# Clinical Article

# Lumbar spinal stenosis

JA Shipley MMed(Orth)
Department Orthopaedic Surgery, University of the Free State, Bloemfontein

Reprint requests:
Prof JA Shipley MMed(Orth)
Department Orthopaedic Surgery
University of the Free State
Bloemfontein
Tel: (051) 405-2242
Fax: (051) 430-7101
legraner@fshealth.gov.za

## Abstract:

Lumbar spinal stenosis is diagnosed by the clinical picture of radiculopathy and / or spinal claudication caused by narrowing of the spinal canal. Anatomical and radiological features correlate poorly with the clinical features, and are of little prognostic value. The natural history is very benign with fluctuating levels and types of symptoms. Indications for surgery must be individualized, but decompression is effective in the short and medium term. Fusion is only indicated in the case of instability.

## Introduction

In 1954, Verbiest[1] reported seven cases of what came to be called spinal stenosis, patients with "a radicular compression syndrome, from developmental narrowing of the lumbar vertebral canal". The patients had a typical clinical picture, including claudication, and the diagnosis was confirmed by myelography.

Although certain aspects have changed, the basic concepts remain of a clinical syndrome, diagnosed mainly on the history, often with minimal physical signs, and confirmed by radiology. Current interest centres on more accurate diagnosis, and selection for treatment.

**Table I: Aetiology (Arnoldi *et al*)[2]**

- Congenital/developmental
- Acquired
  - Degenerative
  - Combined degenerative and developmental
  - Spondylolisthetic
  - Iatrogenic
  - Post traumatic
  - Miscellaneous, e.g. Paget's disease of bone

## Classification

Arnoldi and co-workers[2] proposed an aetiological classification *(Table I)*, essentially dividing causes into congenital/developmental and acquired. With time, the acquired causes have been accepted as far more common and important than congenital/developmental causes.

The most common classification of the anatomical area of stenosis is shown in *Figures 1a & b*:

- Central stenosis occurs in the canal medial to the facets, before the roots have separated from the dura.
- Lateral recess (subarticular) stenosis occurs after separation of the nerve roots, in the area anterior to the facet, medial to the pedicle and bounded anteriorly by disc/ vertebral body.
- Foraminal stenosis occurs in the region between medial and lateral aspects of the pedicles.
- Extraforaminal stenosis occurs lateral to the foramen

**Definition: Lumbar spinal stenosis – narrowing of the spinal canal producing symptoms of radiculopathy or claudication**

An alternative classification into central and lateral stenosis, with lateral stenosis subdivided into inlet, midzone (corresponding to the lateral recess) and outlet zones (corresponding to the foraminal region) is favoured by some authors.[3]

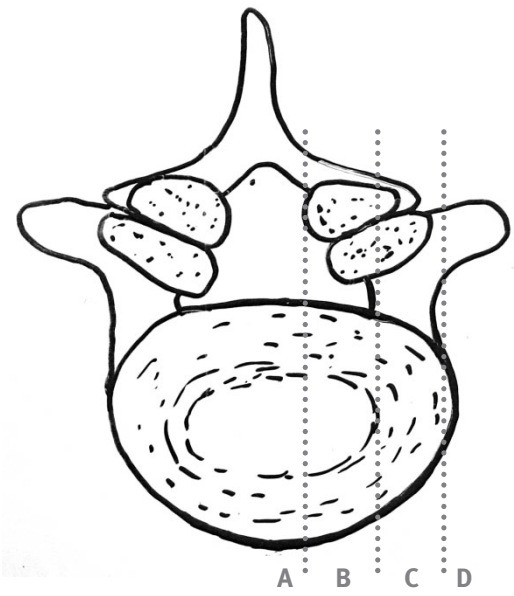


**Figure 1a**

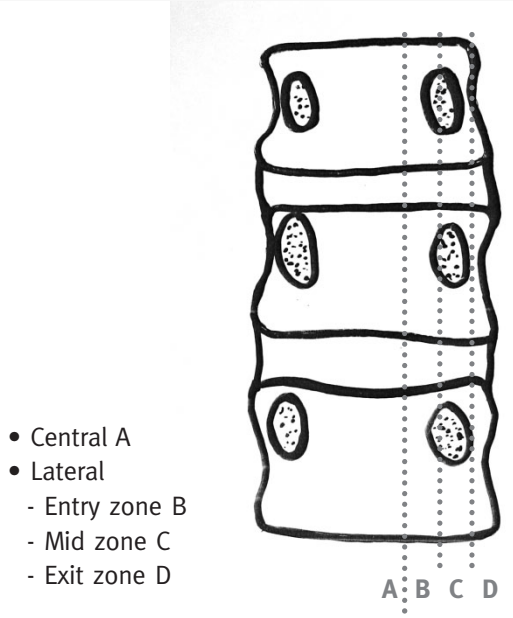


**Figure 1b**

## Pathogenesis

Congenital stenosis is uncommon, and limited to cases such as achondroplasia, where a skeletal dysplasia results in congenitally short pedicles and a shallow canal. Developmental stenosis may occur due to a trefoil-shaped canal, which has a reduced cross-sectional area, and predisposes to some additional factor such as disc disease increasing the stenosis.

Degenerative stenosis occurs when:

- a degenerating disc bulges or herniates into the canal
- the facets form osteophytes, and the capsule thickens with synovitis and effusion, intruding into the lateral recess and foramen
- the ligamentum flavum degenerates, becoming thickened and inelastic, and bulging into the canal especially during extension
- the foramen narrows vertically when the disc collapses, and the vertebrae telescope onto each other. This causes further bulging of annulus and ligamentum flavum, and over-riding of laminae and facets to increase central and lateral recess stenosis.

Instability is often a major factor in degenerative stenosis. With degenerative spondylolisthesis, anterior subluxation of the superior vertebra's lamina and facets causes narrowing in the central and lateral recess regions of the canal respectively *(Figure 2)*. The foramen becomes distorted from the normal vertical oval shape to a figure-of-eight or keyhole shape, and the exiting nerve root becomes trapped between the pedicle of the subluxed vertebra above, and the disc below *(Figure 3)*.

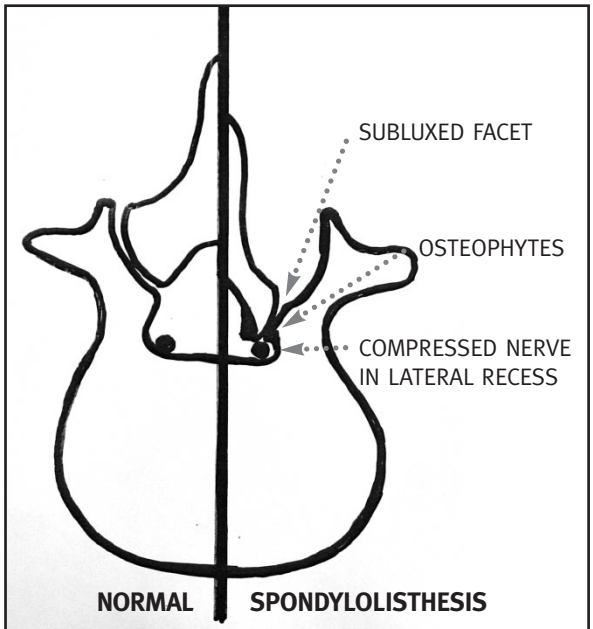


**Figure 2**

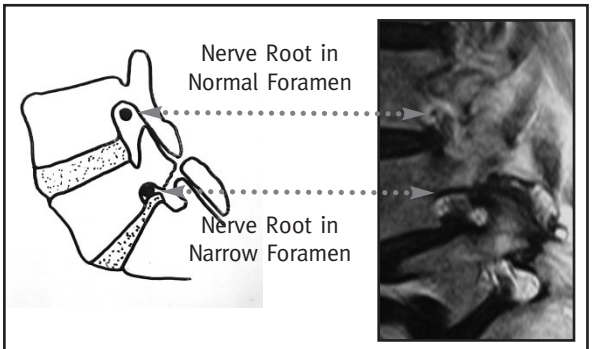


**Figure 3**

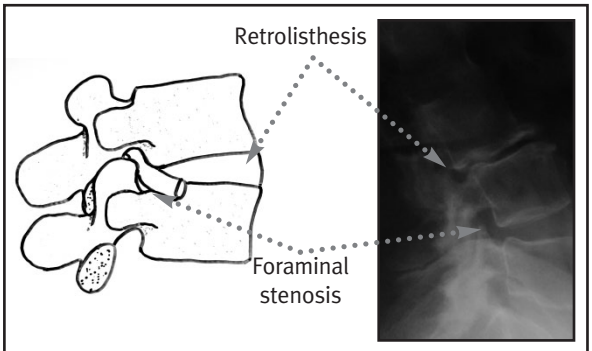


**Figure 4**

**Table II: Neurogenic versus vascular claudication**

| Finding | Neurogenic | Vascular |
|---|---|---|
| Symptoms on standing | Yes | No |
| Symptoms on walking | Yes | Yes |
| Fixed claudication distance | No | Yes |
| Relief on flexion spine | Yes | No |
| Relief on rest sitting | Yes | Yes |
| Relief on rest standing | No | Yes |
| Loss of pulses/ischaemia | No | Yes |
| Onset proximal before distal muscles | Yes | No |

In retrolisthesis, the superior facet of the lower vertebral body subluxes anteriorly causing foraminal narrowing, and may compress the nerve root against the vertebral body or disc *(Figure 4)*.

Degenerative scoliosis may lead to foraminal and lateral recess stenosis in the concavity of the deformity. Where rotation occurs, facet subluxation with intrusion into the foramen may contribute to stenosis.

Extraforaminal stenosis may occur when disc osteophytes compress a root lateral to the foramen, or the root is trapped between the ala of the sacrum and L5 osteophytes or transverse process.

The cause of neurological symptoms is poorly understood, but probably combinations of direct nerve root compression, mechanical irritation, ischaemia and venous congestion are responsible.

Generally, central stenosis causes neurogenic claudication, while lateral recess or foraminal stenosis is more likely to present with a radiculopathy, often painful.

The compression is dynamic, and therefore related to posture, so typically the patient can modulate his symptoms by flexing the spine and opening the canal slightly.

## Natural history

Few studies of the natural history are available, most studies being a comparison of conservative treatment with surgery. It appears that the majority of patients (55-70%) will remain largely unchanged, a minority (15-30%) will deteriorate gradually, and a few (10-15%) will improve.[4,5,6]

No acute deterioration occurs unless some additional acute pathology, e.g. disc herniation, facet cyst, suddenly causes further narrowing of the canal.

In the only controlled study to date, comparing asymptomatic and pure low back pain patients to those with spinal stenosis, Haig *et al*[7] found that patients moved between groups with time, and the general tendency was to improvement. The prognosis was unrelated to the size of the spinal canal, neurological deficit or severity of pain at presentation.

## Clinical picture

The majority of patients present in the sixth or seventh decade, in keeping with a degenerative aetiology. Developmental cases usually become symptomatic in the third or fourth decade. Women are affected three to five times more frequently than men. The L3/4 and L4/5 levels are most commonly involved.

About two-thirds of patients with degenerative stenosis, especially with instability, present with mechanical lumbar pain and this must be evaluated and treated along with the spinal stenosis.

The classical complaint in lumbar stenosis is neurogenic (or spinal) claudication, caused by compression of the cauda equina, in the central region of the spinal canal. The patient's legs become numb and weak (often described as "lame") after standing or walking for a time. Symptoms are reduced by flexing the spine e.g. walking with a stick, or pushing a trolley, and aggravated by extension e.g. working overhead. They may be present when lying on the back, with the lumbar spine in lordosis *(Table II)*.

Radicular pain is the main problem in some four out of five cases, and often indicates lateral recess stenosis. Aggravating and relieving factors are similar to those described for claudication. Urinary symptoms are commonly present, but in this ageing population, they are often caused by urinary tract rather than spinal pathology.

It is important to realise that many elderly patients have similar symptoms to spinal stenosis, without demonstrable spinal pathology. Conversely, many patients with severe radiological stenosis are asymptomatic.

Physical examination is often disappointing, with findings largely limited to the spine, rather than the nervous system. In both instances it is frequently difficult to distinguish significant findings from ageing. Approximately half will have neurological abnormalities such as weak or asymmetrical tendon reflexes, slight motor or sensory loss (usually L4 or L5), and occasionally positive sciatic and/or femoral stretch tests. The most useful test is to extend the lumbar spine for a period of 30-60 seconds, in an attempt to elicit the typical leg symptoms. If symptoms are unilateral, extension may be combined with lateral flexion. The patient may also be made to walk until symptoms develop, and then re-examined neurologically to detect a deterioration. Patients are commonly more tolerant of exercise on a bicycle than a treadmill.

**Any scheme of management must take into account the essentially benign, fluctuating natural history of this condition, and both doctor and patient must understand this**

A careful vascular examination, including the abdomen, is essential to exclude vascular claudication.

Hip degeneration may cause similar symptoms to nerve root compression, and often co-exists in the age group at risk for spinal stenosis. If there is a hip flexion contracture, which increases lumbar lordosis, it may exacerbate the effect of co-existing spinal stenosis. The differential diagnosis also includes knee pathology, peripheral neuropathy, spinal tumours and infections, and other neurological conditions.

## Radiology

Plain radiographs are always necessary to assess for anatomical anomalies, degeneration, deformity and instability, all of which may influence management. Erect P-A, lateral, and often flexion/extension views are required.

MRI imaging, using sagittal and axial cuts, is the most informative investigation, as it shows the soft tissue as well as bone pathology, and allows direct assessment of the foramina, lateral recesses and nerve roots. MRI allows multiple levels to be examined with a single investigation, without irradiation or the risk of myelography, unlike CT. The images must be examined for:

- pressure on the dural sac, loss of CSF, crowding of the roots of the cauda equina and the redundant nerve root sign
- thickening of the ligamentum flavum and facet capsules
- degeneration and subluxation of the facets
- bulging or herniation of the disc
- space available around the nerve root in the foramen

Computerised tomography alone is of limited value, but combined with myelography, provides good visualisation of central and lateral recess stenosis, although the different causes of compression may be difficult to differentiate. The foramina and extra-foraminal regions are poorly demonstrated. The only advantage over MRI is that myelography allows dynamic studies to show stenosis in different spinal positions.

Dynamic MRI and CT technology is being developed to refine the imaging of stenotic spines.

Various radiological measurements have been suggested to assess spinal stenosis *(Table III)*; **the fact is that as yet there is very poor correlation between them and the presence of symptoms**.[8] Haig *et al*[7] note that asymptomatic radiological spinal stenosis is present in up to 85% of the older population.

The role of radiology is to support a clinical diagnosis by confirming the presence of stenosis, and to demonstrate which levels are affected, and what pathology is causing nerve compression. Radiology cannot be the only factor in diagnosis or decision-making. Radiology does not tell the surgeon when to operate, it only gives guidance to what is required and where.

**Table III: Radiological measurements of spinal stenosis**

| | |
|---|---|
| **Antero-posterior canal diameter** | |
| | • <12 mm; relative stenosis |
| | • <10 mm; absolute stenosis |
| **Dural sac cross-sectional area** | |
| | • <100 $mm^2$; relative stenosis |
| | • <75 $mm^2$; absolute stenosis |
| **Antero-posterior size lateral recess** | |
| | • <4 mm |

### Electrodiagnosis

Electromyography is often abnormal in spinal stenosis, but changes are neither diagnostic nor of prognostic value.[7]

### Conservative management

Any scheme of management must take into account the essentially benign, fluctuating natural history of this condition, and both doctor and patient must understand this. A conscientious trial of conservative treatment is indicated, with only very rare exceptions, before progressing to surgery. Delay in surgery does not compromise the final result of an operation.[9]

Conservative measures should include improvement in general fitness, weight reduction and abdominal muscle strengthening to reduce the lumbar lordosis. A lumbar corset to reduce lordosis may be used for short periods when the patient needs to be active, e.g. for shopping, housework.

Medication with analgesics and NSAIDs, with the addition of low dose amitriptyline or gabapentin at night may help to reduce mechanical and nerve pain.

Epidural steroid injections provide short-term relief (but not long-term), and may be of use in an acute flare-up of symptoms, or high-risk cases. They also help confirm the diagnosis, and predict the result of surgery. The caudal route is easiest in cases of severe degeneration.

Calcitonin has been shown to be beneficial in controlled trials, possibly due to an anti-inflammatory effect. Its use appears logical in patients with underlying osteoporosis or Paget's disease.

## Surgical management

The indication for surgery is unacceptable pain or limitation of function. It is therefore subjective, and varies according to patient expectations and lifestyle. There is no indication for decompression in the patient with radiological stenosis, but with no symptoms of stenosis, or with back pain only. The risk of not treating spinal stenosis is minimal, while that of operating on an elderly patient with other health problems is unpredictable and sometimes substantial.

**While the benefits of decompression are recognised, those of fusion (especially instrumented fusion) are still disputed**

There are two components of an operation to be considered: decompression and fusion. Obviously the primary aim of surgery is to create space for the neural elements by decompression. In certain circumstances *(Table IV),* fusion may also be considered, the main indication being instability of the decompressed segment.

A detailed description of decompression technique is beyond this article, but the principles are:

- Decompress all stenotic areas and levels, paying specific attention to the foramen, which is the commonest site of inadequate decompression.
- Wide laminectomy should be performed where a stable spine is anticipated (narrowed disc with osteophytes) or where fusion will be performed. Otherwise laminotomies should be performed.
- Preserve as much of the facet as possible (at least 50%), to reduce the risk of iatrogenic instability. Care must be taken in the region of the pars interarticularis, because excessive bone removal may weaken this area, and cause a facet fracture, with later instability.
- Discectomy should be avoided as far as possible, and reserved for overt herniation.

Decompression has been found to provide long-term relief of stenotic symptoms in two out of three patients, although the results decline with time. The biggest cause of dissatisfaction is persistent back pain, but there is no evidence that fusion improves results in the absence of instability.[1,10,11]

In the first randomised controlled trial reported, both surgical and non-surgical patients improved over 2 years, but the surgical patients improve significantly more regarding leg and back pain as well as disability.[12] This benefit decreased with time.

Instability after decompression is uncommon, unless it existed beforehand. A well-preserved high disc may collapse with time and lead to instability or re-stenosis.

Postacchini[13] reported a 7% incidence of re-stenosis due to bone regrowth after decompression, and linked this to instability and limited decompression of the affected segment.

While the benefits of decompression are recognised, those of fusion (especially instrumented fusion) are still disputed. The only hard indication for fusion is instability, the others being speculative and unproven.

Results of decompression of degenerative spondylolisthesis are certainly improved by fusion.[14] Fusing only the unstable level is recommended, even if multiple levels are decompressed, provided there is no additional iatrogenic instability.[11,14]

Although instrumentation considerably increases the fusion rate (from 45% to 83%) in these patients, Fischgrund[15] found no improvement in clinical outcomes at 2 years. However, longer follow-up of the same group of patients showed a dramatic decline in good results if surgery ended in a pseudarthrosis, and it is currently recommended that fusion for instability should be augmented by instrumentation.[16,17] Anterior-posterior (360°, circumferential) fusion has the possible advantage of indirect foraminal decompression by distraction of the vertebral bodies, in addition to a better fusion rate than postero-lateral fusion alone.

**Table IV: Indications for fusion**

**Proven**
- instability
- degenerative spondylolisthesis
- scoliosis
- iatrogenic

**Unproven**
- chronic back pain
- indirect foraminal decompression with interbody fusion
- high disc
- recurrent stenosis

**Table V: Indications for fusion after decompression in degenerative scoliosis**

- Instability: roto/spondylo/retrolisthesis or iatrogenic instability
- Flexible curve which may progress
- Loss of lordosis with possible sagittal decompensation
- Wide laminectomy required
- Significant mechanical pain

Many cases of degenerative lumbar scoliosis can be treated by decompression alone *(see Table V)*. Multiple level laminotomies may be safely performed on one side without fusion, but have a risk of instability and progression of deformity if performed bilaterally.

A number of non-fusion devices have been propagated in recent years.

Artificial ligaments fixed by pedicle screws, and used to distract or compress the posterior vertebral elements have been used in spinal stenosis. It seems likely that the major improvement in symptoms comes from the decompression performed in most cases, rather than any change in spinal mechanics. Their use is not recommended.

Interspinous spacers can be used to distract the laminae at stenotic levels, relieving symptoms by creating a localised kyphosis. They have a significant failure rate in the osteoporotic spine, and should be reserved for high-risk patients, where there is no alternative and the procedure may be performed rapidly under local anaesthetic.

## Summary

We now have a much better understanding of the pathogenesis and natural history of lumbar spinal stenosis than when it was originally described. Unfortunately there is still uncertainty about anatomical and radiological definitions due to their poor correlation with clinical features of the syndrome. The diagnosis remains a clinical one, supported by significant radiological stenosis.

The disparity between clinical and radiological findings, and their poor prognostic value, makes management difficult. All patients should be offered a trial of conservative treatment, because the natural history is so overwhelmingly benign. Indications for surgery are essentially severe pain and limitation of function, so each patient must be individualised. Decompression is the main objective, and is effective. Fusion improves results if the spine is unstable, but is otherwise of no proven value. Interspinous spacers may help the high-risk patient, but have a high failure rate.

*No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article. This article is free of plagiarism.*

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