

Spinal cord infarction occurring during thoraco-lumbar sympathectomy

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The arterial blood supply of the spinal cord depends on a single anterior spinal artery and two posterior spinal arteries. The anterior spinal artery, which is the more important, commences within the cranial cavity by the union of the anterior spinal branches of each vertebral artery and subsequently is fed by several tributary arteries. Down the length of the spinal cord the anterior spinal artery receives paired tributaries accompanying each anterior nerve root, and, arising from the vertebrals, the ascending cervical branches of the inferior thyroid, the costo-cervical trunk, the costal, lumbar, iliolumbar, and lateral sacral arteries. Most of these arterial tributaries are small and the blood supply is largely dependent on four to 10 of these arterial tributaries which are especially large. Their position and size are variable, but at least one joins the cervical cord, two the thoracic cord, and one the lumbar cord. The largest thoracic tributary has been given the name, the great ventral radicular artery.

Spinal cord infarction, because of interference with an important radicular tributary, is a rare complication of thoraco-lumbar sympathectomy. In a brief survey of the literature we found only 12 previously recorded cases in which this complication was presumed to have occurred. Rubio (1953, quoted by Corbin, 1961) reported three cases, and a further case was added by Mosberg, Voris, and Duffy (1954) who also cited four cases of Bassett (1948) and a personal communication of three cases of Poppen. Nathan (1956) mentions a further case. Detailed pathological observations at necropsy were made in two cases, that of Mosberg and that of Nathan. The following case report with necropsy findings describes spinal cord infarction occurring as a complication of the operation of thoraco-lumbar sympathectomy.

CASE REPORT

In 1947, at the age of 31, Miss V. G. (Radcliffe Infirmary no. 300290), a bank clerk, developed malignant hypertension. In 1950 her blood pressure recordings ranged

from 240/130 to 260/150 mm. Hg and she entered a London hospital for thoraco-lumbar sympathectomy. The left sympathetic chain, from T2 to L2 plus the splanchnic nerves, was extirpated at the uneventful first stage. Three months later the right sympathetic chain, from T1 to L2 plus the splanchnic nerves, was removed through a thoracotomy with ninth rib resection. There were no obvious technical problems or hypotensive episodes. On recovery from the general anaesthesia, an incomplete sensori-motor loss below T9 was immediately apparent. Lumbar puncture revealed entirely negative findings.

Subsequently the paresis and sensory loss partially recovered, but on discharge four months later she was only able to walk a few yards with a stick and a right leg calliper. Both legs remained spastic and without perception of pain, temperature, and vibration, but some perception of touch and joint movement returned. During the next 10 years the neurological picture remained unchanged but the blood pressure slowly increased. The final admission was the result of a fatal subarachnoid haemorrhage.

NECROPSY FINDINGS

The general findings (P.M. 974/60) were left ventricular hypertrophy, fibrous pleural adhesions, and shrunken kidneys (right more than left). Sections showed changes of hypertension with evidence of prior chronic pyelonephritis.

BRAIN The rupture of a congenital berry aneurysm on the left anterior cerebral artery had caused such extensive subarachnoid bleeding as to be the immediate cause of death.

MENINGES The subarachnoid space was filled with recent blood clot.

VESSELS The anterior spinal artery was joined by large radicular arteries accompanying the left C3, right C8, left T1, and left L2 anterior nerve roots. The left L2 branch was especially large and above this there was no major tributary to the anterior spinal artery until that accompanying the left T1 anterior nerve root. The veins appeared prominent,

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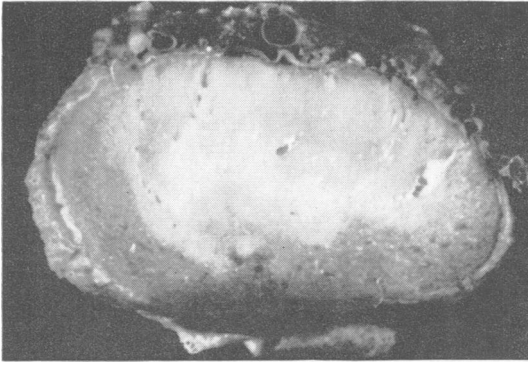


FIG. 1. Transverse cut through the lower thoracic region of the spinal cord viewed from above. The atrophied anterior part of the cord is grey.

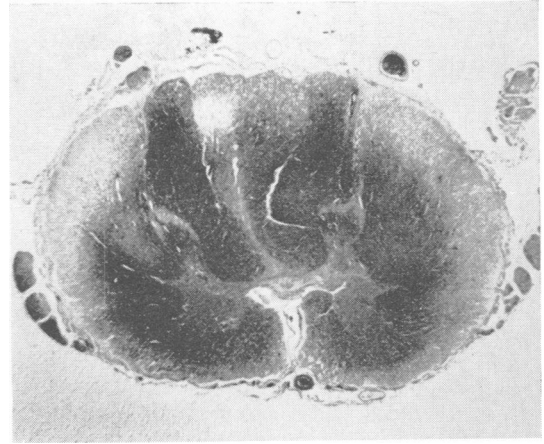


FIG. 3

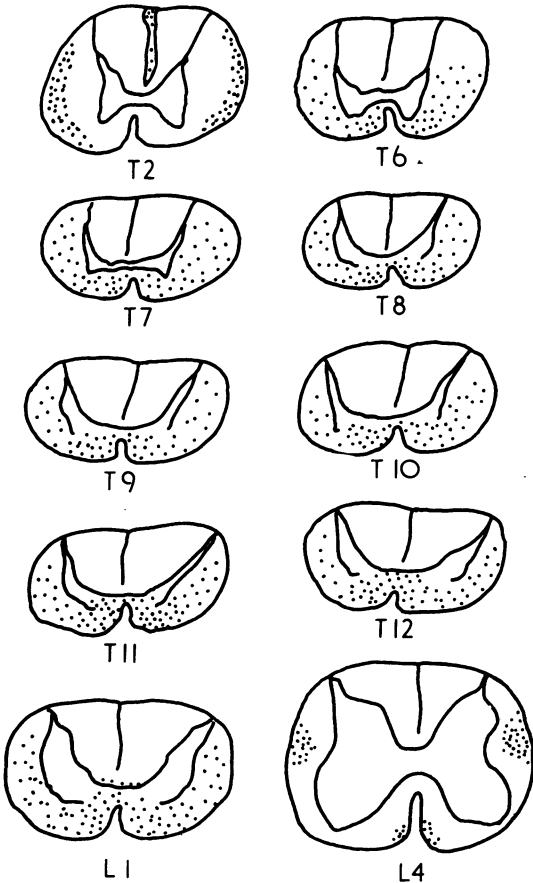


FIG. 2. Drawings from camera lucida tracings of transverse sections through the spinal cord at various segmental levels. The stippled area is gliotic. Note the destruction of the anterior horn and shrinkage of the anterior two-thirds of the cord from T6 to L1. Wallerian degeneration is seen in T2 (above) and L4 (below) the cord lesion.

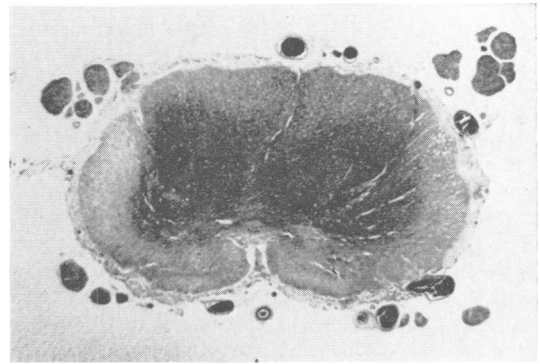


FIG. 4

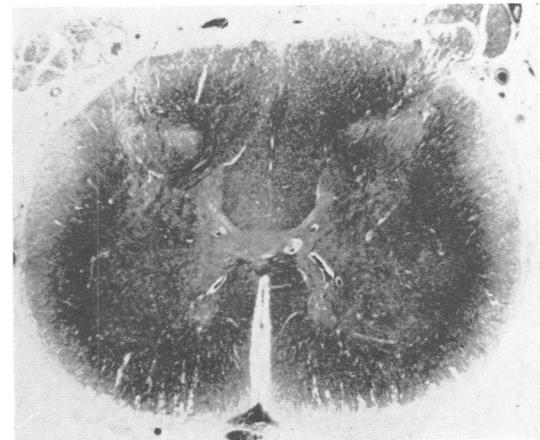


FIG. 5

FIGS. 3, 4, AND 5. Myelin stain of transverse sections through T3, T10, and L4. In T10 there is atrophy of the anterior two-thirds of the cord. Wallerian degeneration is seen in T3 and L4.

particularly on the anterior aspect of the spinal cord.

SPINAL CORD From T6 to L1 the spinal cord was shrunken, the anterior part of the cord being particularly affected (Fig. 1).

HISTOLOGICAL EXAMINATION OF THE SPINAL CORD

Transverse sections were taken through C5, C8, T1-T12, L1-L5, and S2.

The sections C5, C8, and T1-T4 showed similar appearances (Fig. 3). There was leptomeningeal thickening with some recent subarachnoid blood. Myelin and fibres were lost in the posterior columns and in the anterior and posterior spino-cerebellar tracts due to upward Wallerian degeneration above the cord lesion. At these levels the anterior horns showed no neuronal depletion and the nerve roots were normal. The vessels showed no evidence of occlusion. A transverse section through T5 showed the upper limit of spinal cord damage. The anterior horns of this segment showed few neurones which were intermingled with some astrocytic gliosis. The main damage to the spinal cord was from T6 to T12 (Figs. 2 and 4). These segments showed severe depletion or absence of neurones from the anterior horns. The anterior and lateral columns were shrunken, the normal tissue being replaced by dense astrocytic gliosis. All the anterior nerve roots from these segments showed atrophy with myelin and fibre loss. There was considerable leptomeningeal thickening. None of the vessels seen showed any evidence of occlusion. The lower limit of the cord lesion was seen in L1 where the anterior horn showed depletion of neurones with gliosis. Sections of L2, L3, L4 (Fig. 5) and L5, and of S2 showed pyramidal tract degeneration with myelin and fibre loss due to downward Wallerian degeneration.

DISCUSSION

In the case described here the region of spinal cord infarction is the anterior spinal artery territory from T7 to T12 cord segments, with minor changes in T6 and L1. This infarcted region corresponds to the distribution of a major thoracic tributary to the anterior spinal artery. The vessel obstructed in all probability entered from the right side, being disturbed during the second operation of right thoraco-lumbar sympathectomy. The vessel concerned may have been damaged during the rib resection, an operation known to be the occasional cause of spinal cord ischaemic damage. Rouquès and Passelecq (1957) reported a case of the Brown-

Séquard syndrome following thoracoplasty, and Binet (quoted by Corbin, 1961) observed paraplegia following a left pneumonectomy. Alternatively during the actual extirpation of the sympathetic chain a nearby intercostal artery may have bled and so required surgical ligation.

The rarity of this complication of sympathectomy requires consideration. The operation is a common one and accidental damage to intercostal or other arteries occasionally occurs, usually without untoward sequelae. The explanation lies in the peculiar anatomical arrangement of the vascular supply of the spinal cord whereby only a few of the numerous tributaries to the anterior spinal artery are of profound importance. When by chance the artery obstructed gives origin to a major radical like the great ventral radicular artery, extensive ischaemia will occur.

The injection experiments of Bolton (1939) have suggested that the blood flow in the anterior spinal artery is in a downward direction. When an important tributary artery is injected the whole of the anterior spinal artery down to the filum terminale is filled, but the injection material only passes upwards for one or two segments. An anatomical reason for this is that a large tributary artery usually joins the anterior spinal artery in a caudal direction and for a short distance above this junction the calibre of the anterior spinal artery is small, whereas immediately below the junction the calibre increases. In individual cord segments, Bolton could find little evidence of anastomosis between anterior spinal and posterior spinal arteries. He did find at the caudal end of the spinal cord an extensive anastomosis between the anterior spinal artery and both posterior spinal arteries.

In the case described here the extent of the spinal cord infarction agrees with the anatomical studies of Bolton. Caudal to the infarcted spinal cord, the first large tributary accompanied the L2 nerve root. This large vessel had provided for the lumbo-sacral cord but had failed to nourish the thoracic cord above. This finding confirms the caudal direction of blood flow in the anterior spinal artery. The lack of demonstrable anastomosis between anterior and posterior spinal arteries in an individual cord segment was also confirmed since the infarcted region was strictly confined to the anterior two-thirds of the spinal cord.

SUMMARY

A case of spinal cord infarction occurring during thoraco-lumbar sympathectomy is described.

The affected region of the spinal cord (the anterior two-thirds of segment T6-T12) indicated that a

major tributary to the thoracic cord had been obstructed.

The case illustrates anatomical peculiarities of the arterial supply to the spinal cord.

We are grateful to Mr. J. Pennybacker for permission to publish this case and to Dr. D. Oppenheimer who performed the necropsy.

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