

Central spinal stenosis

Classification and pathogenesis

Richard W Porter

Department of Orthopedic Surgery, University Medical Buildings, Aberdeen AB9 2ZD, Foresterhill, Scotland
Tel +44-224 681818. Fax +44-224 685157.

Spinal stenosis is an abnormal narrowing of the bony and/or ligamentous structures of the vertebral canal. Only when it produces symptoms does it attract the clinician's attention, and then stenosis is only one factor amongst many which contribute to the syndrome. What are these syndromes, and what is the role of the small canal? What is the difference between stenosis of the central canal and stenosis of the root canal? And what is the etiology of the small canal?

The anatomy of the vertebral canal is bicompartamental. The first is the central canal—that space within the neural arch which extends to the next segmental level, with artificial boundaries at the proximal part of the root canal. The second compartment is the root canal—the space between the lateral part of the central canal at the intersegmental level extending laterally to the foramen. The shape of the central canal is variable. Most canals are dome shaped or bell shaped throughout the lumbar spine, but approximately 7% are trefoil at L4, and 15% trefoil at L5 (Porter 1980; Figures 1 and 2). The cross sectional area is also variable, and when a canal is trefoil, it also tends to be small in the sagittal diameter. A trefoil shaped canal has a deep lateral recess, with overhanging superior facets, whilst a dome shaped canal may have no lateral recess at all.

Classification

Congenital stenosis is assumed to be genetically determined. In achondroplasia, the canal is generally small in both sagittal and interpedicular diameters.

Developmental stenosis. The vertebral canal reaches maturity in the cross sectional area and in the sagittal diameter by four years of age (Figure 3). Thereafter the pedicular diameter widens, and the shape changes, but not the overall size. Environmental factors which impair growth before four years of age, particularly interuterine factors like toxins, drugs, alcohol, smoking, bacterial or viral infections, placental insufficiency, these can leave a permanently stunted canal, with no capacity for catch-up growth.

The individual may become a six foot man but be hiding a small canal from some early growth deficiency. The whole canal from cervical to lumbar level is small.

Degenerative stenosis. Degenerative change of bone and/or soft tissues can affect isolated segments as a result of disc degeneration, or following a degenera-

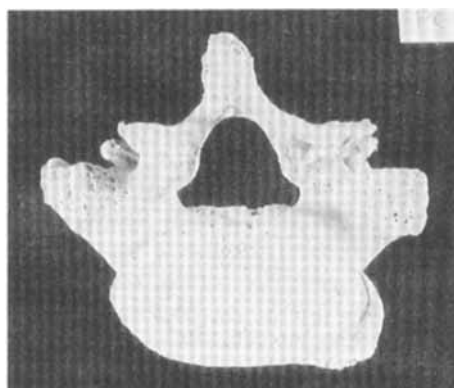


Figure 1. A fifth lumbar vertebra showing a large dome-shaped central vertebral canal

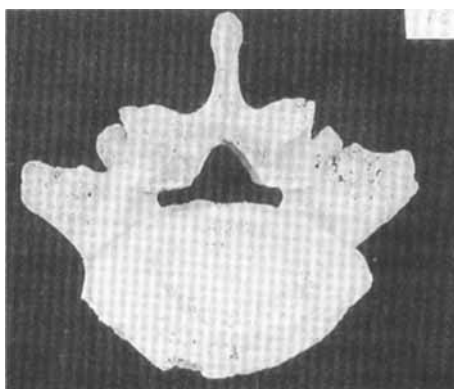


Figure 2. A fifth lumbar vertebra with a trefoil-shaped central canal, a small mid-sagittal diameter and deep lateral recesses.

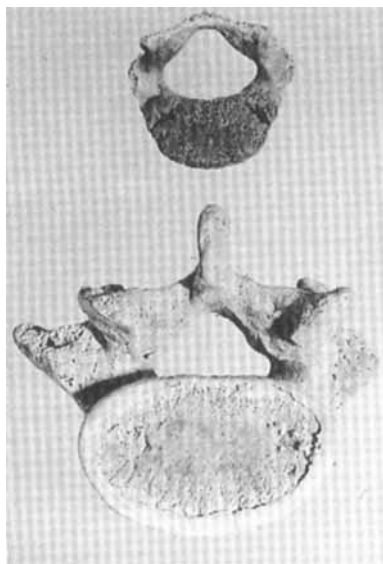


Figure 3. Two fifth lumbar vertebrae. The upper specimen is from a four year old child and the lower from an adult. The size of the vertebral body is different, but the cross sectional area of the vertebral canal is identical. The mid-sagittal diameter is slightly larger in the infant. In the adult the interpedicular diameter is wider.

tive spondylolisthesis. Alternatively many segments may be involved in systemic hyperostosis, or in degenerative lumbar scoliosis. Ossification of the posterior longitudinal ligament will stenose long segments of the spine.

Mixed stenosis is a combination of the above types, and it is this which usually occurs when stenosis produces symptoms.

Symptoms

Stenosis can be present with no symptoms at all. Boden and colleagues (1990) showed that 21 percent of asymptomatic subjects over 60 years of age had spinal stenosis. Stenosis is only one factor amongst many, but there are three syndromes where it is significant.

Neurogenic claudication. Patients experience pain or discomfort in one or both legs when they walk, but this is not present at rest. It is unusual before 50 years of age, and men are affected more often than women. They have little trouble at rest, but after a short distance their legs begin to feel uncomfortable, and at twice this distance they have to stop. They have multiple levels of stenosis, one always in the central canal and sometimes also in the root canal. There is coexistent peripheral vascular disease in about 30%.

Symptomatic disc protrusion. A protruding disc is not uncommonly symptomless. By 60 years of age, 36% of asymptomatic subjects have a herniated disc (Boden et al 1990). The presence of disabling symptoms depends to some degree on the available space for the nerve roots (Porter et al 1978). A small trefoil shaped canal places the root at risk from a postero-lateral bulge, but a far lateral disc can cause nerve involvement in the root canal even when the central canal is wide.

Root entrapment with degenerative changes. This can occur in the lateral recess of the central canal, though more commonly root entrapment is more distal in the root canal. The patient experiences quite severe pain in a root distribution, aggravated by extension, and often there are few abnormal neurological signs.

Pathogenesis

A single level central canal stenosis does not produce neurogenic claudication. If a nerve root is compressed by a bulging or sequestered disc, this will produce a classical syndrome of root pain, worse on coughing, root tension signs, and perhaps a trunk list. But even with a massive and perhaps chronic protrusion, the patient does not have claudication symptoms. A slowly expanding tumor within the vertebral canal may produce bizarre symptoms, but not claudication. A single level degenerative process, with hypertrophied facet joints may cause a subtotal occlusion of the canal without claudication. Furthermore, experimentally the cauda equina of a dog can be occluded by 25% of its cross sectional area without causing any neurological deficit (Delmarter et al 1990).

A single level stenosis of the lateral recess or of the root canal or foramen, may cause thickening and inflammation of the nerve root with severe root entrapment pain, but not claudication.

Patients with neurogenic claudication generally have two or more levels of stenosis. There may be two levels of central canal stenosis or a single level of stenosis in the central canal, and a more distal root canal stenosis. The former tends to give bilateral claudication, and the latter claudication in one leg (Porter and Ward 1992).

A single level stenosis probably causes little neurological dysfunction, as the nerves are well supplied with oxygenated blood from a proximal and distal supply. The veins drain distally to the foramen, and proximally to the conus where they anastomose with other veins and drain distally along other roots to their respective foramina (Figure 4). A two level low pressure stenosis however will produce more profound effects. The arterioles will supply the uncompressed segment between the two blocks, but the venous return

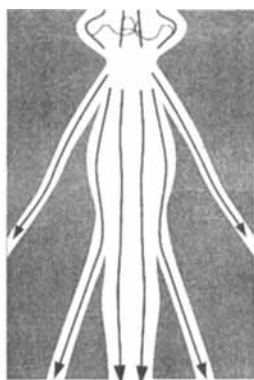


Figure 4. A single level central stenosis causes little vascular impairment.

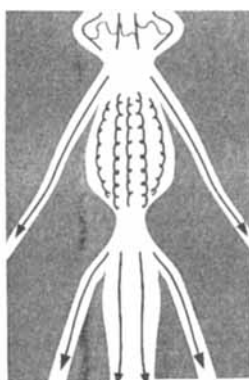


Figure 5. A double level central stenosis at low pressure, results in considerable blood pooling

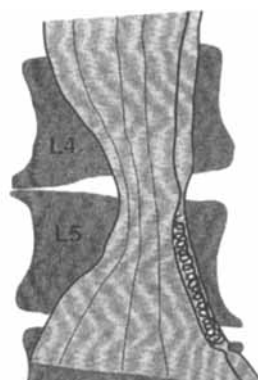


Figure 6. A single central stenosis and a more distal root stenosis causes pooling in the veins of a single root.

will be impaired, and a long segment of cauda equina will become congested. There will be a build up of metabolites, and reduced blood flow with impaired nutrition. In a two level central canal stenosis, all the cauda equina will be congested with bilateral symptoms (Figure 5). When there is a single level central stenosis with a more distal root canal stenosis, only a single root will be congested, with unilateral symptoms (Figure 6).

Forty percent of patients with bilateral claudication have a degenerative spondylolisthesis, with a second level of more proximal stenosis. In unilateral claudication, 50% have a degenerative lumbar scoliosis, with a central stenosis at the apex of the curve and an asymmetrical distal root canal stenosis.

Although spinal tumor does not cause claudication symptoms, McGuire and colleague (1987) reported patients with neurofibromata who had claudication symptoms. In addition to the spinal tumor, they had a previously asymptomatic spinal stenosis.

Exercise is associated with vasodilatation of the cauda equina. If the block pressure of one or both of stenosed segments is just below the venous pressure at rest, then as the cauda equina increases in volume with the dynamics of walking, the pressure can increase to above the venous pressure. This will result in blood pooling, and a reduced flow in the uncompressed segment between the blocks. The patient then experiences claudication symptoms and has to rest until there is a reduction in pressure at the stenosis.

This hypothesis agrees with the clinical and experimental observations of claudication. A single level compression of 10 mmHg in a porcine cauda equina model had little effect on function, whilst a two level compression of 10 mmHg caused marked reduction in blood flow by 64% (Takahashi et al 1992), and significantly reduced protein transport and nerve conduction

(Olmarker et al 1992). A two level compression below arterial pressure is also supported by myelography studies, which show not an ischemic, but a congested cauda equina in claudicating patients (Ooi et al 1990).

Although venous compression is the most acceptable explanation for the pathophysiology of neurogenic claudication, its frequent association with peripheral vascular disease has not been explained. It is possible that neurogenic claudication is not a homogeneous condition but a common expression of a number of different pathologies.

References

- Boden S D, Davis D O, Dina T S, Patronas N J, Weisel S W. Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects. A positive investigation. *J Bone Joint Surg (Am)* 1990; 72A: 403–408.
- Delmarter R B, Bohlman H H, Dodge L D, Biro C. Experimental lumbar spinal stenosis. *J Bone Joint Surg (Am)* 1990; 72A: 110–120.
- McGuire R A, Brown M D, Green B A. Intradural spinal tumours and spinal stenosis. *Spine* 1987; 12: 1062–1066.
- Olmarker K, Rydevik B. Single versus double level nerve root compression. An experimental study on the porcine cauda equina with analyses of nerve impulse conduction properties. *Clinic Orthop* 1992; 279: 35–39.
- Ooi Y, Mita F, Satoh Y. Myeloscopic study on lumbar spinal canal stenosis with special reference to intermittent claudication. *Spine* 1990; 15: 544–549.
- Porter R W. Measurement of the lumbar spinal canal by diagnostic ultrasound. MD Thesis. University of Edinburgh 1980.
- Porter R W, Ward D. Cauda equina dysfunction. The significance of two-level pathology. *Spine* 1992; 17: 9–15.
- Takahashi K, Olmarker K, Holm S, Porter R W, Rydevik B. Double level cauda equina compression: an experimental study with continuous monitoring of intraneural blood flow in the porcine cauda equina. *J Orthopaedic Research*. (In press).