POSTOPERATIVE PARAPLEGIA IN A PATIENT WITH AN UNSUSPECTED DURAL ARTERIO–VENOUS MALFORMATION

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SUMMARY
We report a case in which laminectomy was performed in a patient with symptoms attributed to lumbar canal stenosis. The patient developed paraplegia in the early postoperative period. Further investigation revealed a dural arterio–venous malformation. This was treated surgically, producing some neurological recovery. The possible mechanisms for the perioperative deterioration and their implications for anaesthesia are discussed.

KEY WORDS
Complications: paraplegia, arterio–venous malformation.
Surgery: spinal.

CASE REPORT
A 64-yr-old man presented with a 20-yr history of low back pain. His pain worsened over the previous 12 months, and in addition he developed severe buttock pain aggravated by exercise. He also noted progressive leg weakness with tingling at night and increasing hesitancy of micturition.

At the time of presentation, his quadriceps muscles were markedly wasted, with noticeable weakness that affected also the hamstring and peroneal muscle groups. His reflexes were normal with flexor plantar responses, but there was diminished sensation to pinprick in the L5 dermatome bilaterally. He was found also to have a palpable bladder. His general clinical condition was good; preoperative arterial pressure was 140/95 mm Hg.

Myelography revealed an apparent lumbar canal stenosis at L4–5 (fig. 1) to which the symptoms were attributed. He underwent spinal cord decompression at this level, but at operation

FIG. 1. The AP projection myelogram shows a filling defect at L4–5.
the canal was not found to be stenosed and there was no evidence of intervertebral disc herniation.

Anaesthesia had been induced with etomidate 14 mg and supplemented with fentanyl 100 µg, tubocurarine 30 mg and droperidol 2.5 mg. Intermittent positive pressure ventilation was carried out with 60% nitrous oxide and 1–2% isoflurane in oxygen.

Systolic arterial pressure was controlled at 75–90 mm Hg during the procedure by adjusting the inspired concentration of isoflurane. Surgery was performed with the patient in the prone position, and took approximately 90 min.

In the recovery room, the patient could flex his legs at the knee along the bed, but not against gravity. However, over the following 6 h flaccid paralysis of both legs developed, with a sensory deficit to T10.

Repeat myelography to a higher level demonstrated a filling defect at T12 with no obstruction to flow of contrast at the operative site (fig. 2). Subsequent selective spinal angiography showed that the defect was caused by a large arterio–venous malformation (AVM) with a single supply vessel originating at L1 (fig. 3). This vessel was clipped at operation 4 weeks after the original laminectomy. Surgery confirmed a large dural AVM with no evidence of thrombosis or direct cord compression.

Neurological recovery was slow, but after 3 weeks he was able to stand and he showed some improvement of the sensory deficit.

DISCUSSION
Spinal angiomas consist of anomalous arterio–venous communications without an intervening capillary network. Thus they may be described also as an AVM or an arterio–venous fistula
Coronal veins
Radial veins
Radicular vein
Anterior median spinal vein

FIG. 4. The venous drainage of the spinal cord.

(AVF). Typically, a patient presents with insidious onset of back pain, leg pain, or weakness. These symptoms may be related to posture, exercise or pregnancy [1, 2]. Claudication may be a feature, mimicking peripheral vascular disease or spinal stenosis [3]. Muscular wasting and weakness progress steadily. Acute neurological deterioration occurring in a patient with an AVM may be caused by thrombotic occlusion or haemorrhage [4].

Spinal AVM may be classified into dural or intradural lesions. Dural AVM are fed usually by an arterial branch of an intercostal or lumbar artery. The AVM is embedded within the dura and forms a venous connection with the coronal plexus of veins surrounding and draining the spinal cord [4]. Intradural AVM are located on the surface of, or within, the spinal cord and an AVF exists between an artery supplying the cord and the local venous system.

Most dural AVM are thoraco–lumbar (96%) and tend to present in middle-aged patients. Intradural AVM are more common in the cervical and thoracic regions of the cord and are found in younger patients [5].

The mechanism of development of neurological deficit in patients with a spinal AVM is uncertain. Cord compression is rare and myelographic or manometric evidence of subarachnoid obstruction is uncommon. Decompression laminectomy usually fails to prevent neurological deterioration [6]. Fluctuations in the severity of symptoms, particularly in association with exercise or changes in posture, suggest that alterations in blood flow may be responsible. A form of circulatory steal is thus an attractive theory, with diversion of blood away from the spinal cord thereby producing symptoms. However, the symptoms usually do not correlate with those anticipated from the distribution of a particular spinal artery [7] and, in the case of a dural AVM, it is rare to find a communication between the lesion and a vessel supplying the spinal cord.

It appears that venous drainage of an AVM may be critically important in the development of symptoms. The spinal cord drains via a series of radial veins, the majority of which empty into the longitudinally placed coronal plexus, which in turn connects with the extradural veins (fig. 4). The anterior midline region of the spinal cord drains via the radial veins into the anterior median spinal vein [8]. A dural AVM usually receives blood from one or more arteries that accompany the posterior nerve roots, draining directly into the coronal venous plexus. The A–V shunt produces increased pressure within the coronal veins which is transmitted directly to the radial veins draining the spinal medulla. Thus an area is produced with a reduced A–V pressure gradient and hence impaired tissue perfusion. Symon, Kuyama and Kendall have suggested that an AVF increases venous pressure, reducing spinal blood flow. This results in reflex vasodilatation and an increased tissue pressure, leading to oedema and ultimately ischaemic, compressive cord damage [9].

If this mechanism of production of neurological deficit and cord damage is correct, anaesthetic manoeuvres which affect arterial perfusion pressure and local venous pressure are of critical importance. In the case described it is possible that the postoperative neurological symptoms were produced by medullary ischaemia following a hypotensive anaesthetic technique in a patient with an undiagnosed AVM. Reduction in systemic arterial pressure, either deliberate or accidental, is undesirable, and results in reduced spinal cord blood flow. In addition, the prone position used during surgery may produce increased local venous pressure resulting in exacerbation of cord ischaemia.

Other factors such as the use of intermittent positive pressure ventilation and large tidal volumes may contribute also to an increase in venous pressure. It has been noted that the Valsalva manoeuvre exacerbates symptoms in patients with an AVM [10].

As lumbar spondylosis and spinal AVM occur within similar age groups and have similar clinical and myelographic features, particular care should
be taken to maintain cord perfusion when providing anaesthesia for lumbar spinal surgery.

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REFERENCES