Focus On
Understanding and treating spinal stenosis

Introduction
Lumbar spinal stenosis is narrowing of the central spinal canal, lateral recess or the neural foramen. After initial recognition of spinal stenosis in 1802, a gradual understanding evolved over the next 150 years.1-4 Harris and Macnab described the importance of disc degeneration in the pathogenesis of stenosis.5 Macnab highlighted the lateral recess beneath the posterior facet joint.6 Verbiest identified neurogenic claudication as a result of spinal canal stenosis. 7

Anatomical considerations
The shape of the lumbar spinal canal varies and may be an oval, rounded triangular or trefoil configuration.8 The trefoil configuration usually occurs at the fifth lumbar level, making L4-L5 the narrowest level 9 (Fig. 1), and occurs in 25% of the population, only appearing in adulthood.10 The anteroposterior diameter of the lumbar spinal canal is critical in the pathogenesis and is affected by the length of the pedicles.11

Classification
Lumbar spinal stenosis is classified according to its aetiology (Table I).11 Postacchini divided the stenosis into primary (congenital), secondary (acquired) or combined forms.12

The congenital type is rare. There are anatomical abnormalities such as short pedicles in achondroplasia. Other causes include hypochondroplasia, diastrophic dwarfism, Morquio’s syndrome, hereditary exostosis and cheirolumbar dysostosis. The majority of patients present with acquired stenosis because of degeneration in the fifth to seventh decades.

Anatomically, spinal stenosis can be central, within the lateral recess, or in the foramen. Central canal stenosis occurs at the level of the intervertebral disc with midline sagittal narrowing. Lateral recess stenosis occurs when the traversing nerve root is compressed beneath the superior articular process of the inferior vertebra, i.e. beneath the thickened facet joint.

Hansraj et al divided spinal stenosis into typical and complex lumbar spinal stenosis (Table II).13,14 Typical cases were treated with decompressive surgery, while complex stenosis required decompression and fusion.

Pathogenesis
Kirkaldy-Willis et al described the sequence of degenerative changes which result in central or lateral recess stenosis.15

Central canal stenosis (Fig. 2) is mainly created by hypertrophy of the ligamentum flavum, facet joint osteophyte formation and degenerative spondylolisthesis (Fig. 3).

<table>
<thead>
<tr>
<th>Table I.</th>
<th>Aetiological classification of lumbar spinal stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital</td>
<td></td>
</tr>
<tr>
<td>Idiopathic</td>
<td></td>
</tr>
<tr>
<td>Achondroplastic</td>
<td></td>
</tr>
<tr>
<td>Acquired</td>
<td></td>
</tr>
<tr>
<td>Degenerative</td>
<td></td>
</tr>
<tr>
<td>Iatrogenic – post-surgical</td>
<td></td>
</tr>
<tr>
<td>Metabolic – Paget’s disease, fluorosis</td>
<td></td>
</tr>
<tr>
<td>Post-traumatic</td>
<td></td>
</tr>
<tr>
<td>Stenosis due to spondylolisthesis</td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td></td>
</tr>
<tr>
<td>Congenital with secondary degenerative changes</td>
<td></td>
</tr>
</tbody>
</table>

©2010 British Editorial Society of Bone and Joint Surgery
Lateral recess stenosis results from compression between the medial aspect of a hypertrophic superior articular facet and posterior aspect of the vertebral body and disc (Fig. 4). Hyper trophy of the ligamentum flavum and/or facet joint capsule, vertebral body osteophyte and disc protrusion exacerbate the stenosis. The traversing nerve root is compressed in the lateral recess (i.e. the L5 nerve root in the L4-L5 lateral recess).

Foraminal stenosis is rare and mainly occurs in isthmic spondylolisthesis, where the exiting nerve root (i.e. the L5 nerve root in L5-S1 isthmic spondylolisthesis) is compressed in the distorted foramen. It also occurs in far lateral disc herniation where the exiting nerve root is compressed in the foramen, or extra-foraminal zone (Fig. 5).

Lateral recess and foraminal stenosis are distinct entities with different clinical implications.\(^{16}\)

Natural history
The course of spinal stenosis is chronic and benign. Johnsson, Rosen and Uden reported on 32 patients followed up for a mean of 49 months (10 to 103) without any treatment.\(^{17}\) Of the 32 patients, 15% improved, 70% stayed the same and only 15% became worse. The majority of patients followed up for four years thus remained unchanged.

Despite a benign natural history, the long term is characterised by slow deterioration. A randomised study by Amundsen et al compared surgical and conservative treatment with a ten-year follow-up.\(^{18}\) The outcome was more favourable for surgical treatment, but an initial conservative approach was recommended as late treatment still achieved a good result.

Clinical features
History. The symptoms are insidious, presenting in the sixth or seventh decade. There may be a long history of low back pain but the leg symptoms lead to presentation.

The patient with central canal stenosis has bilateral leg symptoms which are vague and often described as heaviness, soreness or weakness. The cardinal symptom is neurogenic claudication which presents as numbness, weakness or discomfort in the legs; this may come on with walking or prolonged
standing and is relieved by sitting or rest. The patients are able to walk further when leaning on a shopping trolley or walking uphill because spinal flexion increases the space available for the cauda equina and unfolds the ligamentum flavum. The pain-free walking distance may vary.

Patients with lateral recess stenosis present with unilateral radicular symptoms of leg pain along with numbness, paresthesiae or burning in a dermatomal distribution. Pain radiates from the buttock to the posterior thigh and lateral calf because most compression occurs at L4-L5. If there is a higher lumbar lesion then anterior thigh pain will be present. The symptoms may also be provoked by walking.

Cauda equina syndrome or major neurological deficits are very rare in the presence of canal stenosis.

Examination. Physical findings are limited. The diagnosis is made from the history and confirmed with imaging. There may be limitation of lumbar spinal extension, sensory deficit, muscle weakness, limited straight leg raise, absent knee reflexes and absent ankle reflexes. Neurological abnormalities are uncommon at rest. The “Shuttle walking test” has been suggested as a reliable objective assessment of walking distance. Loss of distal vibration sense may be present but is common in older patients anyway. Signs of cervical myelopathy may be detected, as lumbar spinal stenosis is associated with cervical canal narrowing in 5% of the patients (Tandem stenosis).

Differential diagnosis

Peripheral Vascular Disease. Table III shows the differences between neurogenic and vascular claudication

Hip Osteoarthritis. Anterior thigh pain can be present in hip arthritis as well as in spinal stenosis with involvement of the L3 or L4 nerve roots. However, pain on passive rotation of the hip should alert the clinician to the possibility of hip disease. The two conditions can coexist, so nerve root blocks and/or hip joint injections with local anaesthetic and steroid may be necessary to differentiate between them.

Peripheral Neuropathy. This diagnosis is determined more by neurological symptoms than pain, and produces a stocking pattern of numbness. Electrophysiological testing confirms the diagnosis.

Trochanteric Bursitis. Sometimes spinal stenosis patients are diagnosed as having trochanteric bursitis. Some have suggested a predisposition to this condition because of an “altered pelvic balance”.

Investigation

Radiographs. Plain radiographs of the lumbosacral spine may show narrowing of the disc space, facet joint osteoarthritis, degenerative spondylolisthesis or degenerative scoliosis. Radiographs are also useful to exclude conditions such as tumour or infection. Dynamic instability can be demonstrated by lateral radiographs taken in the standing position with spinal flexion and extension.

Computed Tomography (CT) Myelography. CT or CT myelography is useful in patients who have a contraindication to magnetic resonance imaging (MRI) scan (pacemaker, metallic implants, etc.). On CT, midsagittal lumbar canal diameters of less than 10 mm are regarded as an absolute stenosis while less than 13 mm represents relative stenosis.

MRI. MRI is the investigation of choice in confirming the diagnosis of spinal canal stenosis. Standard MRI examination includes T1- and T2-weighted sagittal and axial sequences. The typical findings in central or lateral recess stenosis include thickening of the ligamentum flavum, facet joint hypertrophy and synovial cysts, a trefoil appearance of the thecal sac, vertebral endplate osteophytes and obliteration of perineural fat in the neural foramina.

Parasagittal images can overdiagnose the incidence of foraminal stenosis. We recommend additional T2-weighted coronal and Short T1 Inverted Recovery (STIR) sequences which demonstrate the nerve roots very well and confirm that the incidence of foraminal stenosis is low in degenerative spondylolisthesis or spinal stenosis, but exists mainly in isthmic spondylolisthesis or extraforaminal disc protrusion.

Neurophysiology. Peripheral neuropathy is a frequent concomitant finding or differential diagnosis in elderly patients with spinal stenosis. Neurophysiological assessments including electromyography and nerve conduction studies are useful in identifying peripheral neuropathy and in differentiating between radiculopathy and mononeuropathy (e.g. femoral neuropathy in diabetics).
Management

Conservative treatment. Analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), weight loss and physiotherapy are used in the management of patients with spinal stenosis. Porter and Hibbert reported that nasal calcitonin improved the symptoms of neurogenic claudication but a further randomised controlled study in 2004 showed no benefit. There is conflicting evidence in the literature about the use of epidural steroids. However, they can provide temporary relief of symptoms. Nerve root blocks are useful in treating radicular pain due to lateral recess stenosis.

Surgical treatment. Surgery is indicated when conservative measures fail and if moderate to severe leg symptoms interfere with the patient’s lifestyle. While these indications remain relative, progressive neurological deficit or cauda equina syndrome represent absolute indications for surgery.

The greater the degree of compression, the better the outcome of surgery. While the primary objective of surgery is to relieve the patient’s leg symptoms, it is important to consider the mechanical back pain and/or instability before deciding on the type of surgical procedure.

The surgical options include:

- Decompressive laminotomy and partial facetectomy
- Decompressive laminectomy and partial facetectomy
- Micro-decompression
- Decompression and non-instrumented fusion
- Decompression and instrumented fusion
- Decompression and flexible stabilisation
- Inter-spinous spacer devices.

Decompressive Laminotomy and Laminectomy. The aim of decompression is to remove the pressure on the cauda equina and the individual nerve roots. The standard wide midline decompressive laminectomy involves the removal of the spinous processes, laminae, ligamentum flavum, medial part of the facet joints and the facet capsule at all involved vertebral levels. However, the potential for segmental instability has led to more conservative operations.

The popular technique of multiple laminotomies may preserve segmental spinal stability, but Postacchini et al. found that patients who had laminotomies had less back pain but a slightly higher incidence of nerve damage. Laminctomy was found to be better for tight stenosis, as it allowed safer neural decompres-
• Necessity for wide decompression with facet joint removal
• Recurrent spinal stenosis

Herkowitz and Kurz prospectively compared decompression alone with decompression and fusion in 50 patients with spinal stenosis and degenerative spondylolisthesis with a follow-up of three years. The results were satisfactory (excellent or good) in 44% of the unfused and 96% in the fused group. Fischgrund et al prospectively randomised 76 patients and compared intertransverse non-instrumented fusion with pedicle screw fusion. Excellent or good results were obtained in 82% of the intertransverse group (45% fusion rate) and 76% of the pedicle screw group (82% fusion rate). The authors concluded that the use of instrumentation leads to a higher rate of fusion but there was no statistically significant clinical difference.

Kornblum et al later showed that the patients with a solid fusion had improved long-term clinical results after five to 14 years.

We can thus conclude that an instrumented fusion should be performed in addition to decompression in the management of degenerative spondylolisthesis. *Decompression and flexible stabilisation.* In patients with spinal stenosis who have segmental instability or degenerative spondylolisthesis, spinal decompression and stabilisation with a flexible or dynamic stabilisation system (e.g. Dynesys), is a controversial option, but Schnake, Schraen and Jeanneret found clinical results similar to decompression and fusion with pedicle screws. *Interspinous spacer devices.* Interspinous spacer devices are inserted between the spinous processes to prevent extension of the symptomatic levels, but allow flexion, axial rotation and lateral bending.

Biomechanical studies have shown that in extension the implants increase the dimensions of the spinal canal. There are some favourable reports of outcome with interspinous devices, however, others report high failure rates for these implants and the need for further surgery. The evidence is thus still inconclusive. Interspinous spacer devices may have a place in elderly patients who are unfit for anaesthesia and more major surgery.

**Degenerative scoliosis**

Spinal stenosis associated with degenerative scoliosis is a complex problem and its treatment is controversial. These patients have multilevel disease with varying combinations of central, lateral recess or foraminal stenosis. They have a mixed pattern of symptoms, which include stenotic, radicular and arthritic pain (Fig. 7). Nerve root compression is present more often on the concave side of the curve. True foraminal compression can occur due to lateral disc herniation, collapse of the disc and scoliosis. The incidence of complications is high after surgery as these are patients of advanced age who may have multiple medical comorbidities. Two types of deformity have been described. *Type I* is a lumbar scoliosis with no or minimal rota-
tional deformity. Type II curves have degeneration superimposed on a pre-existing scoliosis with greater rotational deformity and loss of lordosis. Shorter segmental instrumentation is usually possible for type I deformities while longer instrumentation with sagittal plane reconstitution is necessary for type II curves (Fig. 8).\(^\text{5,2}\) The choice of surgical technique remains controversial but most would agree that decompression and instrumented fusion is necessary for a successful outcome.\(^\text{13}\)

Tricky S Rajagopal FRCS, Robert W Marshall FRCS
Department of Orthopaedic Surgery, Royal Berkshire Hospital, Reading RG1 5AN, UK.

Corresponding author: R. W. Marshall, e-mail: robmarshall100@hotmail.com

References


