

Submitted by: J. T. Hughes
The Radcliffe Infirmary
Oxford, England OX2 6HE

Reference: 993

A 34-year-old man was the driver of a car involved in a "head-on" collision. He sustained a non-penetrating 'steering wheel' chest injury which caused a rupture of the aorta 2 cm. distal to the left subclavian artery. Neurological examination was normal.

At a cardio-thoracic surgical unit, through a left thoracotomy at the level of the 4th rib, the damaged part of the aorta (see diagram) was resected and replaced by a 3.5 cm. long Dacron crimped tubular graft, whilst a left atrium to left femoral bypass carried on the aortic blood flow for the required 70 minutes. The blood pressure in the upper aorta was monitored through a cannula in the right radial artery. At the end of the operation, the normal anatomy of the aorta was restored and bilateral femoral pulses were recorded.

On recovery from the anesthetic, the patient was unable to move his legs and the first post-operative examination indicated a severe paraparesis with accompanying sensory loss. By the following day, it was clear that he had complete areflexic paralysis of both lower limbs with complete loss of sensation below the T9 - T10 sensory level. Bladder and bowel function were also paralysed. This neurological state remained static until his death five months later due to mediastinal hemorrhage from a leak of the Dacron graft anastomosis.

The two diagrams provided give the gross findings at necropsy in the aorta, and the histological findings in the spinal cord, every segment of which was sectioned.

The slide of three transversely cut sections of spinal cord (L1, L2 and L3) is stained by hematoxylin and eosin.

Points for Discussion:

1. The mechanism by which the spinal cord was damaged. *check in text*
2. Pertinent features in clinical history, operative procedure and necropsy findings as related to Point #1. *2*