorsal view of the spleen with mottling visible through the fibrin on the serous surface.

FIG. 4. Fibrinous perihepatitis and pericarditis.



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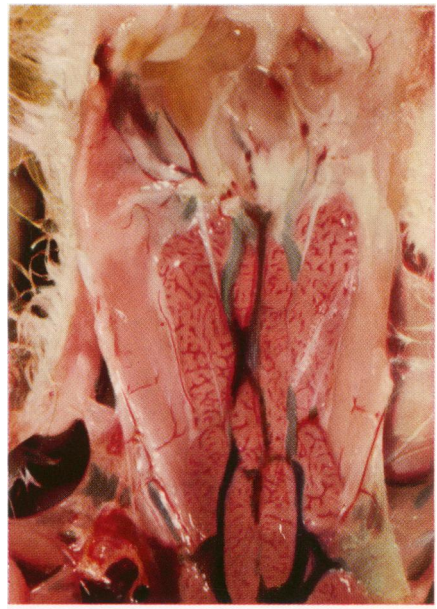

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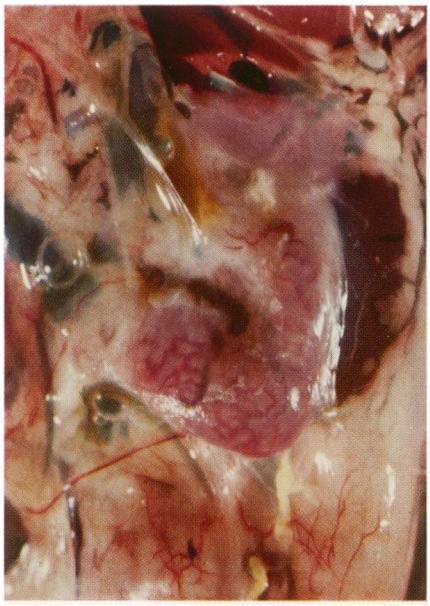

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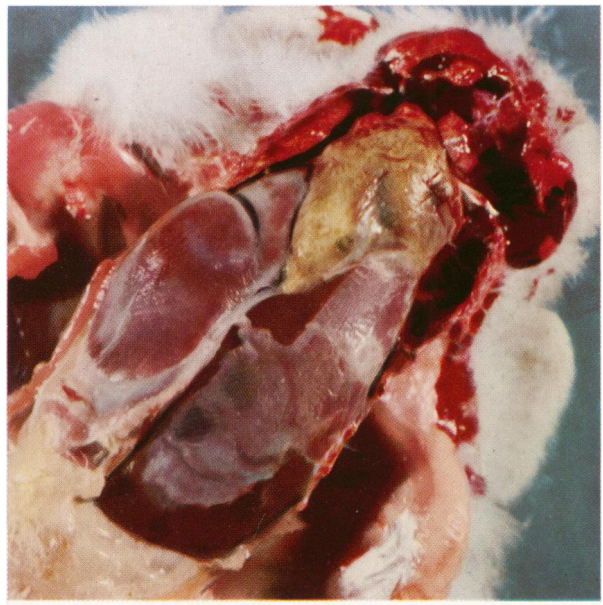

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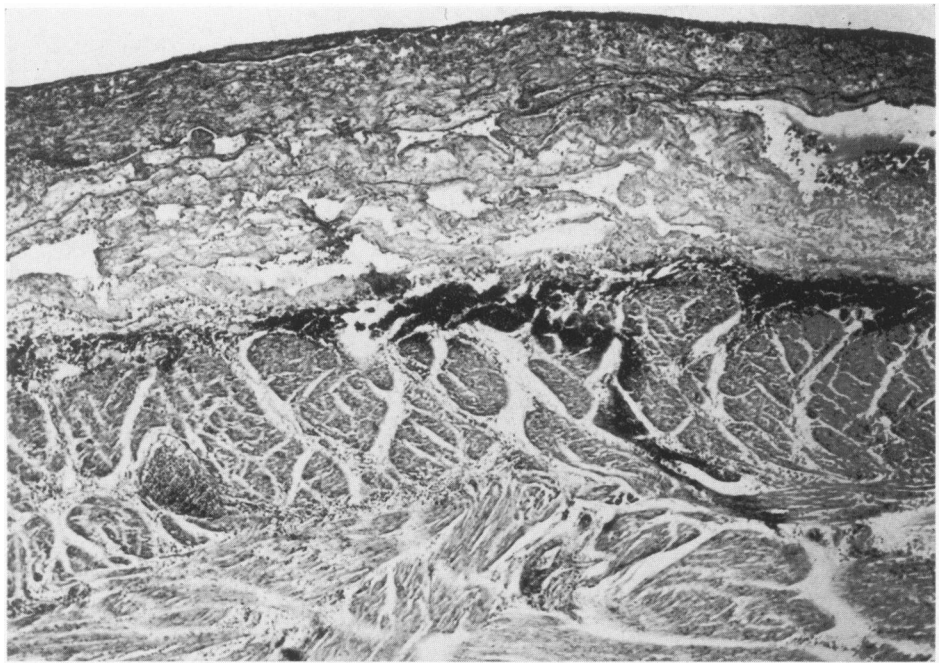
FIG. 5. Pericarditis. A thick layer of exudate on the epicardial surface. × 50. This and all subsequent sections were stained with hematoxylin and eosin.

FIG. 6. Myocarditis. Inflammatory cells and edema between myocardial fibers. × 220.



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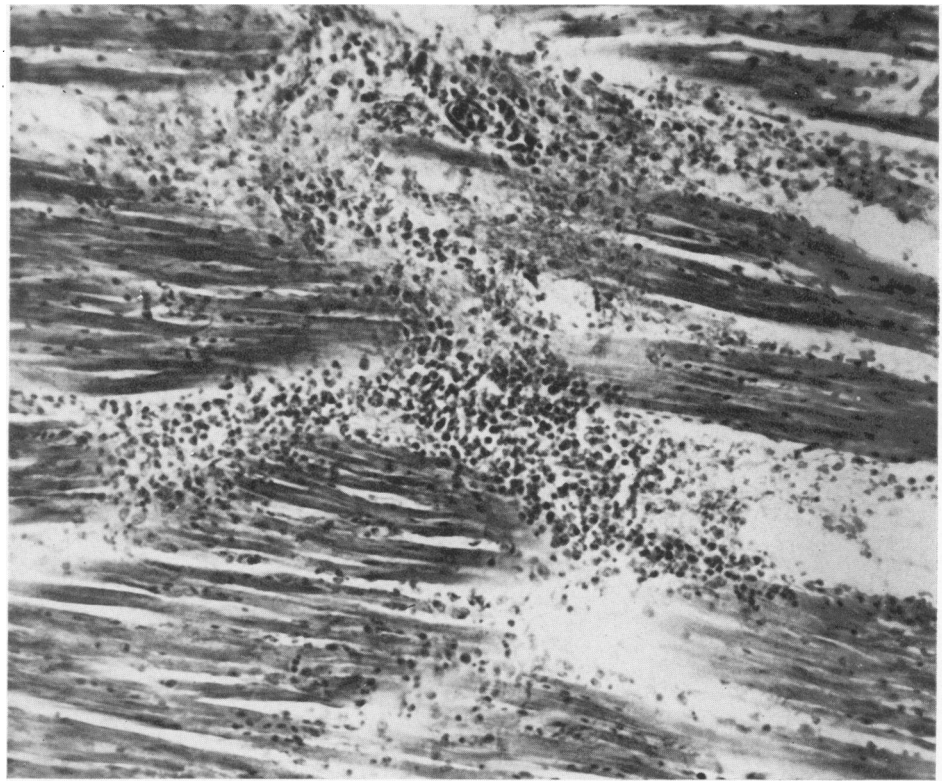

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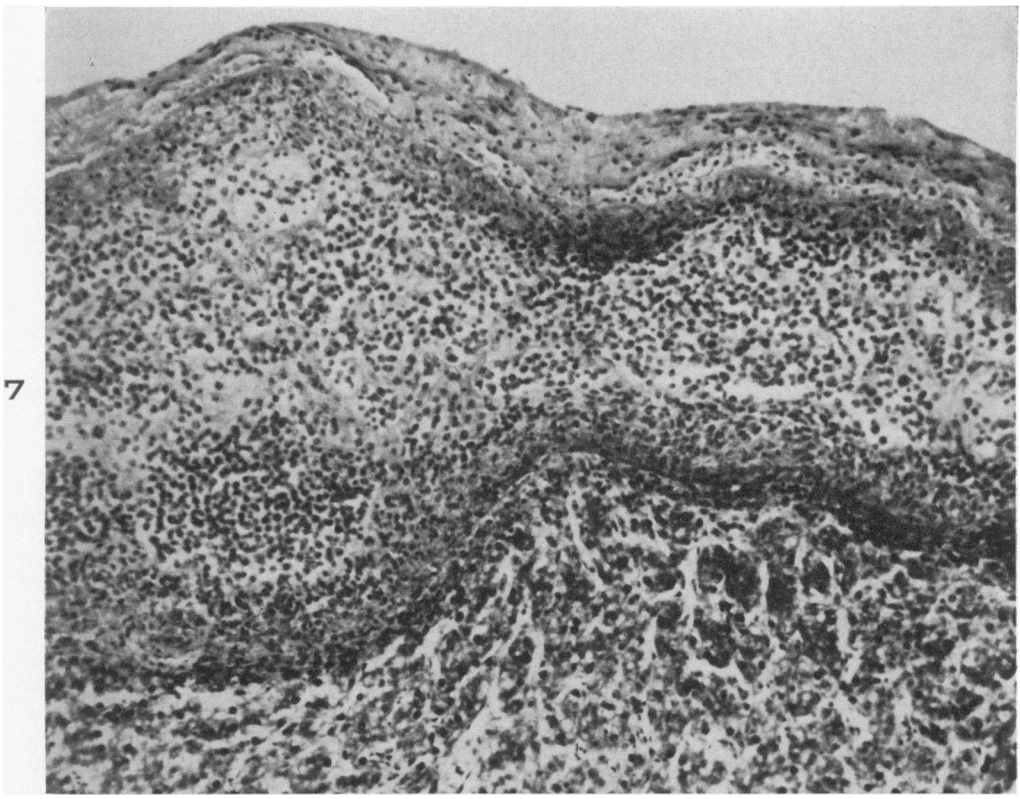
FIG. 7. Liver with thick layer of exudate on its peritoneal surface. × 220.

FIG. 8. Air sac thickened by infiltration with fibrinous and cellular exudate. × 220.

FIG. 9. Oviduct containing caseous exudate. × 18.

FIG. 10. Salpingitis. Infiltration of the submucosa and mucosal folds with inflammatory cells. × 185.



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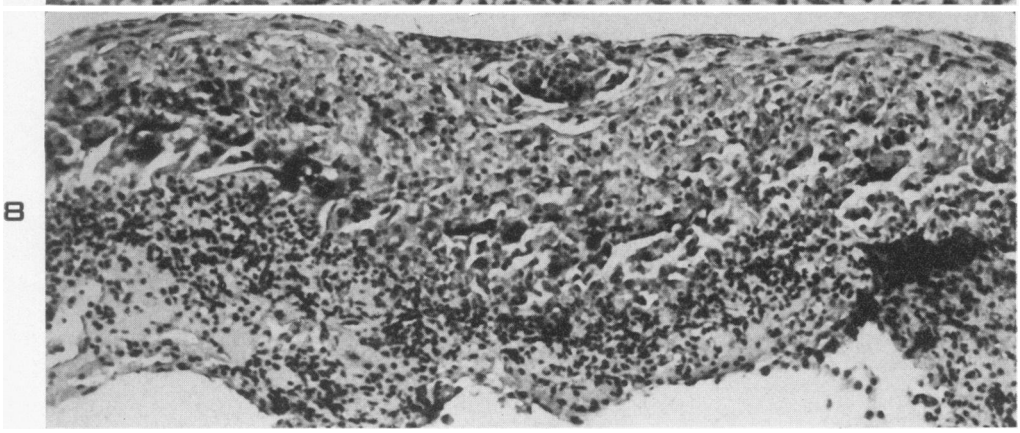

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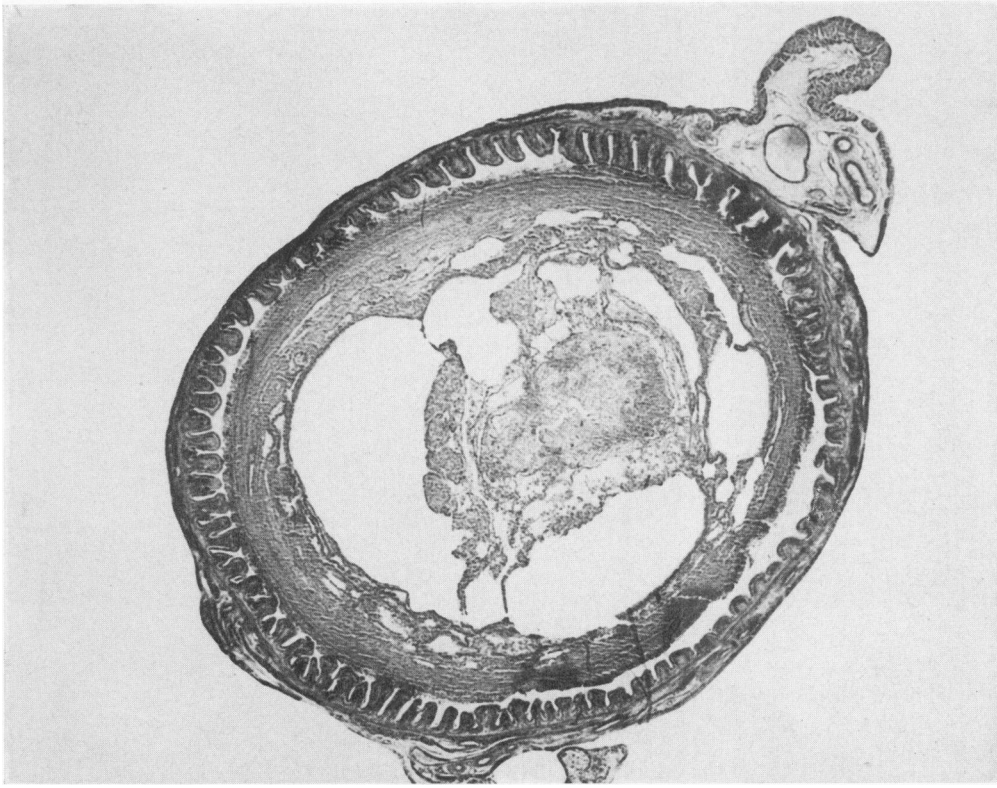

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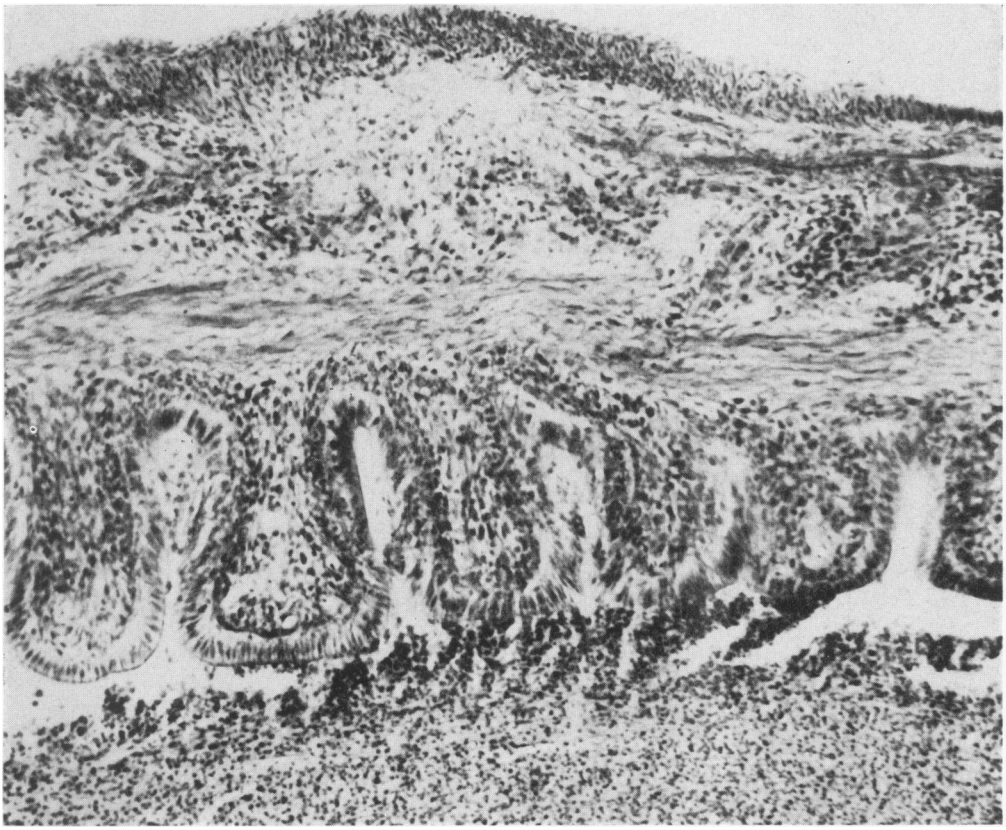

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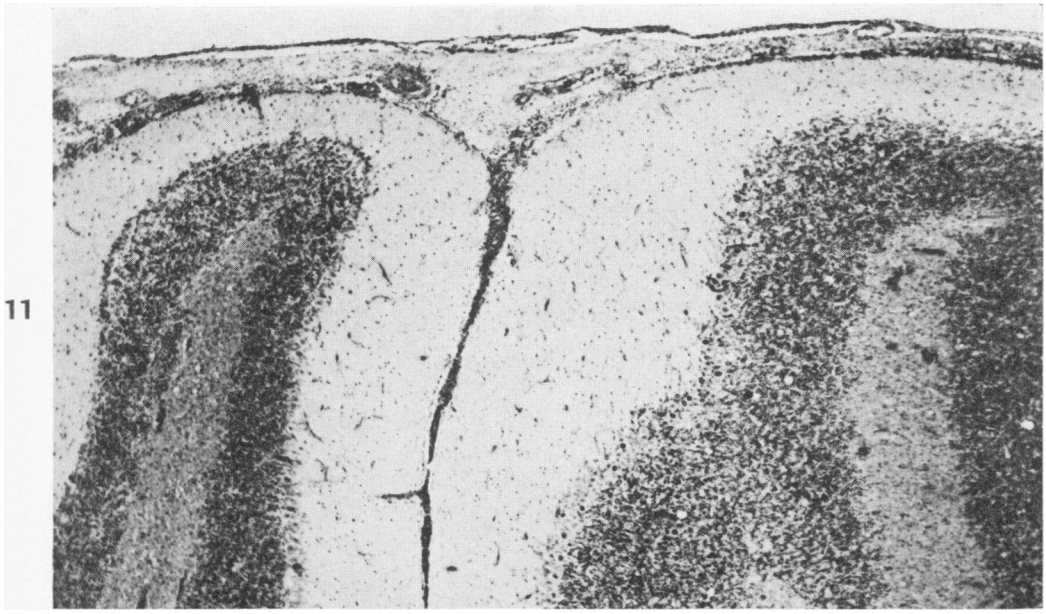


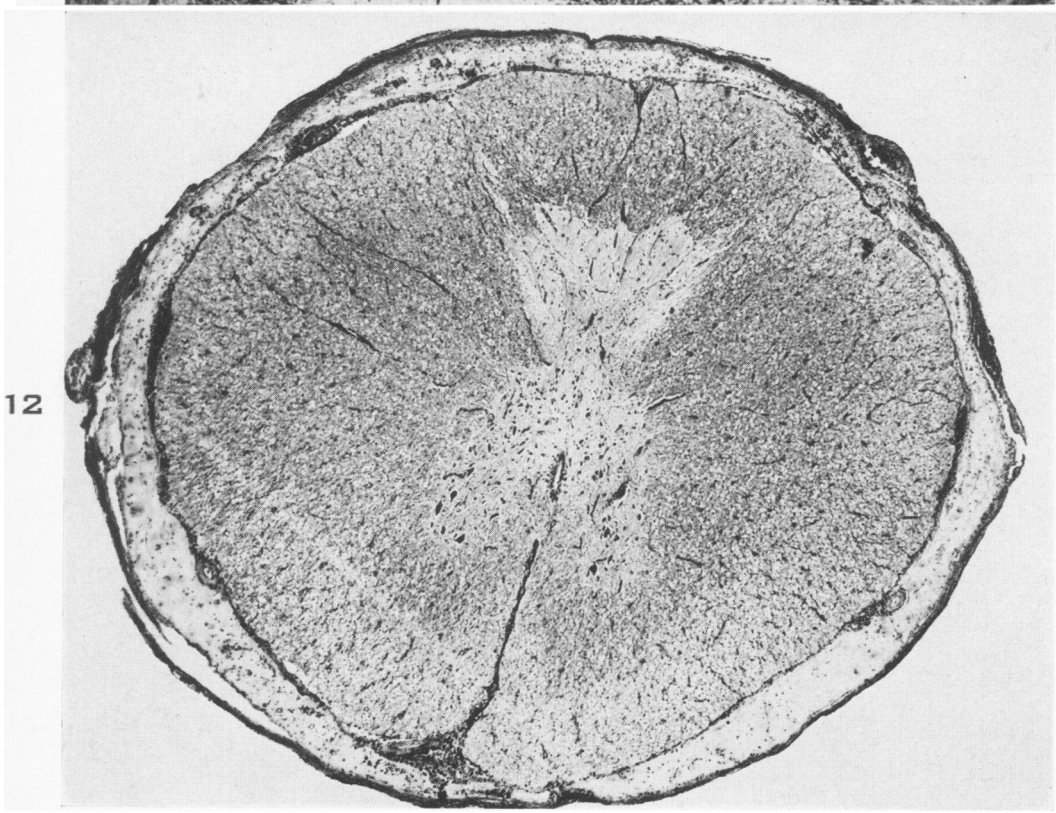
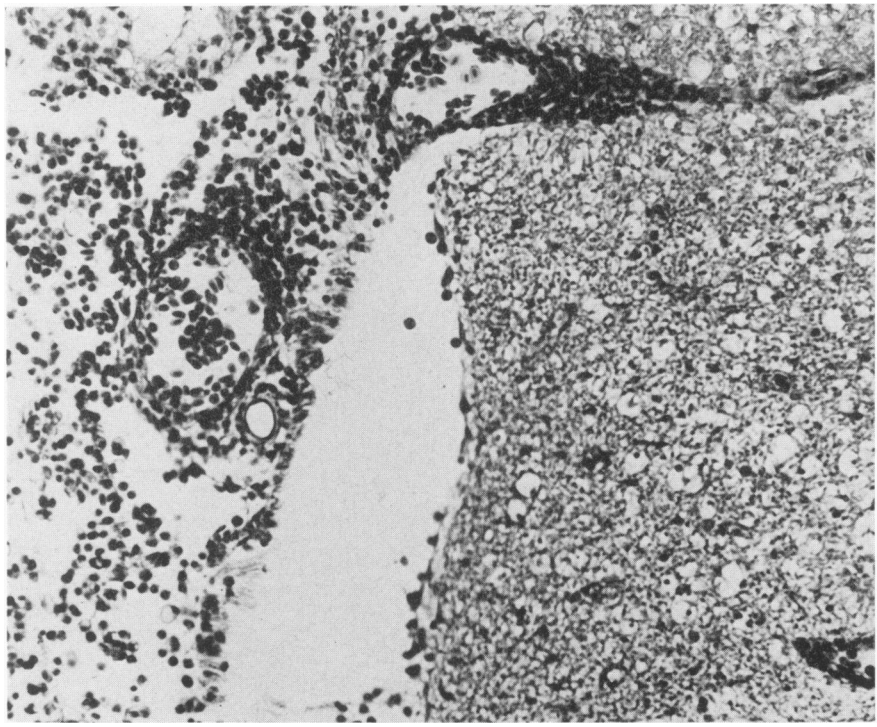


FIG. 11. Cerebellar leptomeningitis. × 45.

FIG. 12. Spinal leptomeningitis. The subarachnoid space distended by fibrinous exudate. × 25.



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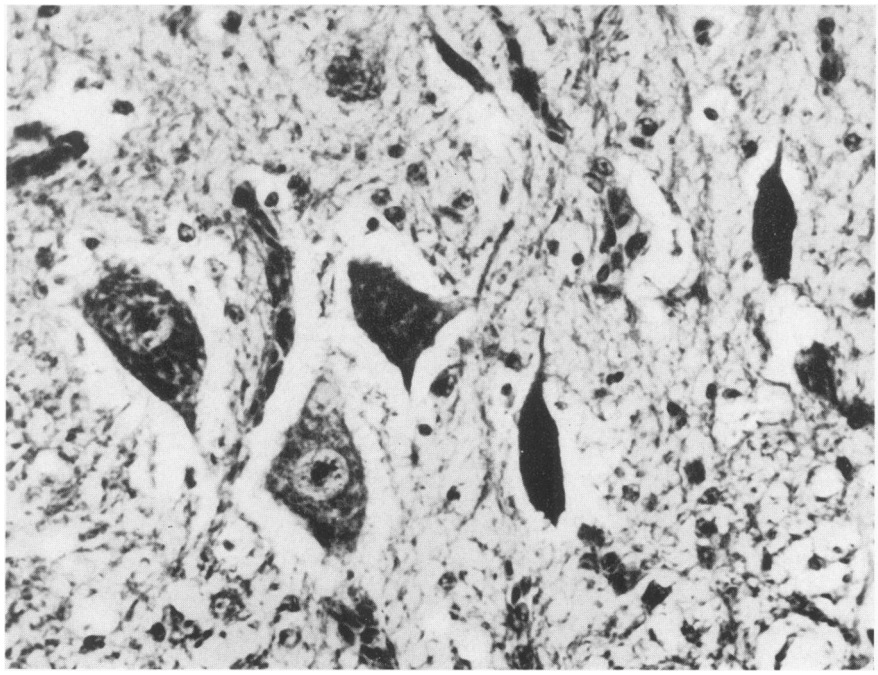

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FIG. 13. Inflammatory cells in leptomeninges of the medulla oblongata and perivascular cuffing of veins. × 220.

FIG. 14. Spinal cord with pyknosis of motor neurons. × 400.