10 g.) taken orally are fatal from acute gastro-intestinal, hepatic, renal, and central-nervous-system damage (Lidbeck *et al.*, 1943). Protracted intake of about 14 parts per million of water over many years may result in calcium fluoride being deposited in bone, tendons, and periosteum (Singh *et al.*, 1962). Smaller doses (1-2 parts per million of water) protect teeth from carious decay (Weaver, 1944).

We have not been able to find any record of fluoride causing ocular damage in man. Sorsby and Harding (1960), however, described retinal oedema followed by degenerative changes in 17 out of 94 rabbits within five days of giving sodium fluoride; and De Berardinis and others have produced retinal degeneration in rabbits by intracarotid injection of 150 mg. of sodium fluoride (Babel and Avanza, 1961; De Berardinis *et al.*, 1962). Fluoride is a powerful inhibitor of glycolysis and has been shown by Holmes (1940) and Kerly and Bourne (1940) to inhibit lactic-acid formation and phosphorylation in retinal extracts. Although it cannot be definitely established that the eye lesion in our patient was caused by sodium fluoride, there was no evidence for any other cause of toxic optic neuritis or retinal oedema, such as methyl-alcohol poisoning or disseminated sclerosis. His serum calcium was normal, thus excluding the rare papilloedema due to hypocalcaemia, and the fundal appearance was not that of raised intracranial pressure. Moreover, the fact that his eye trouble began only six weeks after starting to take a powerful enzyme poison makes the latter highly suspect. It is possible that as his parathyroid glands

had been removed less fluoride was laid down in bone and more in soft tissues. His normal serum alkaline phosphatase after treatment may be relevant to this, since sodium fluoride is known to raise the alkaline phosphatase concentration in the plasma.

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Spinal-cord Infarction Due to Aortic Trauma

Brit. med. J., 1964, 2, 356

In the catastrophe of traumatic aortic rupture the general consequences usually overwhelm any coincidental impairment of the blood supply to the spinal cord. In the following case the aortic damage was more discrete but extensive infarction of the spinal cord had resulted. This association has not hitherto been described, although the similar consequences of spontaneous dissecting aneurysm of the aorta are well recognized, and Thompson (1956), reporting such a case with necropsy findings, reviewed seven previously published cases.

CASE REPORT

A 62-year-old housewife crashed her car into a shop, suffering injuries to her chest and a lacerated chin. Although momentarily unconscious she was able to walk away from her car and an ambulance took her to hospital. On admission she was found to have fractures of the second, third, and fourth ribs anteriorly, with a progressive left haemothorax necessitating a thoracotomy the following day. Removal of 2 litres of blood from the left hemithorax disclosed a 2.5-cm. tear in the pleura over the aorta just above the diaphragm, with neighbouring extensive mediastinal bruising. Massive blood transfusion restored her general condition, but on the fourth post-operative day impaired movement of the legs was noticed and a full neurological examination the next morning showed complete paralysis of the legs and absence of all sensation up to the level of T 3. Plain x-ray films of the spine showed no fracture and a myelogram did not demonstrate any evidence of spinal block. On the sixth post-operative day the patient's blood-pressure suddenly collapsed and 15 minutes later she died.

At necropsy the thoracic aorta showed a transverse intimal tear almost encircling the aorta at a level 4 cm. below the origin of the left subclavian artery. The tear was plugged with thrombus continuous with a large mural haematoma in the damaged tunica media and adventitia. At their origin the second, third, fourth, and fifth intercostal arteries were surrounded and compressed by the mural haematoma. The spinal cord showed extensive infarction from T 3 to T 7 (not examined caudal to T 7), the whole diameter of the cord being involved. Neurones were absent and both myelin and axons were broken up with a perivascular polymorph reaction but few lipid phagocytes. A round infarcted area was present in the posterior columns of C 7 and there were minor ischaemic changes in C 8, T 1, and T 2.

COMMENT

The blood supply to the spinal cord is dependent on several tributaries to the single anterior spinal artery and to the two less important posterior spinal arteries. Most of these tributaries are small and the blood supply mainly comes from a few that are especially large. The anterior spinal artery in the thoracic region of the spinal cord usually receives two tributaries of inconstant position (Hughes and Macintyre, 1963). These tributaries are derived from spinal branches of the intercostal arteries, and it is these intercostal branches of the aorta that were blocked at their origin in this case in the similar but commoner spontaneous dissection of the aorta.

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