ACUTE NECROTIZING ENCEPHALOMYELOPATHY FOLLOWING IODIZED-OIL-VENTRICULOGRAPHY *

by

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(Received for publication Oct. 1, 1958)

Following the introduction of iodized oil by SICARD, PARAF and LAPLANE[25] (1923) as a medium for the visualization of the human cerebral ventricles, BALADO and his co-workers [6,7,8] (1928) recommended the iodized-oil-ventriculography as a valuable and often essential aid in the diagnosis of intracranial tumors. In this country, ASANO[3,4] (1941, 1942) and HOSHINO[17] (1957) in our clinic described the details of the technicalities of the use of iodized oil and the interpretation of the roentgenograms obtained. Nowadays, the role played by the application of iodized-oil-ventriculography in the diagnosis and localization of brain tumors still remains important.

But, because of unfavourable reactions produced, leading to a fatal termination in a few instances, iodized-oil-ventriculography is not always a safe procedure. ARAKI and his associates[1,2] (1956, 1957) reviewed all instances of complications following iodolography observed in our clinic during the period between 1952 and 1956, of which there was a case suggesting acute transverse myelitis. As the case seemed important, we carried out detailed studies of the sections from the central nervous system of this instance. Pathological findings of the spinal cord, characterized by necrosis to a large extent of the cord and great thickening of the walls of intra-and extramedullary blood vessels, corresponded very closely to those of subacute necrotic myelitis described by FOIX and ALAJOUANINE[42] (1926). In addition, there were changes of the cerebral cortex and adjoining white matter which were characterized by the dissemination of small areas of ischemic necrosis.

Necrosis of the spinal cord, which might be referred to the iodized oil injected into the cerebrospinal space, can not gleaned from the literature. It seems worthwhile, therefore, to record this case in detail. Moreover, a survey of this case may throw some light on the problem of the development of spinal necrosis which remains obscure.

REPORT OF CASE

H. S. Male, aged 55. Labourer——married.

Past History.——At the age of 48 years the patient suffered from tuberculosis of the left lung which was treated with artificial pneumothorax during three and a half years. At

* The paper was read in Japanese at the 16th meeting of the Japan Neurosurgical Society, Okayama, October 8-10, 1957.

the age of 53 years he had severe headache. Otological examination revealed a small tumor in the left tympanic cavity, which was removed. Microscopic examination identified the growth as a lymphangioendothelioma. The patient said that he had never had syphilis.

Family History.——His father had died of cerebral hemorrhage, but there was no other family history of nervous disease.

Present Illness.——On September 18, 1955, the patient complained of pain in the right ear, next day followed by headache in the right half, without apparent reason. And next morning, while he was eating breakfast he noticed some difficulty in swallowing and speaking, which was ascribed to disturbed movement of the tongue. On September 25, there developed severe pain in the back radiating to the right hypochondrial region, which was accompanied by weakness and numbness of both legs. He was admitted to the 1st Surgical Division, Kyoto University Hospital, under the care of Prof. Dr. Araki, on September 29.

Examination.——On admission the patient was well nourished and well developed, weighed 65 kg, and did not appear to be acutely ill. The temperature was 36.6°C, the pulse rate, 75, and the respiratory rate, 18. The blood pressure was 140 systolic and 80 diastolic. On auscultation the breath sounds were rough in the lower half of the left lung. The heart and abdominal organs were essentially normal. No deformity or tenderness of the spine could be found. The pupils were equal-sized, regularly circular in outline, and both reacted normally to light and in accommodation. Vision was 1.0 (0.75D 1.2) in both eyes. The visual fields and optic fundi appeared normal. Examination of the cranial nerves revealed slight but obvious impairment of the right hypoglossal nerve, as manifested by slurred speech. When the tongue was protruded there was deviation to the right side, with curving of the tip to the left. Little wrinkles, but no fibrillary tremors were noted. His speech was slow, but not scanning. Sensory examination revealed hyperesthesia over the distribution of the sixth to ninth thoracic segments, especially on the right, and hypesthesia over the posterior surface of both legs, the right being more insensitive than the left. Muscular power in the lower extremities was slightly deficient in general, but there was no localized muscular wasting or fibrillary twitching. In spite of his complaint, gait was not so obviously impaired in ordinary walking, except that he held the legs very tense to protect himself from the backache which motion elicited. Co-ordination tests were well carried out, except for slight ataxia in performing the heel-knee test on the right side. The ROMBERG sign was negative and the posture was normal. The abdominal reflexes were either absent or feeble, and the cremasteric reflexes were unobtainable. All tendon reflexes were normal, and pathologic reflexes were not elicited. He was free from urinary and bowel disturbance.

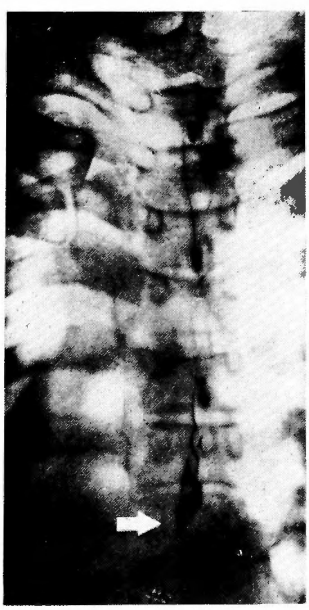


Fig. 1 Myelogram showing the block at the level of the eighth dorsal vertebra.

Laboratory Data.——Urinalysis showed nothing significant. The red cell count was 3,640,000 per cmm with 88% of hemoglobin (SAHLI). The white cell count was 4,700 per cmm, with a differential count of 68% polymorphnuclear cells, 29% lymphocytes, 2% eosinophils and 1% monocyte. The sedimentation rate was 31 mm per hour (WESTERGREN). Lumbar puncture revealed a clear and colorless

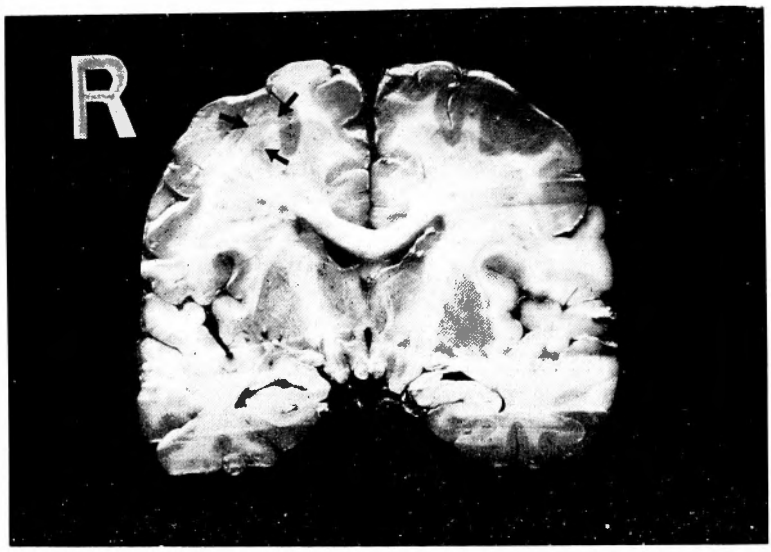


Fig. 2 Photograph of coronal section through the corpus mammillare. The superior frontal gyrus in the right hemisphere contains ring-shaped stripes which, stained for myelin, are shown at higher magnification in Fig. 5.

fluid, under an initial pressure of 170 mm of water with the patient in a horizontal position. After removal of 2.0 cc of fluid the pressure fell to 90 mm. There was no rise on jugular pressure, evidence of subarachnoid block. The PANDY reaction was positive. There were 22 cells per cmm, with lymphocytes predominating. The WASSERMANN reaction was negative for both the blood and the spinal fluid. Flat roentgenogram of the skull revealed no abnormalities. Roentgenogram of the chest was suggestive of a diffuse pleural adhesion over the lower half of the left lung. The electrocardiogram was normal.

Course.——Involvement of the hypoglossal nerve directed our attention to a tumor of the posterior cranial fossa. Because of the uncertainty of diagnosis, however, resort was made to iodized-oil-ventriculography. On November 5, on the sixth day in the hospital, a ventricular puncture was made and 2.0 cc of 40 per cent Moljodol (iodized poppy seed oil made in Japan) was injected into the right lateral ventricle, and the patient was examined fluoroscopically on the tilting table. X-ray showed the oil to pass freely and the ventricles to be no-

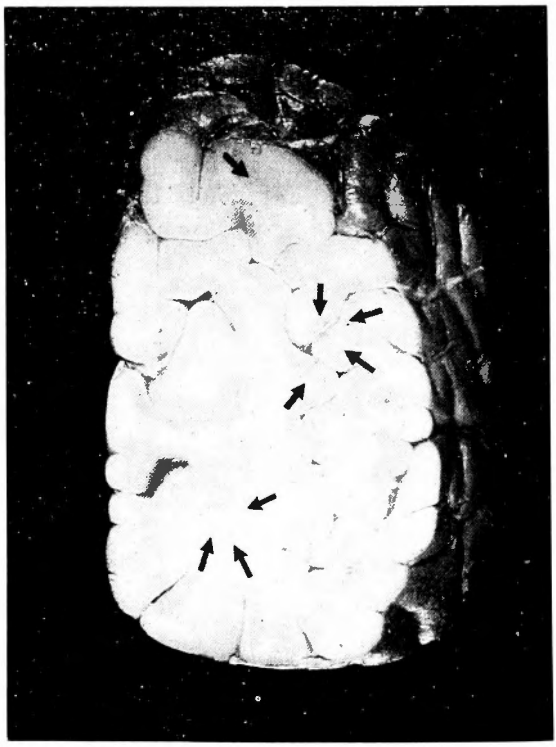
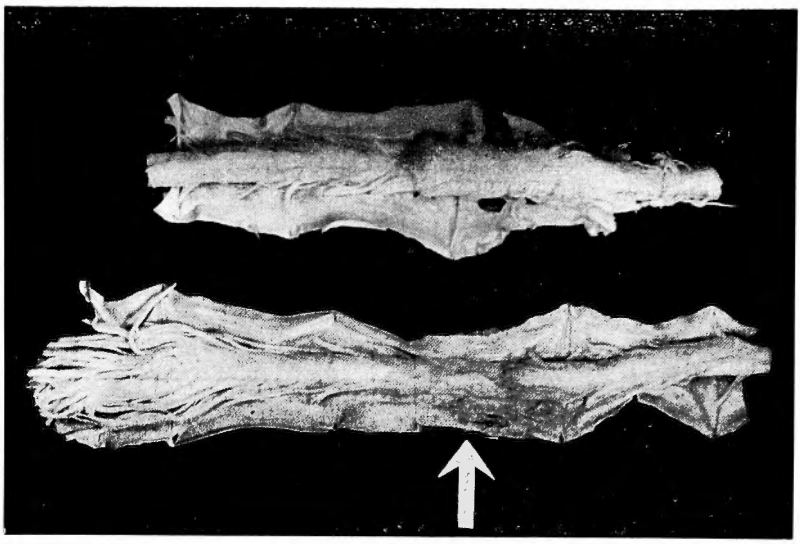
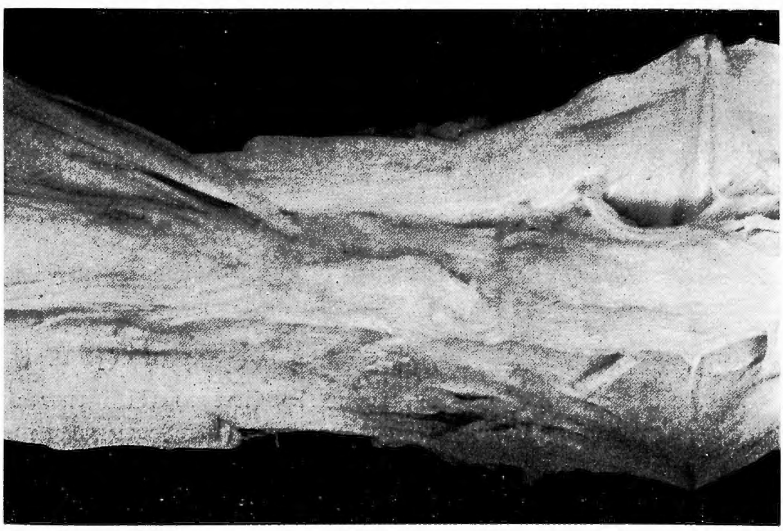


Fig. 3 Photograph of coronal section through the right occipital lobe. Ring-shaped stripes are interspersed in the cerebral cortex and adjoining white matter.



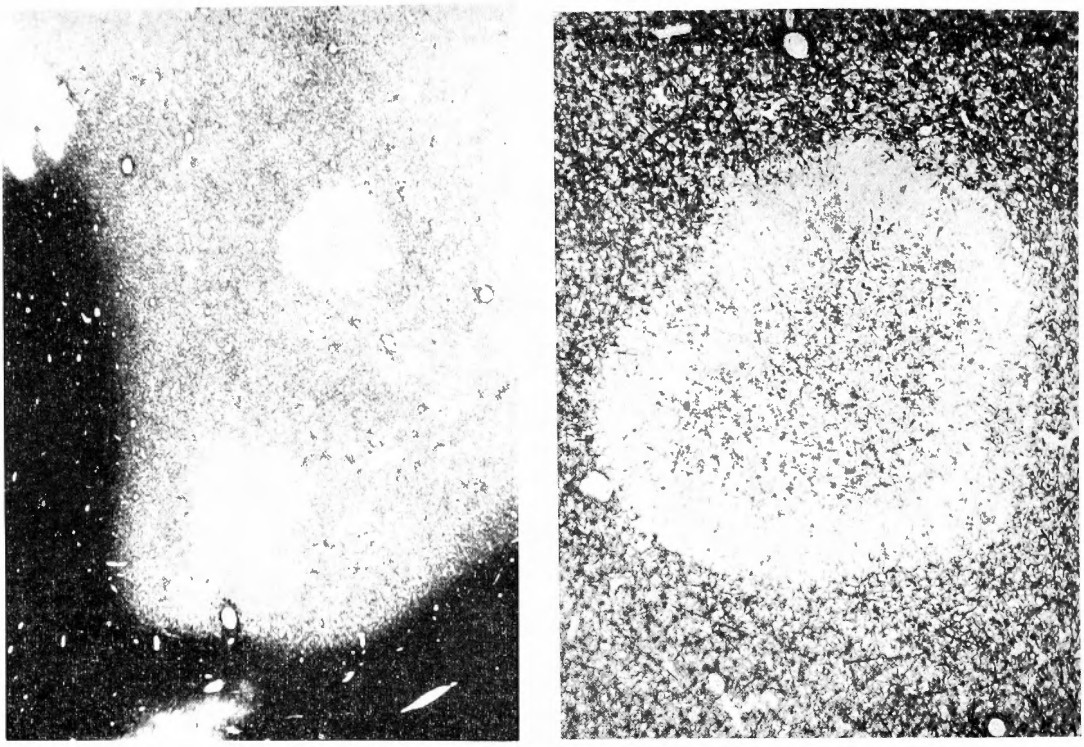
(A)



(B)

Fig. 4 Photograph of the spinal cord with the dura mater opened. The arrow in A indicates the thickened meninges and the flattened cord at the ninth and tenth thoracic segments. B is a higher magnification of this portion.

rmal in size, shape and position, no evidence of a tumor. During examination the patient was apparently in good condition, although he complained sometimes of nausea and headache. About four hours after injection of iodized oil the patient had suddenly a generalized convulsion with loss of consciousness, followed by sleep until the following morning. When he awoke, examination revealed the unexpected occurrence of the pathological condition as follows : 1) complete loss of sensation for all modalities below the level of the fourth thoracic segment and immediately above this a narrow band of hypesthesia, with complete freedom from the troublesome backache which persisted till the previous day, 2) complete flaccid paralysis of both lower extremities with absence of the patellar, ACHILLES tendon and abdominal reflexes bilaterally, 3) a great deal of pain across the shoulders and over the flexor surface



(A) (B)

Fig. 5 Scattered small foci of ischemic necrosis of the right superior frontal gyrus. One involving the subcortical white matter is a patchy lesion, while others involving the cortex are ring-shaped lesions. B is an enlarged view of one of the ring-shaped lesions illustrated in A. SUGAMO myelin stain; ×5 (A), × 100 (B).

of the arms, with the corresponding hyperesthesia in the first to third thoracic dermatomes, 4) loss of sphincteric control of the bowel and bladder. There was no motor impairment of both upper extremities. The temperature was not elevated. The KERNIG sign was negative and rigidity of the neck was not noted. The pulse and respiration remained normal. There was no exacerbation of the slurred speech. A diagnosis of acute transverse myelitis, which was attributed to the iodized oil injected, was made. On the following day there were no changes, except that pain over the arms subsided. Five days after the onset of the accident the temperature became elevated, ranging from 36.7 to 39.3°C until it returned to normal three days later. At that time the hands became flexed and claw-like, and movements became awkward. Thereafter the neurological symptoms remained unchanged. On November 19, two weeks after the onset of the accident, the patient was examined again under the fluoroscope on the tilting table. The iodized oil injected into the right lateral ventricle had descended and remained in the cysterna magna. When the head was lifted upward, the oil flowed freely downward, but did not descend below the lower border of the eighth dorsal vertebra; doubtless the oil was arrested by arachnoidal adhesion (Fig. 1). There were no residuals of iodized oil having descended below this level. The further course was stationary, without remission. There was no ascent or descent of the level of neurological deficit. Dysarthria did not improve. Large bedsores developed and penetrated the soft tissue as far as the sacrum. The temperature increased at times, as the result of an ascending

infection of the urinary tract. The patient became weaker and more helpless. Death occurred on January 22, 1956, approximately three and a half months after the onset of transverse myelitis. No further convulsion occurred until he died. He had never complained of diminution of vision.

Since the onset of transverse myelitis, hydrocortisone was administered in daily dose of from 25 to 150 mg. There was, however, no response to it at all.

Necropsy.——Permission was obtained only for examination of the central nervous system.

Macroscopic Examination.——

Brain: On gross examination the cerebral dura and surface of the brain appeared entirely normal. The arteries at the base of the brain were not pathologic. After fixation in 4.0% formaldehyde the brain was cut with macrotome in parallel frontal planes. The first cut plane corresponded to the most anterior part of the anterior horn of the lateral ventricles. Here the gray and white matter seemed to be normal. The second cut was made through the anterior commissure. Here in the left hemisphere superior temporal gyrus contained a ring-shaped stripe of brownish-gray discoloration encircled by a narrow grayish-white band. The third cut corresponded to the mammillary body. At this level superior frontal gyrus contained a few ring-shaped stripes in the right hemisphere(Fig. 2). The fourth cut corresponded to the middle of the posterior horn of the lateral ventricles. Here postcentral gyrus contained a ring-shaped stripe in the right hemisphere. The fifth cut passed through both occipital lobes immediately behind the posterior end of the lateral ventricles, and on its cut surface the ring-shaped stripes were dispersed in both hemispheres (Fig. 3). The ring-shaped stripes mentioned were scattered mainly in the cerebral cortex and occasionally in the subcortical white matter. The deep white matter widely distant from the cortex was less involved. The basal ganglia and the walls of the ventricles appeared to be entirely intact. The stripes were from 0.1 to 0.2 mm in thickness. The rings were from 0.1 to 0.3 cm in diameter and were rounded, oval or occasionally irregular in shape. The ventricles were symmetrical, undilated, being lined with smooth ependymal membrane. Droplets of iodized oil were seen in the posterior horns of the lateral ventricles. There were no gross pathological changes in the cerebellum or the brain stem.

Fig. 6 Necrotic area of geographical pattern in the cerebral cortex. The changes are much more advanced at the margins of the lesion. Klüver-Barrera stain; × 120.

Spinal Cord: The gross examination of the surface of the spinal cord revealed changes in the lower thoracic portion of the cord. There were adhesions between the dura and the arachnoid membrane at the level of the ninth and tenth thoracic segments, with dusky gray discoloration of their surfaces. The arachnoid was markedly hyperplastic and firmly adherent to the spinal cord (Fig. 4). The spinal cord was found to be caught in the intense adhesive

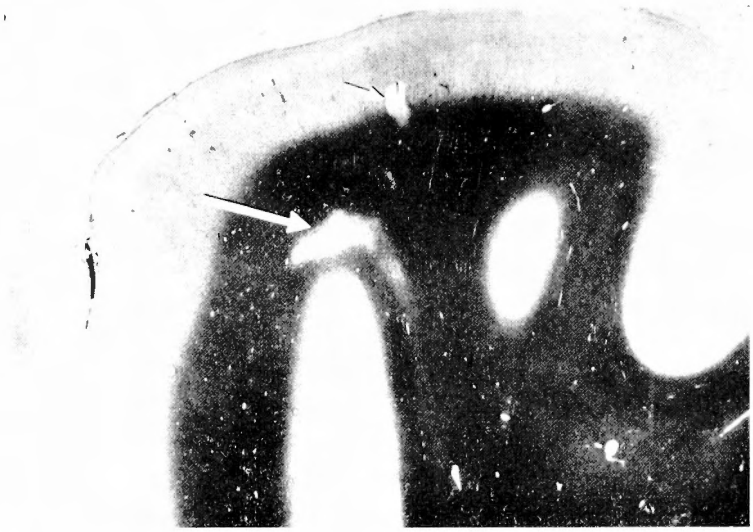


Fig. 7 Small areas of ischemic necrosis of the right postcentral gyrus. The large arrow indicates a sickle-shaped focus separated from the cortical substance by a thin layer of the arcuate fibers. KLÜVER-BARRERA stain; × 4.

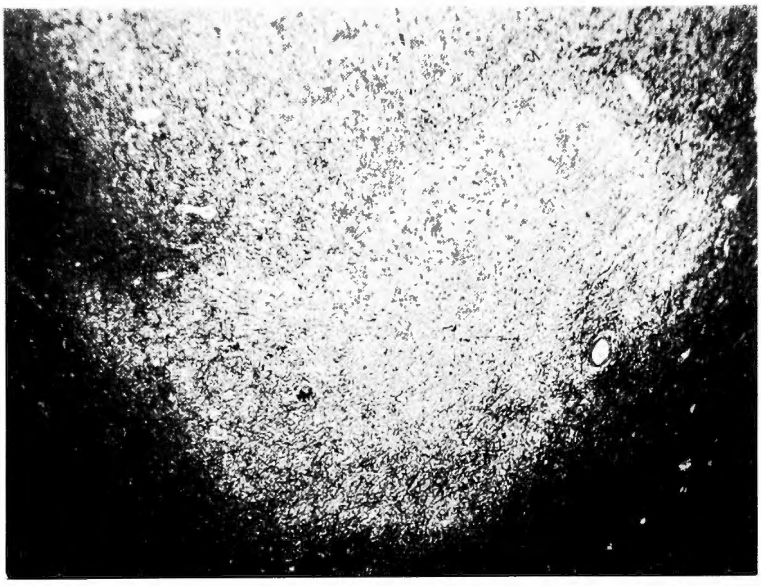
mass which was peeled from the cord with difficulty. Also spinal nerve roots passing through this region were entangled in the dense fibrous tissue. On palpation the spinal cord appeared softened and spongy in this affected region. Droplets of iodized oil were seen enmeshed in the upper part of this adhesion. Considerable congestion and edema on the surface of the cord were not seen. The gross examination of the veins and arteries on the surface of the cord, anteriorly and posteriorly, failed to reveal any sign of thrombosis or hemorrhage. There was no hemangiomatous malformation of the veins. Section after hardening of the spinal cord disclosed the region transformed into a yellowish and creamy mass at the level of the ninth and tenth thoracic segments. The mass came out freely on the slightest pressure from outside. An immediate microscopic examination of the mass showed that it consisted only of necrotic cord tissue. At the level of the eighth thoracic segment it was impossible to distinguish the normal demarcation of the white and gray matter, for the substance of the cord was yellowish, pultaceous and in part semiliquid. The sectioned surfaces appeared yellowish translucent in some areas and whitish opaque in others throughout the whole thoracic segments. The cut surfaces of the cervical, lumbar and sacral portions showed much less changes than the thoracic region.

Microscopic Examination.——

Brain: The following staining methods were employed: 1) the medullary sheath stain of SUGAMO, 2) that of YASHIRO, 3) combined stain of cells and fibers of KLÜVER-BARRERA, 4) the NISSL stain, 5) glia fiber stain of HOLZER, 6) axis cylinder impregnation of BIELSCHOWSKY 7) iron hematoxylin VAN GIESON stain, 8) hematoxylin-eosin stain.

As contrast, the brain of a woman, aged 36, who died of carcinomatous metastasis to meninges, and had been injected iodized oil that had passed freely through the ventricles to the cysterna magna and had produced no other effects than usual, was examined in the similar way.

Microscopic examination revealed scattered small foci of ischemic necrosis of the cerebral cortex of all lobes and occasionally of the adjoining white matter (Fig. 5). In deeper structures of the cerebrum such as the white matter widely distant from the cortex, the basal ganglia and the walls of the ventricles, no lesion of this kind could be seen. The optic nerves, chiasm



(A)

(B)

Fig. 8 Necrotic area at the junction of cerebral subcortex and cortex. B is a higher magnification of a part of A to show details of the nerve cells which are exeedingly pyknotic and hyperchromatic. KLÜVER-BARRERA stain; ×90 (A), × 1200 (B).

and tracts were intact. Many of the ischemic areas were ring-shaped (Fig. 5), but some were patchy (Figs. 5 & 7) or of geographical pattern (Fig. 6). The ring-shaped foci corresponded to the ring-shaped stripes mentioned above. To trace the extent of the lesions, some of them were examined in serial sections. The lesions were seen running tortuously and perpendicular to the frontal plane. As the lesions approached the cortical surface, they tended to be broader in dimension. Some reached the cortical surface at the depth of the sulci (Fig. 6) or on the crest of the gyri. Some occupied all laminae. Some penetrated into the subcortical

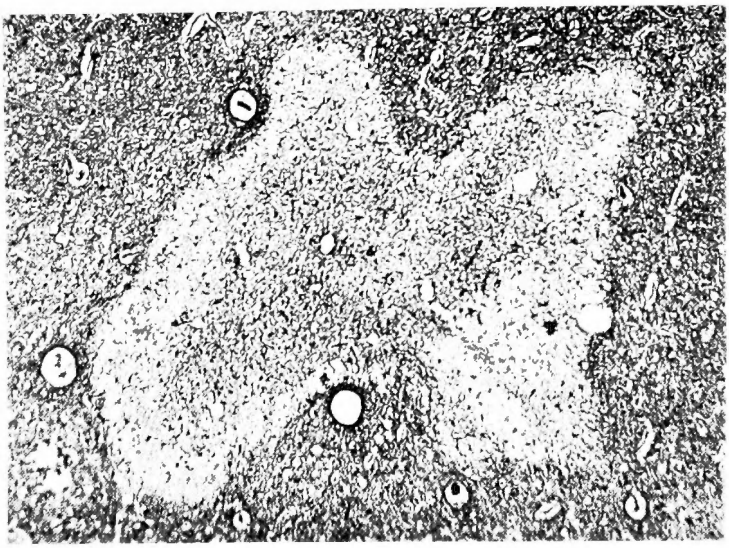


Fig. 9 Another level of the same lesion as in Fig. 5, B. The axis cylinders are affected as well as the myelin sheaths. BIELSCHOWSKY'S silver impregnation method; × 100.

white matter (Figs. 5 & 7). The lesions involving the subcortical white matter were occasionally sickle-shaped and were separated from the cortical substance by a thin layer of normal white matter, i. e., fibrae arcuatae (Fig. 7). All of the lesions were sharply defined and were isolated from each other (Fig. 5). The external border of the ring-shaped lesion was more distinct than the internal border (Fig. 5). In necrotized areas some of the nerve cells disappeared (Fig. 8), numerous myelin sheaths were irregularly swollen, beaded, or fenestrated or disappeared (Fig. 5), and axis cylinders were pale stained or disintegrated or disappeared (Fig. 9), and many glial nuclei were faded or shrunken. Proliferation of astrocytes was not brought out by the HOLZER glia fiber stain. In the center of the ring-shaped lesions most of the nerve cells were extremely pyknotic and hyperchromatic (Figs. 6 & 8), and myelin sheaths remained stainable, although they were of varying thickness, varicose and occasionally broken down into myelin globes (Fig. 5). Also in patchy lesions the changes tended to be more intense at the periphery than in the center. Thus one may suppose that necrosis occurred primarily at the periphery of the lesion and degeneration ensued secondarily in the center. The lesions always contained some small vessels (Figs. 5 & 8). Within or in the vicinity of necrotized areas the veins were extremely thick-walled (Fig. 10), and the arteries were occasionally occluded by comparatively fresh thrombus (Fig. 11). Cellular infiltrates were completely absent.

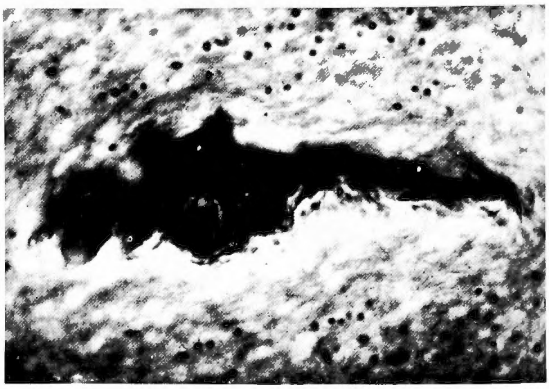


Fig. 10 Thick-walled vein in the necrotic area illustrated in Fig. 8, A. VAN GIESON stain; × 200.

The cerebellum was free from any conspicuous change.

The medulla oblongata and pons were examined in serial sections. In spite of the clinical sign suggesting involvement of the hypoglossal nerve, no definite focus in the medulla oblongata

could be made out, and the hypoglossal nerve roots emerging from the medulla were normal. Therefore, we have gained no information as to the nature of involvement of the hypoglossal nerve demonstrated clinically. It is probable that the nerve was impaired in its extracranial course. There was demyelination of the fasciculi graciles, the spinothalamic and spinocerebellar tracts due to secondary degeneration from the focus at the lower level of the spinal cord to be described later (Fig. 12). The nerve cells in the nuclei of GOLL were undergoing chromatolysis and were swollen, but there was no reduction in number. Sections from the pons failed to show any conspicuous change. No vascular changes were seen at the base of the brain. There was no evidence of more advanced sclerosis of the basilar artery than would be expected in a man of this age. The leptomeninges were slightly thickened, but there was no adhesion.

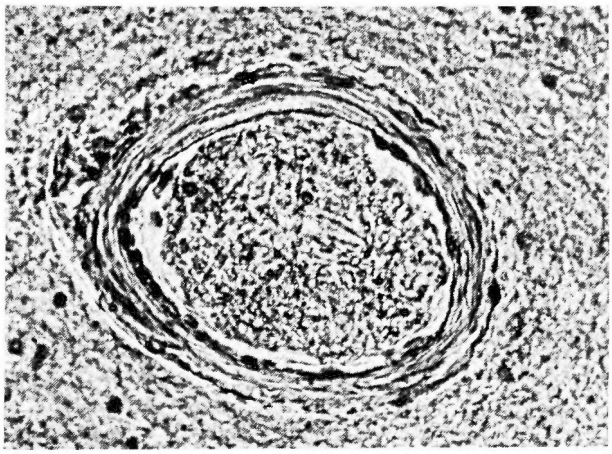
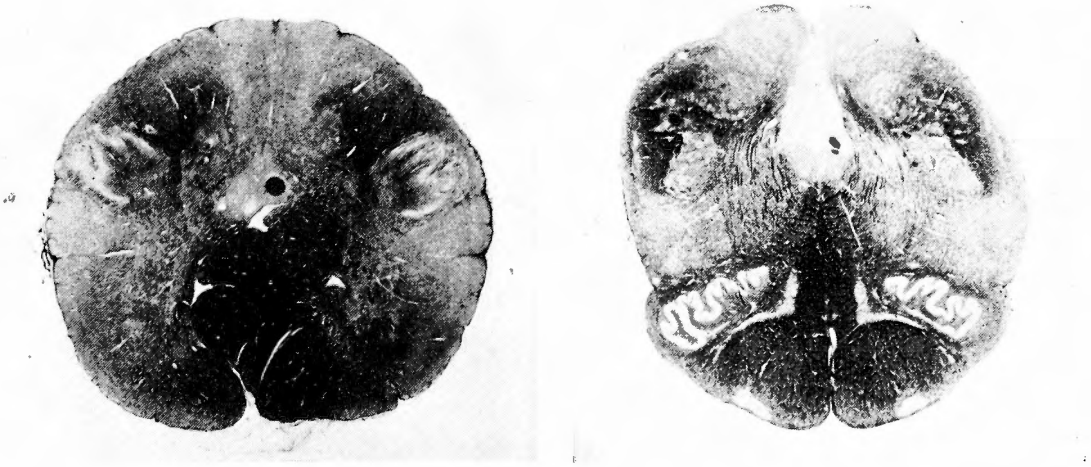


Fig. 11 An artery in the necrotic area illustrated Fig. 8, A. The lumen is filled with recent thrombus. Hematoxylin-eosin stain; × 700.

Spinal Cord: The cross-sections from every segment of the spinal cord embedded in celloidin were examined histologically after having been stained by methods similar to those employed on sections of the brain.

As contrast a normal spinal cord was examined in the same way.

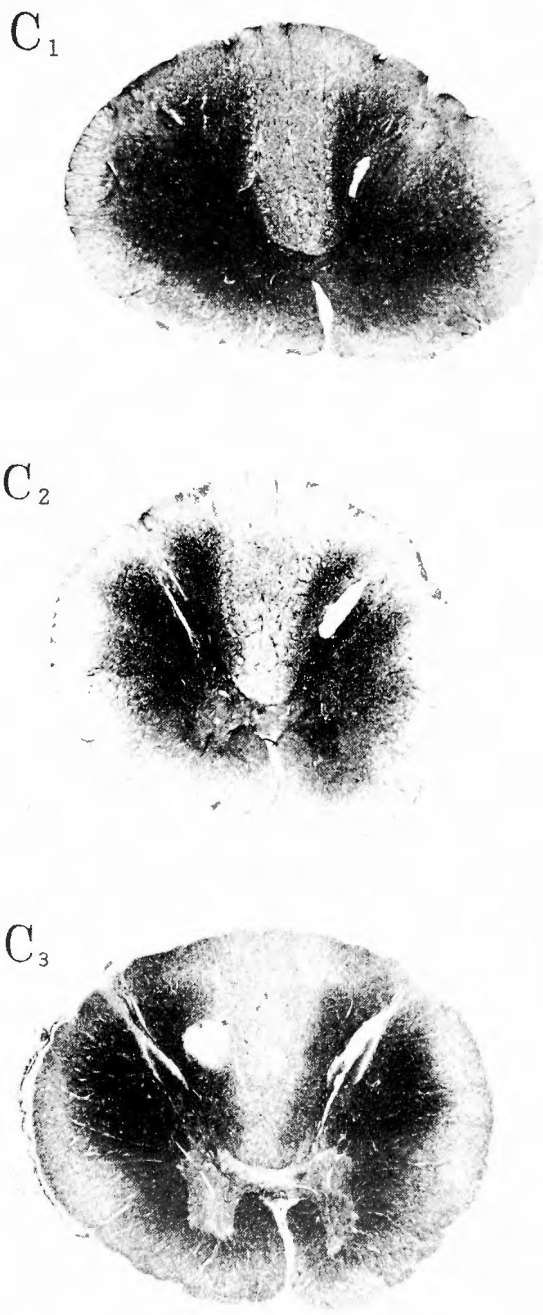
The microscopic examination revealed changes in all sections that were examined (Fig. 13). The most striking change was a widespread destruction throughout the whole thoracic segments. In the destructed area, there was a small central area of acellular necrosis surrounded by a zone of phagocytic infiltration (Fig. 14). The destructed area was irregular in outline. The white substance was involved predominantly, but the gray matter was not spared. Especially

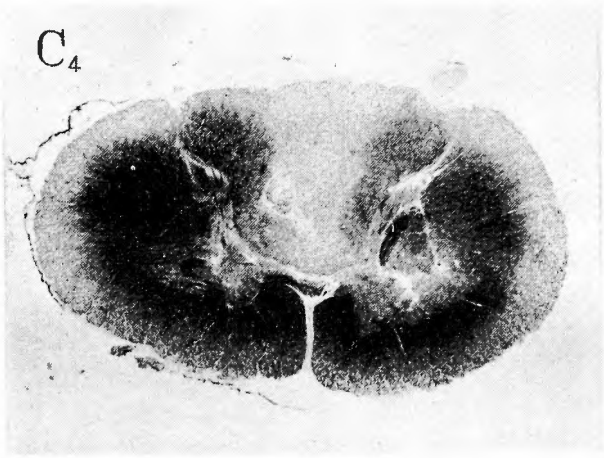


(A) (B)

Fig. 12 Cross-sections of the medulla oblongata showing secondary degeneration of the fasciculi graciles, the spinothalamic and spinocerebellar tracts. KLÜVER-BARRERA stain; × 7.

Fig. 13 Series of cross-sections of the spinal cord showing the longitudinal spread of necrosis. The details are given in the text. SUGAMO myelin stain (except C_1 and C_2 stained with YASHIRO's method); × 8.

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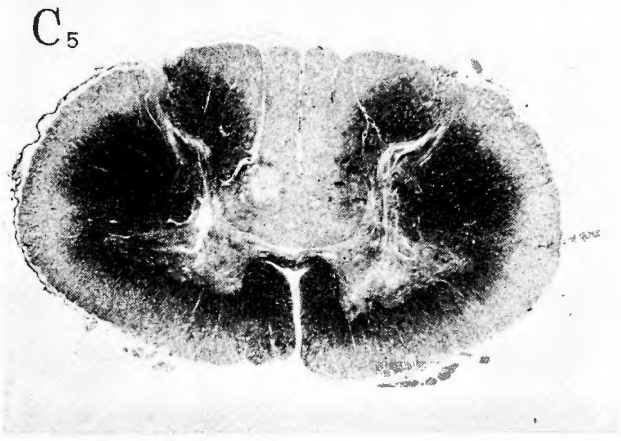
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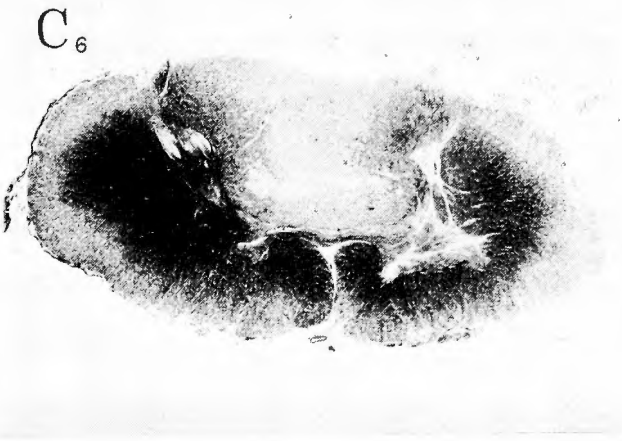
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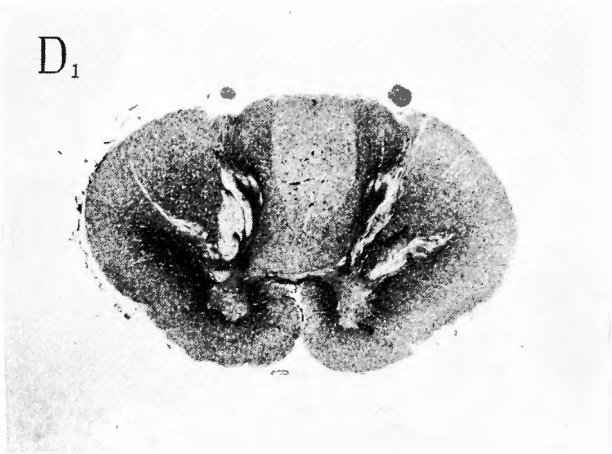
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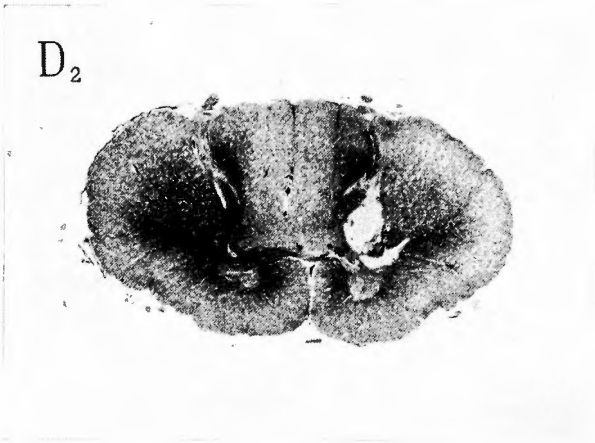
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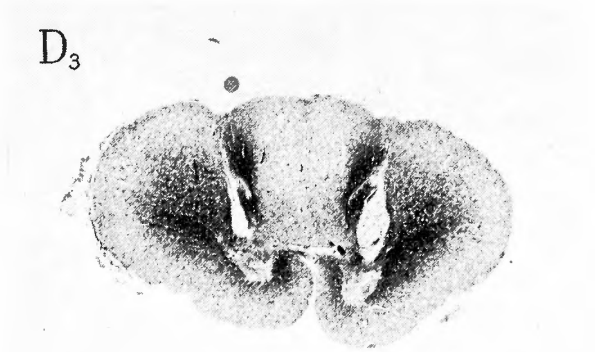
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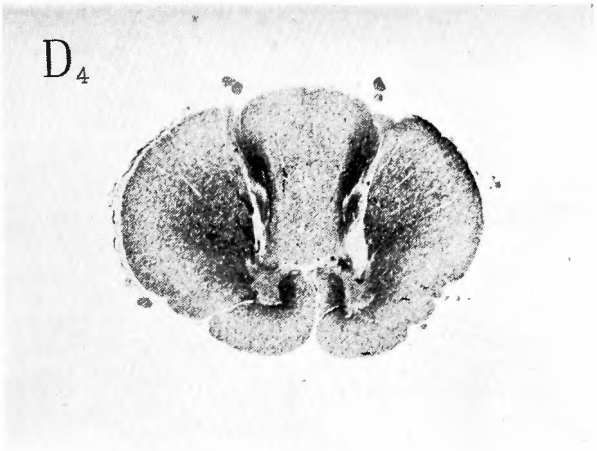
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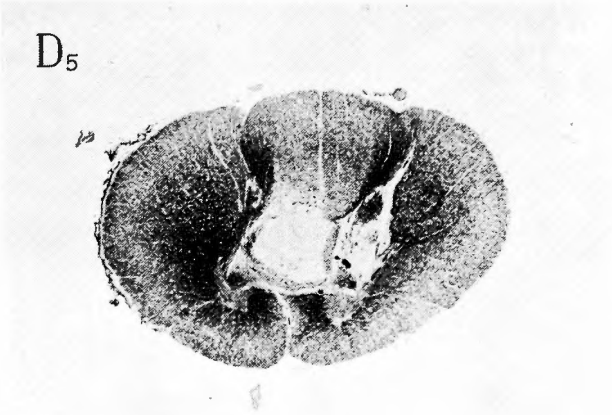
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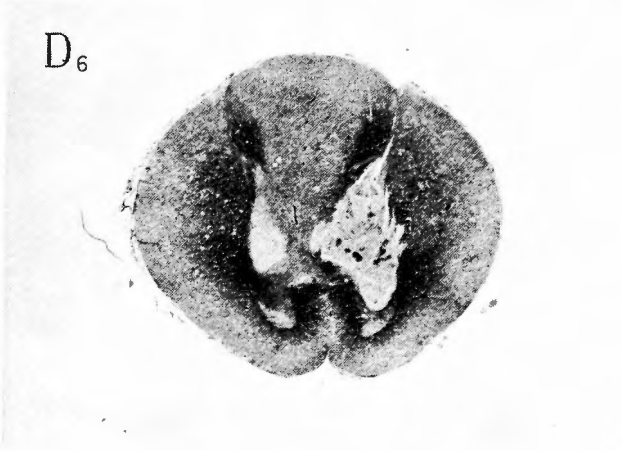
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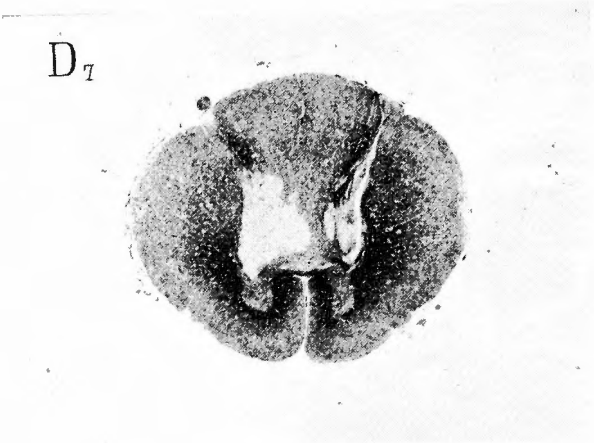
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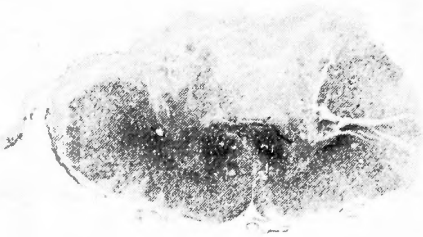

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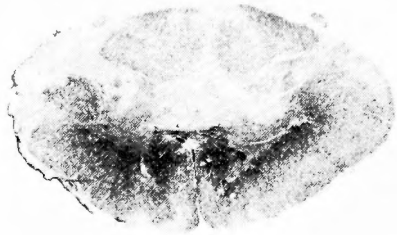

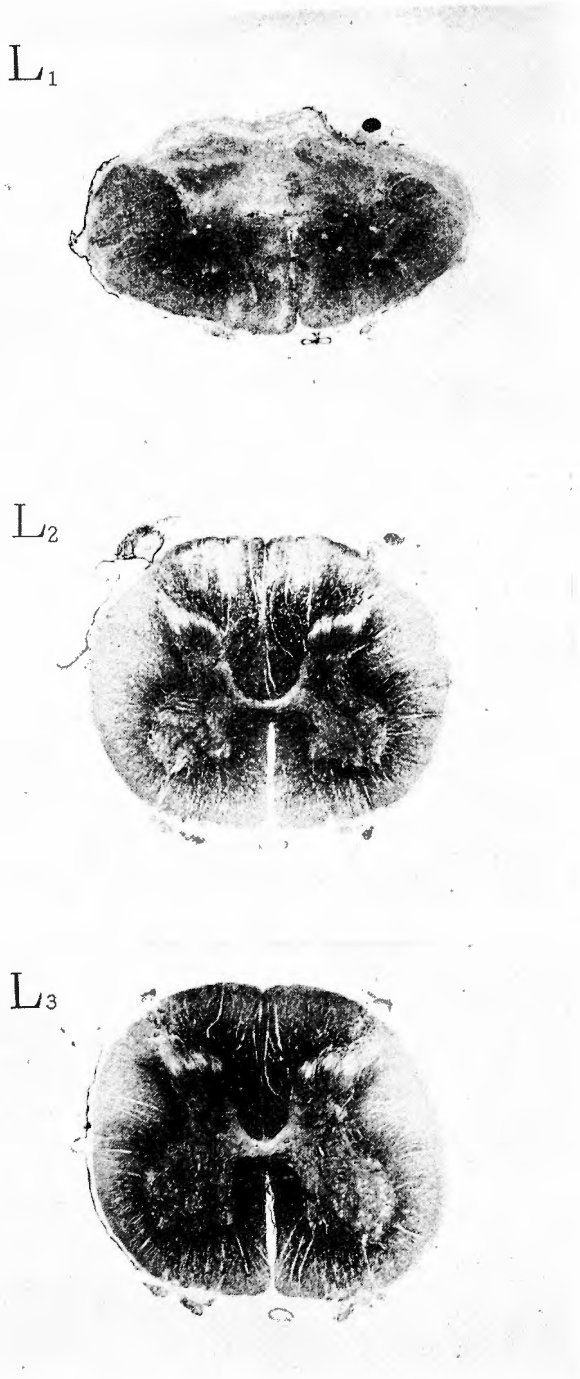
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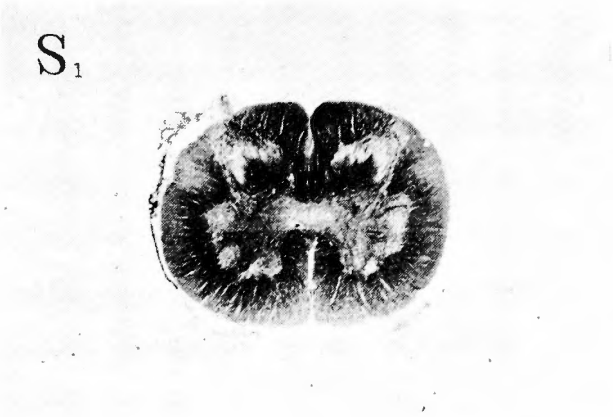
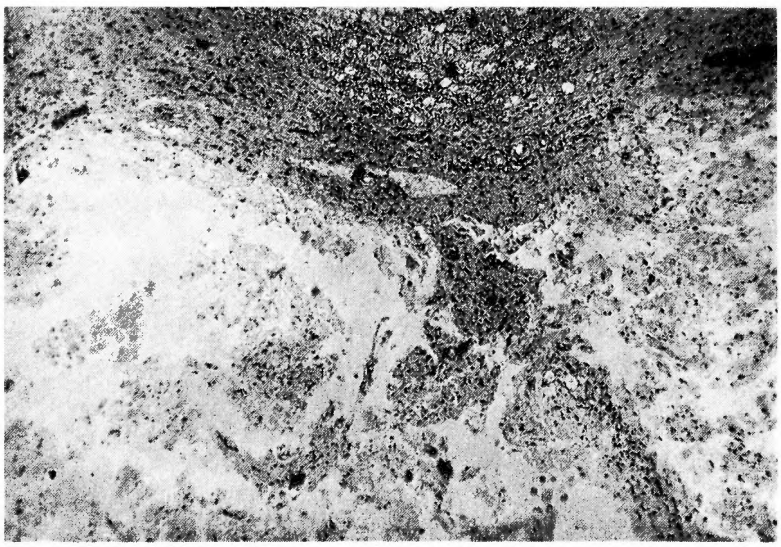


in the region of the ninth and tenth thoracic segments, containing a large necrotic cavity, any recognizable architecture could scarcely be made out. The sections from these segments, which had lost their affinity for the SUGAMO myelin sheath stain, showed a great excess of connective tissue in the leptomeninges as was demonstrated by the VAN GIESON stain (Fig. 15). At the level of the eighth thoracic segment, it was impossible to distinguish the gray from the white matter in the posterior half of the cord. The necrosis, the center of which was in the region of the ninth and tenth thoracic segments, extended up to the third cervical segment and down to the second lumbar segment. From the eighth up to the fifth thoracic segment, the necrosis involved largely the ventral portion of the dorsal columns, showing the central necrosis. Around this main necrotic area there were numerous small patches of necrosis in both white and gray matter, some of which were communicating with the central necrotic area. In the upper four thoracic segments, the necrosis was located at the junction of white and gray matter in the dorsal portion of the cord. From the eleventh thoracic down to the first lumbar segment inclusive, a large necrotic zone occupied the dorsal half of the cord and small wedge-shaped foci of necrosis were scattered in the marginal areas of the remaining portion. Above and below the level of the maximum involvement, i. e., the ninth and tenth thoracic segments, the areas of necrosis gradually decreased in extent, forming a fusiform syrinx. More or less demyelination was seen also in the unnecrotized portion of the cord, but the myelin nearest the gray matter tended to be better preserved than elsewhere. The anterior columns were relatively undamaged. The nerve roots entering and emerging from the spinal cord were fairly well preserved. The central canal was obliterated throughout the



Fgi. 14 Area of destruction in the seventh thoracic segment containing débris and phagocytic cells. Hematoxylin-eosin stain; × 160.

Fig. 15 Great thickening of the leptomeninges of the ninth thoracic segment. Blood vessels and nerve roots are embedded in dense connective tissue. VAN GIESON stain; × 8.

Fig. 16 Longitudinal section of the same spinal cord as in Fig. 13, D_7. SUGAMO myelin stain; ×8.

entire spinal cord. Longitudinal sections through the thoracic portion of the cord disclosed changes similar to those observed in the cross-sections (Fig. 16).

The cervical segments showed much less changes than the thoracic. Below the level of the third cervical segment there were small areas of necrosis scattered in the posterior columns and in the posterior horns. There was demyelination due to secondary degeneration in the unnecrotized portion of the posterior columns, particularly in the columns of GOLL, and in the periphery of the lateral columns.

In the lumbar and sacral segments, aside from the aforementioned necrotic foci at the level of the first and second lumbar segments, descending degeneration of the pyramidal tracts was demonstrated.

Reparative gliosis had taken place at the edge of the necrotic cavity (Fig. 17), but in the region of secondary degeneration there was no evidence of glial proliferation.

The anterior horns including CLARKE's columns showed marked loss of nerve cells due to

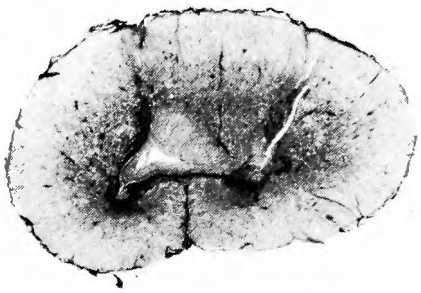


Fig. 17 Fibrous gliosis along the margins of necrotic area in the fifth thoracic segment. HOLZER stain; × 8.

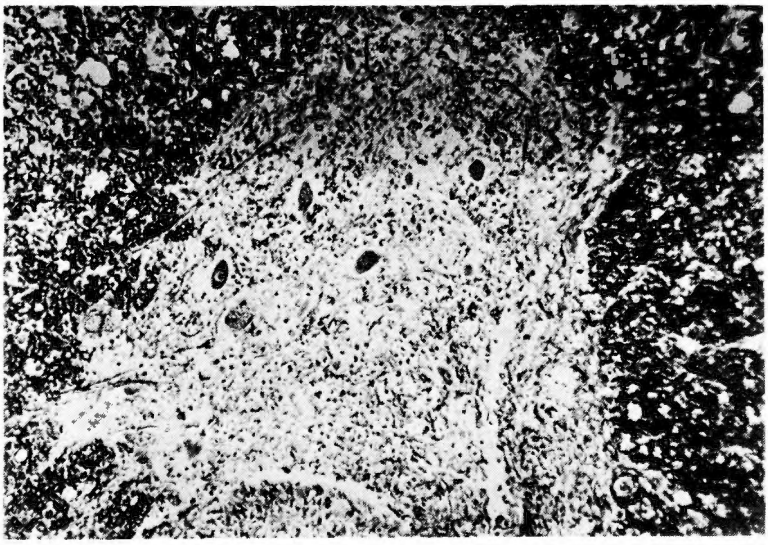


Fig. 18 Decrease in number of nerve cells in anterior horn of the fifth thoracic segment. KLÜVER-BARRERA stain; × 150.

destruction of the tissue (Fig. 18). Regressive changes in the surviving cells were seen in all NISSL stained sections, showing rounded outlines, eccentric nuclei and loss of chromatin (Fig. 19).

In spite of the wide extension of necrosis and the degeneration secondary to this profound lesion, there were no inflammatory changes throughout the entire spinal cord.

The blood vessels were extremely hyperplastic. The anterior spinal artery had thickened walls and consequently narrowed lumen, particularly in the lower thoracic portion of the cord (Fig. 20). There was also concentric thickening of the walls of the smaller arteries on the surface of the cord. Here the media and intima presented a thickened lamellation, suggestive of the "onion skin appearance" described by FOIX and ALAJOUANINE[42] (1926), but to a lesser degree (Fig. 21). The veins on the surface of the cord were affected similarly. The walls of

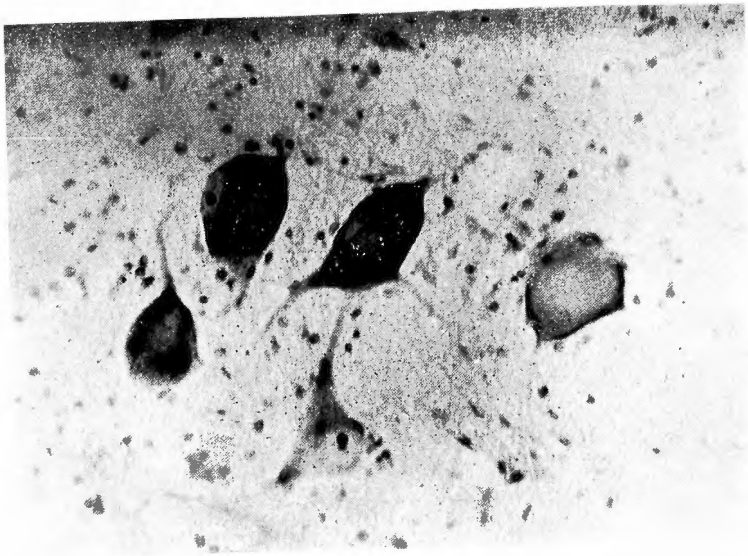


Fig. 19 Degenerative changes of nerve cells in anterior horn of the third lumbar segment showing abnormal shapes and chromatolysis. NISSL stain; × 350.

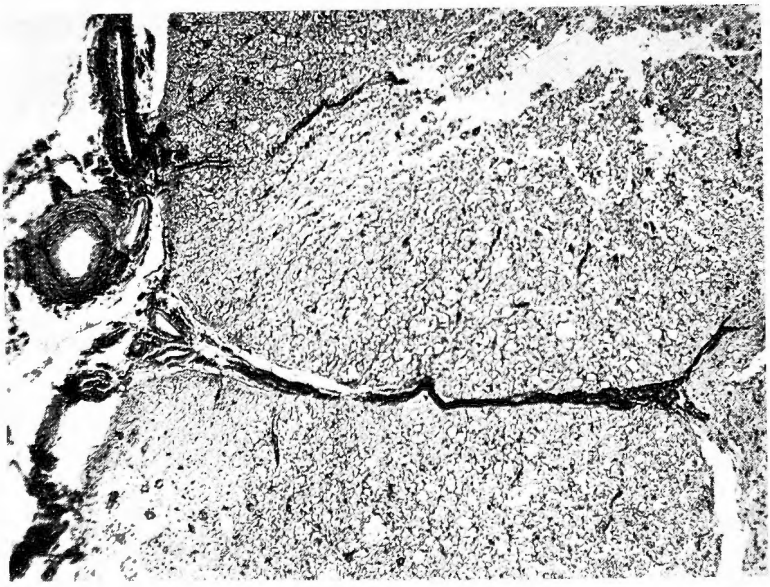


Fig. 20 Anterior spinal artery and vein from the eleventh thoracic segment and their branches lying in the thickened fibrous leptomeninges. The vessels have greatly thickened walls and narrowed lumens. VAN GIESON stain; × 140.

the veins appeared to be composed entirely of concentric layers of elongated connective tissue cells of adventitia (Figs. 21 & 22). On the dorsal surface of the maximally damaged segments there were veins, whose lumina were filled with connective tissue as the result probably of organization of a thrombus (Fig. 22). Within the substance of the cord, all of the small vessels showed pronounced hypertrophy, with thickened intima and narrowed lumen. Immediately around the necrotic areas there were newly formed blood vessels with thickened walls. Especially in the areas adjacent to necrotic cavity in the eleventh thoracic segment,

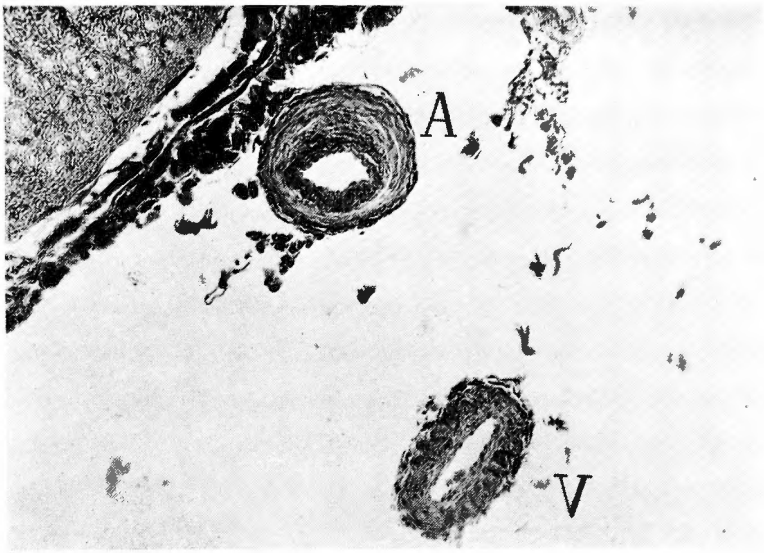


Fig. 21 Small artery and vein in the pial membrane of the fourth thoracic segment. The artery (A) has thickened intima and media, suggestive of "onion peel appearance," while the vein (V) shows only adventitial thickening. VAN GIESON stain; × 120.

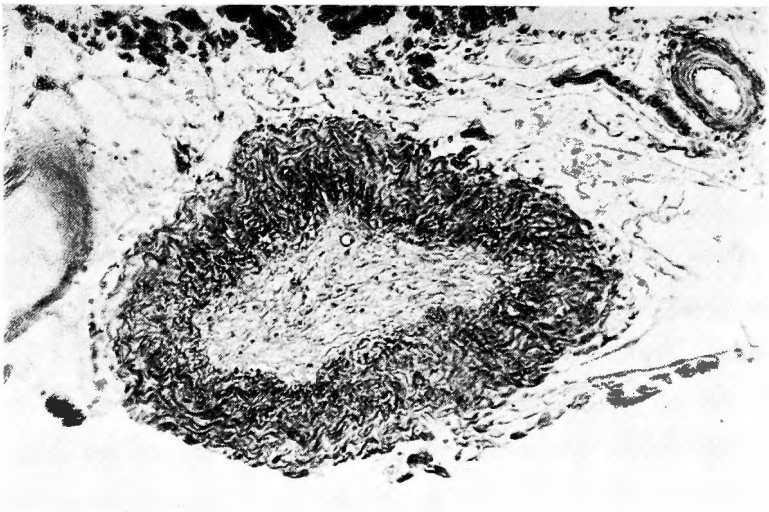


Fig. 22 A vein on the dorsal surface of the eleventh thoracic segment. The lumen is filled with connective tissue, the result of organization of a thrombus. VAN GIESON stain; × 140.

vascular tufts were formed (Fig. 23). The lumina were in some instances almost totally obliterated on account of excessive hyaline thickening of their walls (Fig. 23). Above and below the level of maximum necrosis the vascular changes gradually diminished in severity. Vascular hyperplasia was most prominent in areas showing maximal destruction of the cord, and little or no hypertrophy of vessels was seen in other areas of the cord. There was neither vascular dilatation nor recent hemorrhage in any part of the cord.

Changes in the meninges were noted at the level of the ninth and tenth thoracic segments, where vessel changes and cord necrosis were most marked. The dura mater, for the most part, was normal in thickness. The leptomeninges, howevr, showed a great thickening. There

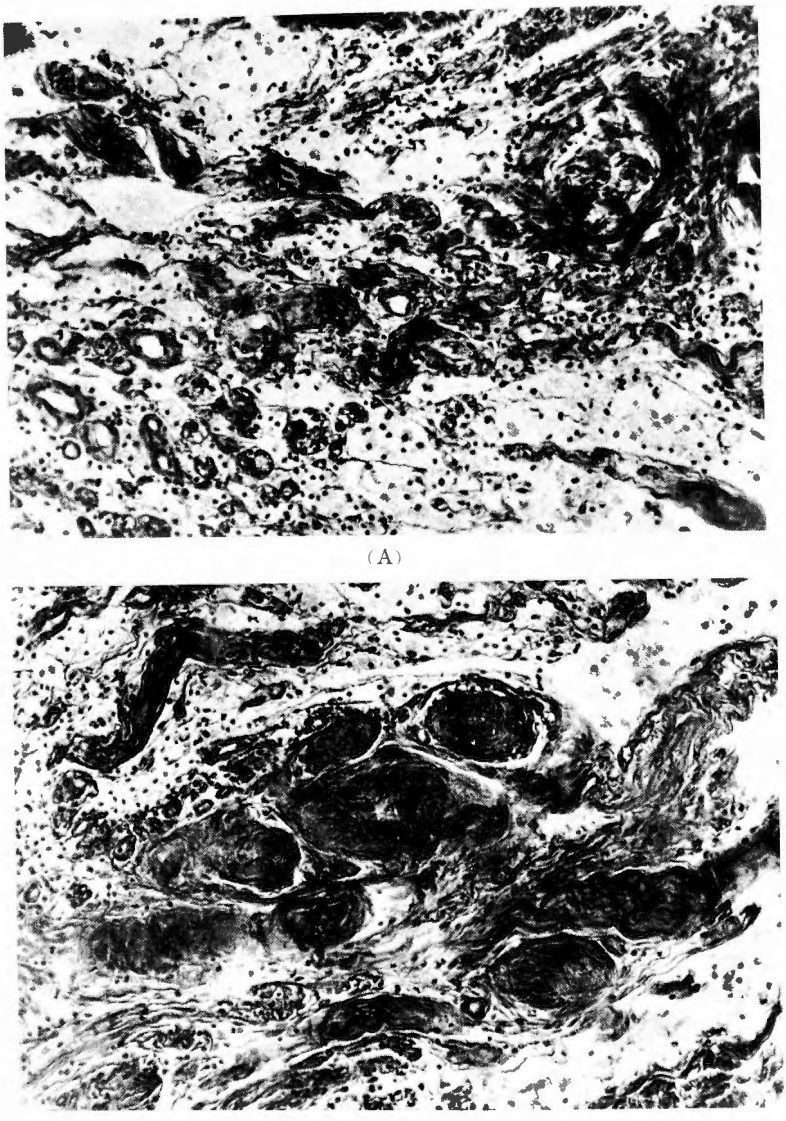


Fig. 23 Newly formed blood vessels located immediately adjacent to necrotic cavity of the eleventh thoracic segment. A shows vascular tufts. B shows the vessels to be almost completely obliterated by excessive hyaline thickening of their walls. VAN GIESON stain; × 100.

was dense connective tissue between dura and spinal cord, having approximately two or three times the thickness of the normal dura (Fig. 24). Blood vessels and nerve roots were embedded in this connective tissue (Fig. 15); the former had greatly thickened walls, and the latter was undergoing degeneration. The fibrotic process extended into the anterior median fissure, surrounded the anterior spinal artery and vein, and penetrated into the cord around most of the small, deep blood vessels (Fig. 20). Perivascular collections of round cells were seen in the thickened arachnoidal membrane (Fig. 21).

COMMENT

Prior to discussion of etiology and pathogenesis of the present case, the

available literature may be reviewed.

Many[12,13,16,20,26,29] have written on the ill effects of the intrathecal injection of iodized oil, since SICARD and FORESTIER[23] (1922) began the use of this substance in the subarachnoid space. Aside from the numerous observations, both clinical [9,11,15,22,27,28] and experimental,[5,10,19,21] of meningeal irritation or meningitis due to injection of iodized oil into the spinal canal, there are a few reports of the experimental spinal cord lesion which in some respects seems to resemble the lesion observed in the present case. KLOSE and PEIPER [18] (1925), after injecting iodized sesame oil in the rabbits in doses greater than 0.1 cc intraspinally and intracysternally, reported following pathological changes: in the nerve cells the NISSL bodies were clubbed and pale stained; other intracellular structures were stained poorly and sharp ouline of the cells was lost; there were also changes around the central canal, characterized by necrosis with a surrounding wall of leucocytes. DAVIS, HAVEN and STONE[14] (1930) injected 1.5 cc of iodized poppy seed oil intracysternally in dogs after an artificial block had been produced in the spinal subarachnoid space, and they found proliferative changes in the leptomeninges and degeneration of the gray matter. Necrosis of the spinal cord was not described. It is noteworthy that VAN GIESON's stain showed enlarged vessels and thickening of their walls on the ventral surface of the cord and the vascularity of the anterior horns was increased with many new capillaries, and vessels of the anterior half of the gray matter were often thrombosed. SICARD and FORESTIER[24] (1932) commented, however, that the amount of iodized oil used in these animals was much greater, in proportion to the body weight, than that used in human patients and there was a difference between animals and man in their susceptibility to iodized oil. These experimental studies, however, suggest that the same lesions as were observed in animals may possibly be produced in man.

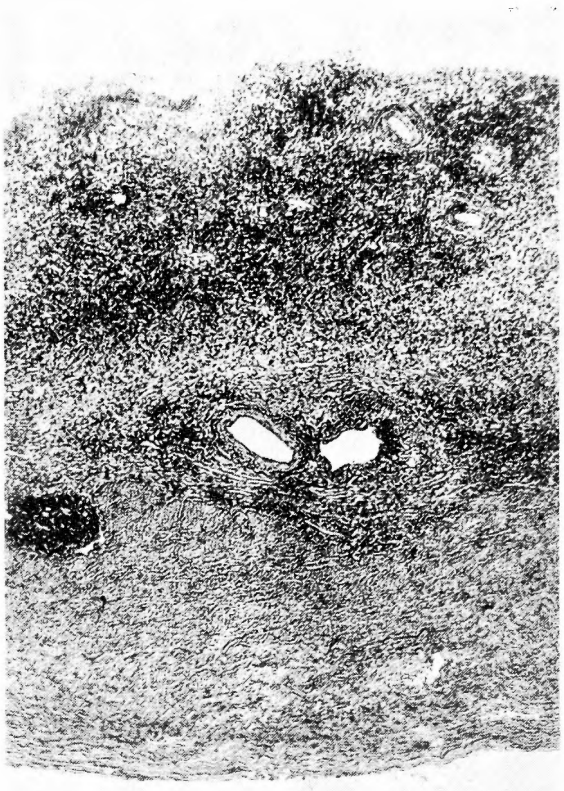


Fig. 24 The dura mater with arachnoidal fibrosis above. There are marked perivascular collections of the thick arachnoid with round cells. VAN GIESON stain; ×120.

There are reports on the ill effects of intra-aortic injection of roentgen contrast media containing iodine upon the spinal cord, since the early report of sodium iodide used for aortography by Dos SANTOS, LAMAS and PEREIRA[32] (1929). Experimentally,[33,34,35,36,37,38] it has been recognized that a large amount of these agents

produces necrosis of the spinal cord. Paraplegia and death of one patient were reported by ANTONI and LINDGREN[30] (1949) following aortography using iodopyracet (Diodrast). Autopsy revealed necrosis of the spinal cord. BOYARSKY[31] (1954) reported paraplegia and survival of one patient following aortography using sodium acetrizoate (Urokon). This patient had a complete motor and sensory loss below the level of the eighth thoracic segment but later recovered from the sensory loss and partially regained bladder function and motility of lower extremities.

Since FOIX and ALAJOUANINE[42] (1926) reported two cases of a condition which they named subacute necrotic myelitis, many reports[41,45,51,52,56,61] concerning necrosis of the spinal cord have been made under various headings, including acute necrotic myelitis (VAN GEHUCHTEN[44] 1927), angiohypertrophic gliosis of the spinal cord (LHERMITTE, FRIBOURG-BLANC and KYRIACO[54] 1931), progressive necrosis of the spinal cord (MOERSCH and KERNOHAN[57] 1934), spinal necrosis and softening (JAFFE and FREEMAN[48] 1943), necrotic myelopathy (GAGEL and MÉSZAROS[43] 1948), angiodysgenetic necrotizing myelopathy (SCHOLZ and MANUELIDIS[59] 1951), diffuse necrotic myelitis (KAHLE and SCHALTENBRAND[49] 1955), acute necrotic myelopathy (HOFFMAN[47] 1955), and FOIX-ALAJOUANINE's disease——angiodysgenetic myelomalacia (BODECHTEL and ERBSLÖH[40] 1957). There is, however, little agreement of opinions regarding the etiologic factors or the nature of the pathologic changes. Without going into detailed descriptions of the clinical and pathologic features of this disease entity, I should only say that, since the contribution of FOIX and ALAJOUANINE[42] (1926), many reports have pointed out the importance of a vascular factor in the pathogenesis of this cord lesion. With regard to the etiologic factors the existence of some toxic or infectious agent has been assumed since the first report of FOIX and ALAJOUANINE[12] (1926). Many investigators expressed the opinion that both vessel changes and cord necrosis were simultaneous responses to it. The experimental work of LOTMAR[55] (1913) presented further evidence of this opinion. Injection of dysentery toxin into rabbits produced necrotic foci in the spinal cord, with degeneration of nerve elements and regressive glial changes, without much evidence of inflammation.

RISER, GERAUD and PLANQUES[58] (1937) described a case of subacute necrotic encephalomyelitis in which both brain and spinal cord were coincidently affected. In the cerebrum there were lymphocytic infiltrations, foci of rarefaction, gliosis, vascular occlusion and some perivascular hemorrhages. The spinal cord was noticeably softened and spongy at the seventh cervical segment. SHUNK and KERNOHAN[60] (1939) reported a case of progressive necrosis of the spinal cord. In the cerebrum of this case there were widespread foci of degeneration of the white matter. These foci simulated in some respects the lesions of acute multiple sclerosis or of encephalitis periaxialis diffusa. The spinal cord was seen to be completely necrotic in the lumbar and sacral portions. These authors hesitated to assume that they were dealing with two separate disease because of the histologic similarity of the two processes.

Adhesive spinal arachnoiditis leading to cavitation of the spinal cord has hitherto been reported in at least 13 verified cases,[65,66,68,70] since the first case of

Schwarz[69] (1897). Most of the workers considered that the cavitations were due to ischemia resulting from thickening and/or compression of vessels in the leptomeninges by adhesive arachnoiditis. The experimental work of McLaurin, Bailey, Schurr and Ingraham[67] (1954) added further support to this opinion. They observed chronic adhesive spinal arachnoiditis produced in dogs by the intracysternal injection of 3 to 6 cc of ethyliodophenylundecylate (Pantopaque). The arachnoiditis led to communicating myelomalacia, with multiple cavitations located at the junction of white and gray matter in the cervico-thoracic portion of the spinal cord.

The experimental work of Langworthy[53] (1932) is of interest in its relation to the problem of the development of spinal necrosis. He produced necrosis in the spinal cord of rats by application of alternating electric currents of different potentials (18 to 1.000 volts). The contacts of the circuit were made on the dorsal surface of the head and the tail. Many of the animals developed a complete paralysis of the posterior portion of the body as soon as they recovered from the shock. The lesion, in general, remained confined to the dorsal columns, although sometimes the whole cord became necrotic. The cavity contained rather few red blood cells but many fat droplets and some amorphous débris. Tauber and Langworthy[62] (1935) stated that these cavities were not the result of hemorrhage inasmuch as so few red cells were present and that it was also difficult to regard them as having arisen by a direct action of the current upon the cord. These authors concluded that the sudden shock of the current might produce a transient anemia and eventually a necrosis of the cord. In other words, it seems to me that vascular spasm due to the shock of the current might give rise to necrosis of the cord.

Pathologic changes of the cerebrum due to disturbance of cerebral blood flow during convulsive process have been recognized not only in the experimental work [1,72,73,74,76,80] but also in the study of cases recorded as unexpected death after electroshock therapy. Schulte and Dreyer[77] (1950) described an autopsy case of cerebral complication after electroshock therapy. In the cerebrum there were hemorrhagic purpura, ring-hemorrhage and small areas of necrosis in both white and gray matter. Scheidegger[75] (1952) added a case in which small areas of necrosis and circumscribed regions of light lavender color were found mainly in the cerebral cortex and occasionally in the subcortical white matter. He expressed that these lesions were accounted for on the basis of a marked anemia of the cerebral cortex due to a vascular spasm attending the shock of the current. In this country, two reports of each one case of similar nature have been made by Takasaka et al[79] (1954) and Shida et al[78] (1957) respectively.

It is of interest to speculate on the sequence of events which occurred in the case of this paper.

After admission iodized-oil-ventriculography was performed because there were clinical manifestations of a circumscribed disease of the posterior cranial fossa. Iodized-oil-ventriculogram, however, disclosed no tumor. Shortly after iodolography the patient had a transient convulsive attack with loss of consciousness, followed

by "transverse lesion of the spinal cord." During the period of survival no recovery of function took place in the cord at any level below the lesion. At autopsy, adhesive arachnoiditis at the level of the maximally damaged segments was found. The histological changes of the spinal cord, characterized by extensive necrosis of the cord and pronounced hypertrophy of the vessels both inside and outside the cord, resembled those found in the disease called subacute necrotic myelitis by Foix and Alajouanine[42] (1926). In the cerebrum, some small areas of ischemic necrosis were sporadically found only in the cerebral cortex and adjoining white matter. With regard to designation in the diagnosis, therefore, it seems to me that the case reported here is best described as "acute necrotizing encephalomyelopathy following iodized-oil-ventriculography."

From the clinical point of view, there can be little doubt that the patient had suffered from adhesive arachnoiditis at the level of the ninth and tenth thoracic segments prior to injection of iodized oil. The symptoms on admission including the backache, weakness and numbness of legs and difficulty in gait are referable to the adhesive arachnoiditis. Therefore, it is inferred that the preexisting adhesive arachnoiditis might have affected the blood vessels in the pial membrane and produced more or less reactive hyperplasia of the vessels, causing diminution of the blood supply of the spinal cord, and thus resulting in slight regressive changes of the nervous elements of the cord. When iodized oil reached the cerebral base it might exert an irritating influence responsible for the generalized convulsion which occurred about four hours after iodolography. Cerebral vascular spasm attending the convulsive attack might be followed by the scattered small areas of ischemic necrosis of the cerebrum where the blood vessels might already have undergone some changes. It would be reasonable to infer that the lesions of the spinal cord were also the result of vascular spasm due to iodized oil that entered the spinal subarachnoid space. But for bringing about a grave irreversible change, i. e., massive necrosis of the spinal cord, not only vascular spasm but also the preexisting changes of the blood vessels and spinal cord secondary to adhesive arachnoiditis seem to be necessary. It is possible that further hyperplasia of blood vessels and meninges may follow necrotic process of the cord substance. Accordingly vascular and meningeal hyperplasia mentioned in this case may be for some part secondary to tissue necrosis. Affection of blood vessels on the entire circumference of the cord might cause the least blood supply in the central portion of the spinal cord, consequently resulting in the central necrosis. It is well known that only the terminal branches of the arteries that enter the marginal portion of the cord supply the central portion. In the present case the necrosis was not found in the specific distribution of the anterior or posterior spinal arteries.[39,50,63,64] The affected region contained the terminal branches of both major arteries. At least, the dramatic suddenness of the onset suggests a vascular accident, and ischemia would seem to be a much more probable mechanism than inflammation. The pathological changes of both the brain and spinal cord may well be explaned as the result of an ischemic phenomenon; i. e. sudden and complete interruption of the blood supply. Inspite of this, in the histologic pictures, no evident vascular

occlusion could be demonstrated except for the venous thrombus on the surface of the most severly damaged segments of the cord and some arterial occlusions in necrotized areas of the cerebrum, which may have been of secondary rather than of primary importance. At any rate, it would be plausible to assume a vascular spasm to account for the clinico-pathological correlation, although there is no definite morphologic evidence for this assumption.

But, any answer can not be given at all to the question as to why iodized oil, which is being widely employed for the diagnosis and localization of adhesive spinal arachnoiditis, might be directly responsible for vascular spasm in this case alone. With the experimental works[14,18] mentioned above in mind, however, it is obvious that iodized oil injected played a part, if not as a single causation, at least as a precipitating factor in producing the necrosis. In other words, the etiology is largely of exogenous toxic origin.

This case has been presented in the hope that attention may be drawn to a dangerous fact that iodized oil, if injected into the cerebrospinal space, can bring about severe damage of the spinal cord. From the standpoint of the pathogenesis of "necrotic myelopathy," it would appear that the study of this case makes a foreward step in the understanding of the problem of the development of spinal necrosis in which some toxic agent as an etiologic factor has long been considered, since the contribution of Foix and Alajouanine[42] (1926).

SUMMARY

A case of acute necrotizing encephalomyelopathy following iodized-oil-ventriculography is described with the details of pathological findings. The histological changes of the spinal cord, characterized by massive necrosis of the cord and enormous hypertrophy of both intra- and extramedullary blood vessels, are identical with those found in the condition called subacute necrotic myelitis by Foix and Alajouanine[42] (1926), although the present case is unique in that the disease occurred shortly after injection of iodized oil into the lateral ventricle. In addition, there are changes of the cerebral cortex and adjoining white matter which are characterized by the dissemination of small areas of ischemic necrosis.

From the clinical and pathological findings, the problems of etiology and pathogenesis are discussed, with special reference to the vessel changes and the iodized oil injected.

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* written in Japanese.

和文抄録

ヨード油脳室造影により惹起された ACUTE NECROTIZING ENCEPHALOMYELOPATHY

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55才の男子. 軽度の言語及び嚥下障害，背痛，両下肢の脱力感ないし痺れ感，軽度の歩行困難等を愁訴として入院し，脳腫瘍の疑いでヨード油脳室造影を施行したが，異常所見を認めず，従つて腫瘍は否定された．所が，造影後4時間程して，突然意識喪失を伴つて全身痙攣が現われ，又翌朝第4胸髄節以下に完全横断麻痺のあることが発見された．その後，この横断麻痺像には少しの緩解も見られず，褥瘡及び尿路感染による全身衰弱のため，造影後約3ヵ月半を経過して死亡した．

著者は本症例について，中枢神経系全般にわたる詳細な組織学的検索を行い，次の如き所見を得た．

1) 大脳に於いては，総ての脳葉に小さい断血性壊死巣が認められ，而もこれらは主に大脳皮質に，又時としてそれに近接した白質に限局して散在性に認められる．又，これらの壊死巣の大部分は輪環状を呈するが，一部には斑点状のものや地図状のものも認められる．又，病巣部及びその近辺の静脈は著しく肥大し，動脈には一部に血栓形成が認められる．

2) 脊髄に於いては広範な壊死が認められる．即ち，第9及び第10胸髄節では脊髄実質は完全に破壊され，こゝを中心に上は第3頸髄，下は第2腰髄に及ぶ略々紡錘状の壊死巣を形成し，主に脊髄の後半部で，白質，灰白質共に罹患している．又同時に，脊髄内外の血管に著しい肥大と，そのための内腔狭窄を認める．以上の所見は，Foix & Alajouanine(1926)の記載した亜急性壊死性脊髄炎の組織像に一致する．

3) 第9及び第10胸髄節の部位に，著明な癒着性蜘網膜炎が認められる．

本症例に見られた全身痙攣は，脳底部に達したヨード油の刺戟によるものと考えられる．しかし，このような痙攣は普通には見られないものであるから，何らかの大脳病変(恐らく血管性病変)の既存を想定する必要がある．又，壊死の発生には，血管攣縮による急激な断血機転が直接関与したものと思われるが，大脳に於いてはこのような既存病変が，又脊髄に於いては既存の癒着性蜘網膜炎が，ヨード油注入以前に既に或る程度の退行性病変を齎らしていたものと考えられる．