Lumbar spinal stenosis — a current view

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Abstract

This article considers the pathophysiology, diagnosis and management of spinal stenosis. Although spinal stenosis may at times be easily diagnosed, it can also be difficult to identify and the differential diagnosis is discussed. Treatment options ranging from nonoperative management through to the latest experimental surgical treatments are described in this overview of our current thinking on this common spinal condition.

Keywords Spinal stenosis; decompression; spinal stabilisation; cauda equina syndrome; neurogenic claudication

Introduction

Spinal stenosis is a disorder causing neural compression in the spinal canal. It is a common, acquired spinal disorder usually affecting elderly patients, though some patients have congenital narrowing of the bony canal and foraminae of the mobile cervical and lumbar spine which becomes symptomatic in the third or fourth decade.[1–12] An example is the spine of the achondroplastic individual, and some other short-stature syndromes, in which a low Pavlov ratio (spinal canal: vertebral body depth ratio) is demonstrated.[1] Stenosis is caused primarily by disc degeneration occurring adjacent to the narrowed bony canal (itself a result of short pedicles, thickened laminae and hypertrophic facet joints due to osteoarthropathy) and hypertrophic soft tissue (ligamentum flavum). Lumbar spine stenosis (LSS) is classified by its aetiology (Table 1, Figure 1).

The symptoms of lumbar spinal canal stenosis are produced by constriction neuropathy of the involved neural elements, such as the cauda equina and nerve roots. Dynamic factors, such as posture and spinal instability, also participate in the induction of symptoms; further compression as a result of these factors exacerbates the symptoms. The severity of the symptoms, however, is not necessarily associated with the magnitude of the compression as suggested by imaging studies. Vascular impairment is also thought to participate in the generation of symptoms.

Many different terminologies are used in describing the stenosis, for example based on the anatomical site of compression; central canal stenosis, lateral recess stenosis, subarticular stenosis, subpedicular stenosis, lateral gutter stenosis, foraminal stenosis, intervertebral foraminal stenosis. These terms have caused confusion in the literature.

Classification of lumbar spine stenosis

- Congenital or developmental (primary canal narrowing)
  - Idiopathic
  - Achondroplastic
  - Osteopetrosis
- Acquired (secondary canal narrowing)
  - Degenerative
    - Central
    - Lateral recess and foraminal
    - Degenerative spondylolisthesis
  - Iatrogenic
    - After laminectomy
    - After arthrodesis
    - After discectomy
  - Miscellaneous disorders
    - Acromegaly
    - Paget disease
    - Fluorosis
    - Ankylosing spondylitis
  - Traumatic
- Combined: any combination of congenital, developmental, or acquired stenosis

Table 1

Regional anatomy and movements

The ranges of the movement seen in the lumbar spine are presented in Table 2. The lumbar spinal canal can be divided into two functional components; static and dynamic. The static segment is the osseous canal between the discs, while the dynamic (mobile) segment corresponds to the distribution of the ligamentum flavum (Figure 2). The segments which cause symptoms of stenosis are the dynamic segments, described by the level of the spinal root associated with symptoms.

The cauda equina is surrounded anteriorly by disc, posterior longitudinal ligament and vertebral bodies, and laterally by the pedicles and the lateral extensions of the ligamentum flavum. The ligamentum flavum, laminae and the facet joints form the posterior constraints (Figure 3).

Each spinal nerve root canal (neural foramen) is bounded anteriorly by the disc and adjacent vertebral bodies, posteriorly by the facet joints and above and below by the pedicles.

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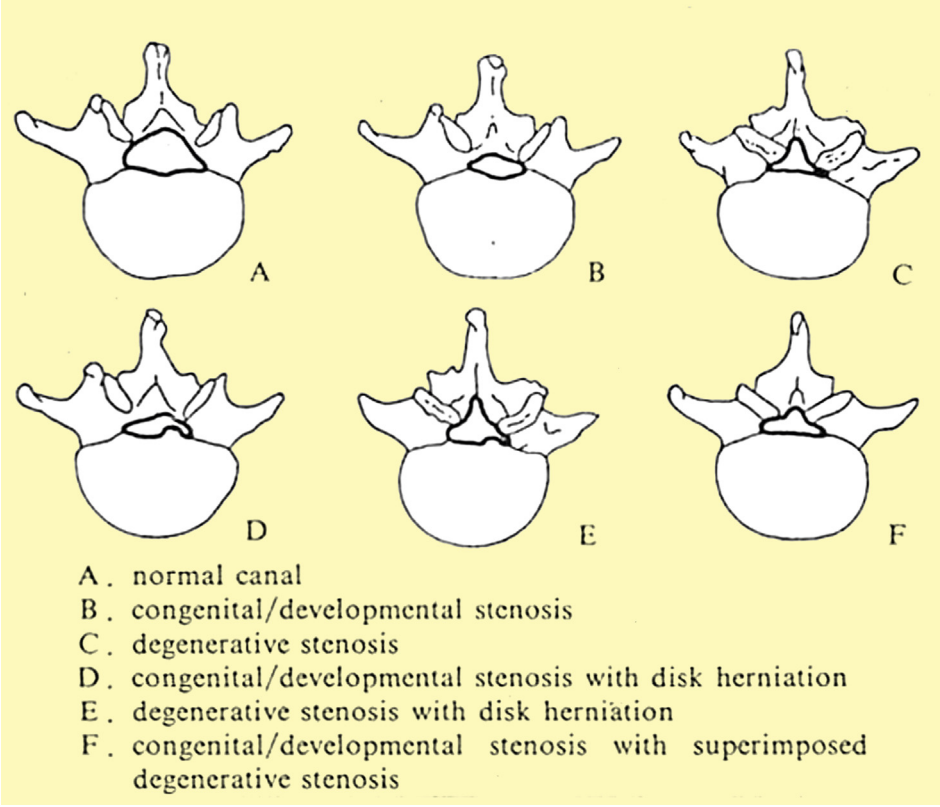

Figure 1 Various types of spinal canal stenosis as described by Arnold et al. in 1976.

A mean A-P diameter of the spinal canal of 12 mm and minimum cross-sectional area of at least 77 ± 13 mm^2 are essential to accommodate the average-sized neural elements.

The normal intervertebral foraminal height is 16–19 mm and the foraminal cross-sectional area is 40–160 mm^2. A height ≤15 mm is defined to be pathologic.

According to Mayoux-Benhamon et al., 1989, foraminal width is a minimum of 5∼6 mm, and superiorly the width is 7∼8 mm. Forward flexion increases foraminal area by 12% (30% of foraminal width) whilst there is a 15% decrease (narrowing) in extension. However, there is more leeway for the nerve roots and spinal nerves in the lumbar foramina than in the cervical area. The reason for this is that the ratio of the size of nerve root to the foramen in the cervical spine is 1:2, while in the lumbar spine it is 1:5 (30% difference reported by Jenis and An; 10–30% by Hayland et al).

Movement in the lumbar spine

Disc level	ROM (degrees)			Remarks
	Begg & Falconer	Allbrook	Louis R	
L_1 (L_{1-2})	10°	6°	11°	
L_2 (L_{2-3})	12°	8°	12°	
L_3 (L_{3-4})	14°	13°	18°	
L_4 (L_{4-5})	15°	19°	24°	Hallinshead $L_4 > L_5$
L_5 ($L_5.S_1$)	15°	18°	18°	
Total	60°	64°	83° (30°/53°)	Forward: 3 Backward: 1
• Ratio of lateral bending: forward-backward bending; 2:3				
• Total axial rotation: 16° (Rt/Lt: 8°/8°)			short period or long period	5–6°
• Rotation at L_5-S_1			frequency of compression	1.5° in normal walking
• (30°/50°): extension/flexion				
• Lateral bending: 40° (Rt/Lt: 20°/20°)				

Table 2

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