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Article *in* Nature Reviews Neurology · August 2009

DOI: 10.1038/nrneurol.2009.90 · Source: PubMed

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Lumbar spinal stenosis: syndrome, diagnostics and treatment

Eberhard Siebert, Harald Prüss, Randolf Klingebiel, Vieri Failli, Karl M. Einhäupl and Jan M. Schwab

Abstract | Lumbar spinal stenosis (LSS) comprises narrowing of the spinal canal with subsequent neural compression, and is frequently associated with symptoms of neurogenic claudication. To establish a diagnosis of LSS, clinical history, physical examination results and radiological changes all need to be considered. Patients who exhibit mild to moderate symptoms of LSS should undergo multimodal conservative treatment, such as patient education, pain medication, delordosing physiotherapy and epidural injections. In patients with severe symptoms, surgery is indicated if conservative treatment proves ineffective after 3–6 months. Clinically relevant motor deficits or symptoms of cauda equina syndrome remain absolute indications for surgery. The first randomized, prospective studies have provided class I–II evidence that supports a more rapid and profound decline of LSS symptoms after decompressive surgery than with conservative therapy. In the absence of a valid paraclinical diagnostic marker, however, more evidence-based data are needed to identify those patients for whom the benefit of surgery would outweigh the risk of developing complications. In this Review, we briefly survey the underlying pathophysiology and clinical appearance of LSS, and explore the available diagnostic and therapeutic options, with particular emphasis on neuroradiological findings and outcome predictors.

Siebert, E. *et al. Nat. Rev. Neurol.* **5**, 392–403 (2009); doi:10.1038/nrneurol.2009.90

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Competing interests
The authors, the Journal Editor H. Wood and the CME questions author C. P. Vega declare no competing interests.

Introduction

The term lumbar spinal stenosis (LSS) refers to the anatomical narrowing of the spinal canal and is associated with a plethora of clinical symptoms. The annual incidence of LSS is reported to be five cases per 100,000 individuals, which is fourfold higher than the incidence of cervical spinal stenosis.[1] The characteristic symptom of LSS is neurogenic claudication, which was a term coined by Dejerine (1911)[2] and defined by von Gelderen (1948)[3] and, later, Verbiest (1954).[4] In his report, von Gelderen described neurogenic claudication as "localized, bony discoligamentous narrowing of the spinal canal that is associated with a complex of clinical signs and symptoms comprising back pain and stress-related symptoms in the legs (claudication)".[3] This characterization is still in use today. LSS has become the most common indication for lumbar spine surgery, in part because of the increasing quality and availability of radiological imaging.[5] The increasing frequency of LSS surgery also reflects the elevated demand for mobility and flexibility in the aging population. Propagated by the increasing prevalence of this condition, controlled, evidence-based advice for individual treatment decisions is starting to emerge.[5–7]

LSS can be classified according to etiology (primary and secondary stenoses) and to anatomy (central, lateral or foraminal stenosis), as summarized in Box 1. Primary stenosis is caused by congenital narrowing of the spinal canal,[8,9] whereas secondary stenosis can result from a wide range of conditions, most often chronic degeneration,

which leads to a destabilized vertebral body. Other causes of secondary stenosis include rheumatoid diseases, osteomyelitis, trauma, tumors, and, in rare cases, Cushing disease or iatrogenic cortisone application.[10]

In this Review, we explore the underlying pathophysiology of LSS, focusing on degenerative LSS, and discuss the characteristic hallmarks of the resulting clinical syndrome, paraclinical determinants of the condition and the results of interventional trials.

Pathophysiology

Stenosis development

LSS can be monosegmental or multisegmental, and unilateral or bilateral. Anatomically, the stenosis can be classified as central, lateral or foraminal. Depending on the extent of the degeneration, central, lateral and foraminal stenosis can occur alone or in combination. The L4–5 spinal discs are most frequently affected by LSS, followed by L3–4, L5–S1, and L1–2.

Multiple factors can contribute to the development of spinal stenosis, and these can act synergistically to exacerbate the condition. Degeneration of the vertebral disc often causes a protrusion, which leads to ventral narrowing of the spinal canal (central stenosis; Figure 1a). As a consequence of disc degeneration, the height of the intervertebral space is further reduced, which causes the recess and the intervertebral foramina to narrow (foraminal stenosis), exerting strain on the facet joints (Figure 1). Such an increase in load can lead to facet joint arthrosis, hypertrophy of the joint capsules and the development of expanding joint cysts (lateral stenosis), which in combination propagate spinal instability.[11] The reduced height of the segment leads the ligamenta flava to form creases, which exert pressure on the spinal dura from the dorsal side (central stenosis). Concomitant instability due to loosened tendons (for example, the ligamenta flava) further propagates pre-existing hypertrophic changes in the soft tissue and osteophytes, creating the characteristic trefoil-shaped narrowing of the central canal.[8,9,11–16]

LSS can also be subdivided into relative and absolute LSS—a classification that has not yet been clinically validated—according to the anterior–posterior diameter of the spinal canal (Figure 1a). Relative LSS (spinal canal 10–12 mm in diameter; physiological value is 22–25 mm) is usually asymptomatic, whereas absolute LSS (spinal canal <10 mm in diameter) is often symptomatic and is associated with absence of free subarachnoid space (as observed on lateral plain X-ray films). The lateral recess can be considered stenotic if it has a diameter of <2 mm (physiological diameter is 3–5 mm).

From stenosis to claudication

Each of the various degenerative processes that participate in the development of LSS can independently cause clinical symptoms that frequently make diagnosis and the choice of therapy difficult. The most common symptom associated with LSS is neurogenic claudication, which comprises limping or cramping lumbar pain that radiates into the legs primarily during walking. Degenerative LSS can ultimately lead to the compression of individual

Key points

- A patient's medical history and clinical symptoms are more-decisive factors than radiological observations in confirming a diagnosis of lumbar spinal stenosis (LSS)
- Patients with mild to moderate symptoms of LSS should be treated with conservative therapies, including delordosing measures, and epidural injections and other pharmacological measures
- In cases of severe symptomatic LSS, surgery is indicated if conservative therapy proves ineffective after 3–6 months
- Class I evidence-based recommendations cannot be made for any conservative or surgical therapy in relation to mid-term and long-term patient outcomes
- Future mid-term and long-term studies should identify subgroups of patients who are more likely to benefit from surgery than from conservative treatment

Box 1 | Classification and differential diagnoses of lumbar spinal stenosis

Classification according to etiology

Primary stenosis

- Idiopathic stenosis
- Achondrodysplasia

Secondary stenosis

- Degenerative (for example, spondylosis, spondylolisthesis, scoliosis)
- Ossification of the ligamentum longitudinale posterius and ligamentum flavum
- Metabolic or endocrine causes (for example, epidural lipomatosis, acromegaly)
- Infections (discitis, osteomyelitis, Pott's disease [tuberculous spondylitis])
- Neoplastic
- Rheumatological conditions (for example, Paget disease, spondylosis ankylopoetica, rheumatoid arthritis)
- Posttraumatic or postoperative stenosis (for example, fracture of vertebrae, laminectomy, fusion, fibrosis)

Classification according to anatomy

- Central stenosis (with or without lateral stenosis)
- Isolated lateral stenosis
- Foraminal stenosis

Differential diagnoses

- Intermittent claudication or vascular claudication
- Radiculopathies or polyneuropathies
- Intraspinal synovial cyst
- Disc prolapse
- Tethered cord or spina bifida
- Coxarthrosis or arthrosis of the iliosacral joint
- Abdominal aortic aneurysm
- Neoplasia (for example, tumor of myelon, spinal roots, meninges, bones or filiae)
- Inflammatory conditions (for example, spondylodiscitis, meningeosis, arachnoiditis)
- Dissociative syndromes

Derived from Haarmeier and Stolke.[58]

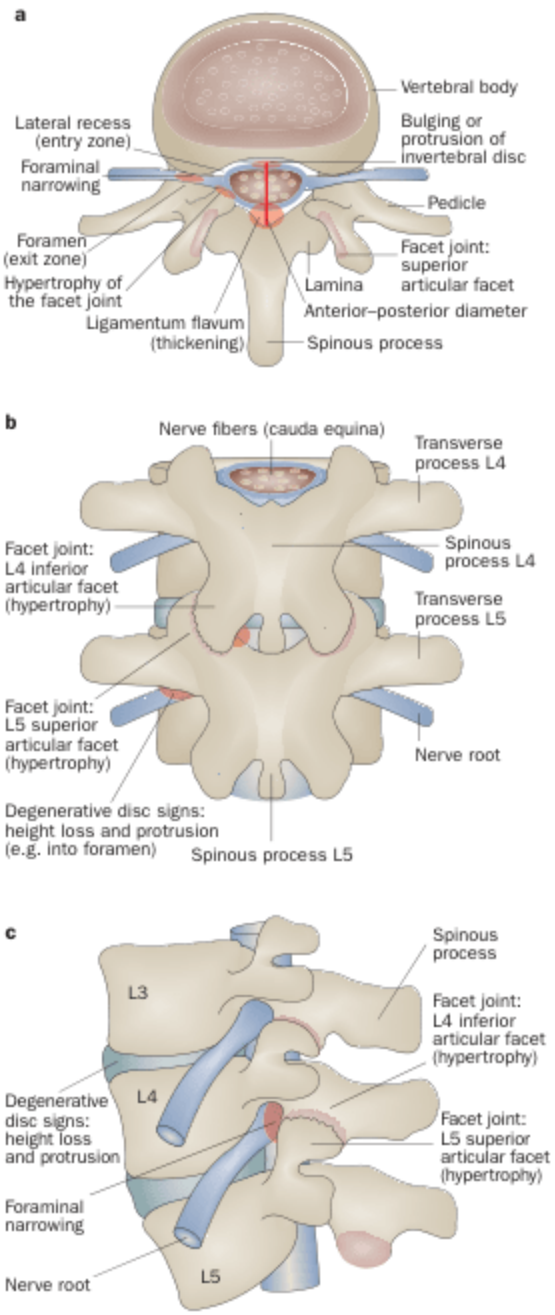

Figure 1 | Pathoanatomical illustration of LSS. Osteolisthesis and disc prolapse are distinct entities from LSS, although these conditions will frequently exacerbate the pre-existing lumbar stenosis. **a** | Coronal, **b** | dorsal and **c** | lateral views of LSS. In **a** | distinct stenosis areas are depicted in red. Ventral compression can be caused by medially bulging or protrusion of intervertebral discs. Lateral stenosis can be caused by lateral prolapse, stenosis of the neuroforamen or hypertrophy of the facet joints. **b** | Dorsal view of lateral stenosis (red dots) caused by hypertrophic facet joints and narrowing of the neuroforamen. **c** | Corresponding lateral perspective of narrowed neuroforamen causing a lateral stenosis. Abbreviation: LSS, lumbar spinal stenosis.

nerve roots, the meninges, the intraspinal vessels, and, in exceptional cases, the cauda equina (Figure 2).[17] Nerve root compression triggers localized inflammation, which affects the nerve root's excitatory state.[18] In addition, at least two interdependent vascular mechanisms are hypothesized to contribute to the development of neurogenic claudication in LSS: reduced arterial blood flow resulting in ischemia, and venous congestion with compression of the nerves and secondary perfusion deficiency.[19,20] Conversely, compressive radiculopathy can cause autonomic dysregulation and impaired circulation in the legs.[21] The extent of compression is increased by hyperextension or hyperlordosis of the lumbar spine, because these postures cause additional narrowing of the spinal canal. By contrast, hyperflexion abrogates lordosis, resulting in a widening of the spinal canal. A functional LSS can be diagnosed only if a clinically relevant LSS develops in certain spinal postures (for example, when standing as opposed to sitting). Such stenoses are frequently exacerbated further by vertical load.[22] Indeed, epidural pressure is elevated while standing or walking, and lowered when sitting and in flexion.[23,24]

Experimental animal models have been developed to investigate the underlying pathophysiology of LSS in more detail[25] and to test pharmacological interventional strategies,[26] but the validity of these models for the multifaceted, etiologically diverse human condition remains limited. In one such experimental model for spinal canal stenosis, a piece of silicon is placed under the lamina at lumbar level 4 in young adult rats. This model might only deliver incomplete information, since acute narrowing of the spinal canal *per se* does not fully recapitulate the features of chronic degenerative LSS in humans, and the young adult animals used in those experiments lack comorbidities. In addition, the spine biomechanics of quadripedal rats differ substantially from those of bipedal patients.

Signs and symptoms

In contrast to the well-defined pathoanatomical hallmarks of LSS, the clinical features of the condition are heterogeneous, and often, but not always, include neurological symptoms.[27] Typically, patient symptoms comprise unilateral or bilateral (exertional) back and leg pain, which slowly develops and persists over several months, or even years (Box 2). The back pain is localized to the lumbar spine and can radiate towards the gluteal region, groin and legs, frequently displaying a pseudoradicular pattern. In cases of lateral recess stenosis or foraminal stenosis, isolated radiculopathy can occur. Neurogenic claudication is the most specific symptom of LSS,[6] although it is nearly always accompanied by further symptoms. Taking into account all the symptoms, LSS can be clinically classified into grades I–III.[28] Grade I (neurogenic intermittent claudication) is characterized by a reduced walking distance (caused by pain) and short intermittent sensomotoric deficits that at rest might be unremarkable, but can deteriorate while walking.

However, not all patients with LSS exhibit symptoms consistent with neurogenic intermittent claudication, which is why other classifications of LSS exist. Grade II (intermittent paresis) refers to already persistent sensitivity deficits, loss of reflexes and intermittent paresis. Grade III is reached if persistent, progressing paresis is present, accompanied by partial regression of pain.[28]

Neurogenic claudication can be clinically distinguished from vascular intermittent claudication by the presence in the former of pain regression following flexion (delordosis) of the spine (for example, while cycling). In contrast to vascular claudication, pain sensation in patients with LSS does not ease while standing. The relative proportions of the low back pain component (an indicator of pathology such as concomitant vertebral instability or facet joint arthrosis) and the leg pain component have proved helpful for clinical orientation.[11] Lasègue testing (a passive leg flexing test) often remains negative in patients with LSS and is frequently accompanied by a feeling of 'heavy legs', a characteristic sign of LSS. Straightforward detection of LSS is hampered by a number of frequent comorbidities such as peripheral neuropathies, which can themselves be relevant differential diagnoses (Box 1).

Approximately 20% of patients with LSS exhibit symptoms of depression and 25% are dissatisfied with their life before surgery—a similar pattern to that seen in patients with other chronic disorders.[29,30] Evaluation of mood and contentment in patients is important, as both can markedly differ between patients with LSS and healthy controls, and can influence diagnostic and therapeutic decisions. Patient-reported symptoms—even those that are transient in nature—should be considered seriously in the diagnostic work-up, especially during initial consultations.

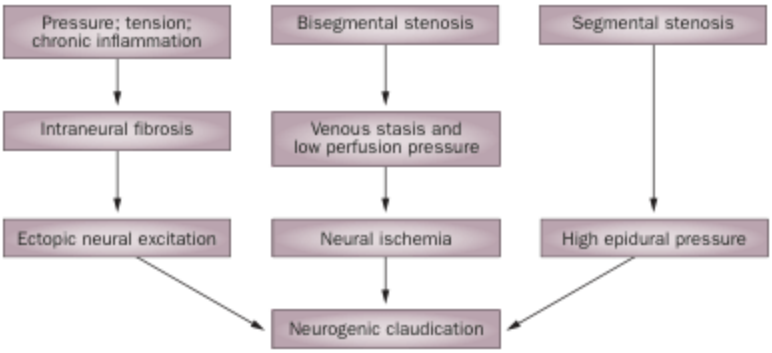

Figure 2 | Processes involved in neurogenic claudication development in lumbar spinal stenosis.

Box 2 | Symptoms and features of lumbar spinal stenosis

Classic symptoms

- Lumbago
- Neurogenic claudication
- Hypesthesia and paresthesia of the legs
- Ataxia
- Weakness and feeling of heavy legs

Features

- Improvement during lumbar delordosing
- Deterioration during lumbar lordosing
- Weakness of the legs
- Attenuated reflexes (pseudoradicular)

Derived from Haarmeier and Stolke.[58]

Diagnosis

The frequency of degenerative LSS diagnosis has risen over time, as a result of increasing lifespan and demand for a better quality of life, awareness of the disease, and the availability of advanced imaging techniques. LSS can be difficult to diagnose, however, because the symptoms can mimic other diseases. On the other hand, various comorbidities, which are prevalent in the aging population, can result in secondary stenosis or imitate symptoms of it. Thus, the differentiation of LSS from numerous other pathologies is vital (Box 1). Clinical symptoms of LSS are often absent at rest. In addition, it can be difficult to establish whether pain (and other patient-reported symptoms) relates to LSS or to other factors (for example, instability, facet joint arthrosis, osteoporosis, arthritis, or diabetic polyneuropathy). Hence, a diagnosis of LSS can only be established through a combination of clinical history, physical examination and radiological changes.

Differential diagnoses

In contrast to the situation in LSS, hyposensibility resulting from peripheral neuropathies usually exhibits a bilateral distal stocking-shaped pattern, irrespective of posture, rest or physical stress. Iliosacral joint disorder occasionally mimics LSS, with low back pain radiating to the buttocks and the thighs when standing and walking. Unlike LSS, however, iliosacral joint pain is characterized by tenderness of the joint. The development of cauda equina syndrome, which comprises sacral hypesthesia, loss of tendon reflexes in the lower limbs and incontinence, as a result of LSS is only found in exceptional cases. Sphincter involvement is very rare in LSS, as the sacral nerves are relatively protected from compression owing to their central position within the cauda equina.[31] In patients exhibiting vesicorectal voiding and upper motor neuron signs (for example, Babinski's reflex and hyperreflexia), cervical or thoracic myelopathy needs to be ruled out.

Neuroradiological assessment

When performing radiological assessment of LSS, some inherent problems with imaging of the lumbar spinal canal need to be considered. First, imaging of symptomatic patients is confounded by the fact that degenerative changes in the lumbar spine are highly prevalent in the asymptomatic population: among patients over 60 years of age, 20% will reveal signs of LSS.[32] Second, imaging tends to exaggerate pronounced degenerative changes

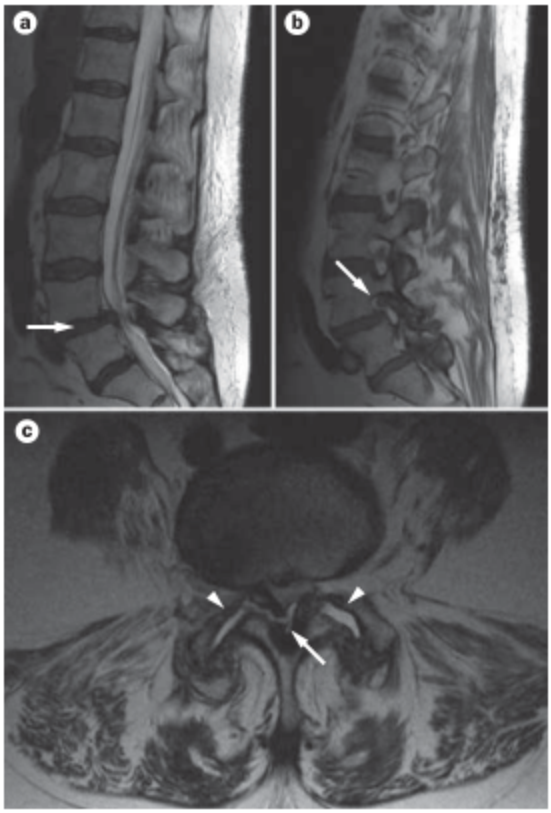

Figure 3 | Spinal alterations in a patient with monosegmental LSS at L4–5. Sagittal and axial images were obtained using T2-weighted turbo spin-echo MRI. **a** | A sagittal image reveals reductions in the disc signal and the disc space height, which are attributable to dehydration. Disc bulging and slight ventral listhesis of L4 (arrow) can also be observed. **b** | Narrowing of the neuroforamen (arrow), which affects the right L4 radix, is caused by **c** | consecutive hypertrophic facet joint degeneration with intra-articular effusions (arrowheads) and hypertrophy of the ligamentum flavum (arrow), as observed on an axial image.

and effects on the spinal canal.[33] Thus, radiologically diagnosed LSS usually identifies involvement of more segments than is suspected clinically. In the majority of cases, a presumptive diagnosis of LSS can be made on the basis of the clinical appearance of the condition and the patient's medical history. Imaging tends, therefore, to be selectively employed when any type of interventional or surgical therapy is contemplated. Notably, imaging tends to be used most frequently in patients with medium to severe symptoms of LSS.[34]

In presurgical patients with symptoms of LSS, the purpose of imaging is to confirm the presence or absence of LSS, to exclude differential diagnoses, to relate congesting symptoms to osseous and discoligamentous structures, and to identify the exact location of LSS for accurate presurgical planning.[35] As described previously, LSS mostly results—at least initially—from degenerative disc disease. Morphological alterations, such as loss of disc height, disc signal, bulging discs, disc herniations, reactive end-plate and bone marrow changes, and spondylophytes, can be visualized to differing degrees by the various imaging techniques applied. Increased stress on the facet joints leads to hypertrophic facet degeneration, as well as inwardly buckled and hypertrophied ligamenta flava. These changes can lead to central, lateral or foraminal stenoses (Figure 1).

MRI

MRI is the preferred imaging modality for the radiological assessment of LSS.[36] This technique provides superior soft-tissue contrast compared with other imaging modalities, has multiplanar imaging capabilities and does not produce ionizing radiation. In patients with pacemakers, certain other types of metal implants or claustrophobia, however, MRI is contraindicated or impossible to perform.

MRI of patients with LSS usually comprises orthogonal T1-weighted and T2-weighted images (sagittal and axial). A fat-suppressed T2-weighted sequence can be added, as such images seem to allow more accurate detection of associated degenerative bone marrow changes. With T2-weighted images and the inherent signal intensity of cerebrospinal fluid, 'myelography-like' images that illustrate the thecal sac, the intrathecal and intraforaminal nerve roots, and the spinal cord can be obtained noninvasively. LSS can be monosegmental (Figure 3) or occur on multiple levels (Figures 4 and 5). Like CT imaging, MRI can define the contribution of osseous and discoligamentous structures to LSS.

Despite detailed depiction of the spinal anatomy, studies have produced conflicting results concerning the clinical usefulness of the information gained by MRI.[37,38] Results from a study conducted by Modic and colleagues, in patients with radiculopathy, low back pain and sciatica, implied that changes observed by means of MRI add little or no clinically useful information to clinical assessment alone in relation to prognosis and predicting the outcome of surgery.[37,39]

Gadolinium-based contrast media are not routinely required for imaging of LSS unless previous surgery was performed and fibroid scar tissue might have to be identified by its contrast enhancement.[40,41] Some studies, however, indicate a possible superior role for contrast-enhanced MRI in LSS patients with neurogenic claudication, as enhancement of compressed nerve roots can be visualized in a subset of these patients.[42–44] This enhancement is thought to reflect either obstructed periradicular veins, indicating venous stasis, or breakdown of the blood–nerve barrier, a sign of chronic compressive radiculitis (Figure 2).

Through the use of heavily T2-weighted fat-suppressed sequences, magnetic resonance (MR) myelography can be performed noninvasively and without contrast administration. Despite the capacity of this technique to accurately depict the thecal sac, however, studies have yielded contradictory results regarding the usefulness of this sequence.[45,46] The use of MR myelography is, therefore, only advocated as an additional sequence to

conventional MRI. Currently, open MRI is the only technique that enables a functional investigation of spinal flexion and extension during the application of axial loading, or even in the supine position.[47]

CT scanning

CT can be performed rapidly and allows precise evaluation of the spinal canal and differentiation between spinal canal compression caused by discs, ligaments and bony structures. In the latter respect, this approach is superior to MRI. At present, CT is usually performed using a spiral multislice technique, acquiring isotropic data that enables multiplanar reformatting in any desired plane and three-dimensional reconstructions. Even in pronounced torsion scoliosis, therefore, multisegmental imaging in one plane can be achieved, which is not possible with MRI. A limitation of CT is that intrathecal nerve roots and the spinal cord cannot be visualized, because these structures have similar densities to the cerebrospinal fluid. This problem might be circumvented by using CT myelography (Figure 5). CT myelography entails spiral CT imaging acquired after inthecal administration of iodine, which is commonly performed under fluoroscopic guidance. In some cases of extensive degenerative or postsurgical changes, lumbar puncture can also be performed under CT guidance. Such CT-guided puncture of the thecal sac is a robust technique, even in patients who cannot be assessed by other means. CT and CT myelography might be indicated in patients where MRI is contraindicated, the MRI results are inconclusive or where clinical symptoms correlate poorly with MRI findings.[47] Furthermore, CT techniques might be used for presurgical planning in cases where bony anatomy needs to be accurately depicted.

Conventional X-rays and myelography

The usefulness of routinely acquired plain radiographs in the initial evaluation of patients with LSS has been questioned.[48,49] Indeed, the acquisition of such radiographs is no longer part of the Agency for Health Care Policy and Research guidelines.[34] Many patients, however, undergo conventional radiography as part of their initial evaluation, as this procedure is inexpensive and uncomplicated to perform. Conventional radiographs might be of use, albeit in a limited fashion, in assessing the contribution of bony degeneration to LSS and the alignment of the vertebral bodies in lateral and coronal planes. This technique can also potentially be used to rule out traumatic changes or other unanticipated findings (for example, Paget disease, spondylodiscitis or scoliosis) as possible differential diagnoses.[50] After surgery, plain radiographs are useful in determining the integrity and the correct position of fusion material, and to visualize signs of loosening of implanted fixating plates and/or screws. The sensitivity and specificity of plain radiographs concerning the contribution of bony changes to central spinal stenosis were reported to amount to 66% and 98% respectively of those of CT. The acquisition of additional lateral radiographs in flexion and extension positions (so-called functional radiographs) to rule out segmental instability is not routinely required, as signs of segmental instability can be detected on conventional lateral radiographs in a sufficiently accurate manner.[51] Furthermore, no additional benefits were gained from these additional views in a recently conducted study.[50] Even in patients for whom segmental instability was expected, the diagnostic value of lateral radiographs in flexion and extension could not be definitively determined.[52]

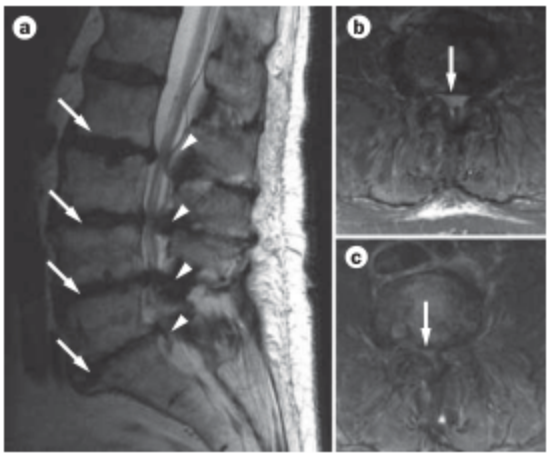

Figure 4 | Spinal alterations in a patient with multisegmental LSS. Sagittal and axial images were obtained using T2-weighted turbo spin-echo MRI. **a** | A sagittal image reveals multisegmental LSS with signs of spondylosteochondrosis, such as disc height and signal reduction, disc herniation, irregularity of end plates and bone marrow degeneration (arrows), and spondylarthrosis (hypertrophy and sclerosis of the facet joints; arrowheads). The axial images depict **b** | a moderate (arrow) and **c** | a severe (arrow) central LSS with different degrees of disc pathology, hypertrophy of ligamenta flava and facet joints. Abbreviation: LSS, lumbar spinal stenosis.

Conventional functional myelography has long been the method of choice for diagnosing LSS and is still an important method for investigating the influence of hyperextension and hyperflexion on the extent of the stenosis. This technique might still be the only routine method that is suitable for detecting the morphological correlates of a functional, posture-dependent, symptomatic LSS (Figure 6).[35] Furthermore, it is the only accurate imaging technique for patients with spinal metallic implants, which can cause artifacts on MRI and CT. Moreover, conventional functional myelography allows the lumber spine to be examined in a standing position, and, hence, under the normal stress of the body weight. Conventional myelography is an invasive procedure that requires intrathecal administration of iodinated contrast agent, and is consequently associated with adverse effects such as postpunctional headaches, and rare life-threatening complications such as anaphylactic reactions and spinal infections. Like other imaging techniques, conventional myelography frequently reveals

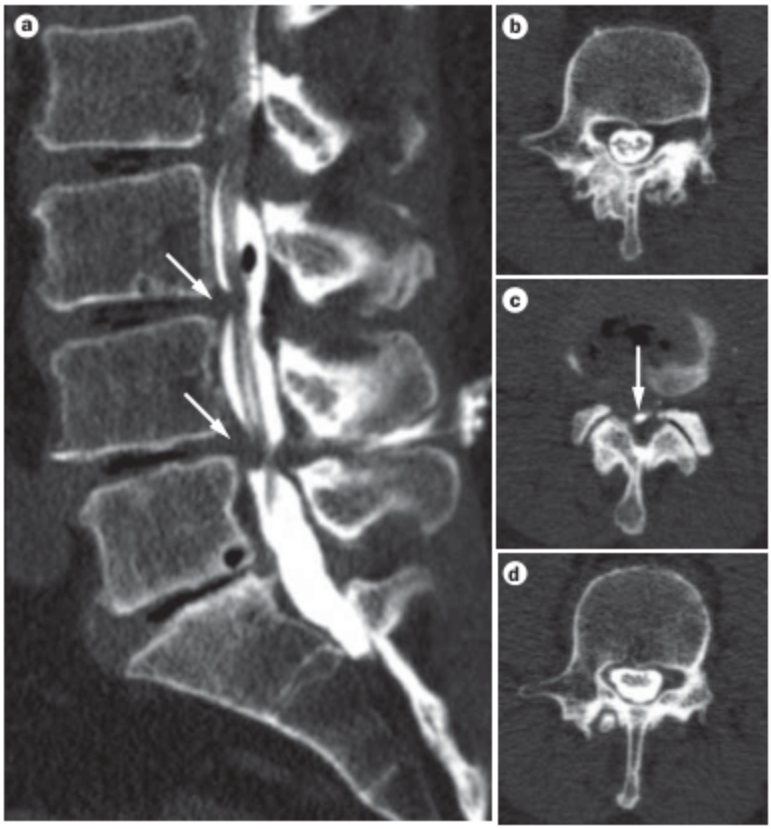

Figure 5 | Multisegmental disc degeneration revealed by CT myelography. **a** | A sagittal reformatted CT myelograph reveals a multisegmental severe disc degeneration, with disc space height reduction, vacuum phenomenon and endplate sclerosis of the lower lumbar spine, as well as thecal sac compressions at the L3–4 and L4–5 levels (arrows). **b**–**d** | Axial reformatted images show a circumscribed severe LSS of L3–4, with a typical hourglass constriction of the thecal sac (arrow) adjacent to relatively normal areas. Abbreviation: LSS, lumbar spinal stenosis.

abnormalities that were not suspected clinically.[33] One of the few reliable prognostic signs is the block of contrast flow, which is a good predictor of the successful outcome of decompression surgery.[53] Conventional myelography is limited by its inability to determine the cause of block or compression and to visualize extrathecal nerve root compression. This technique is usually combined, therefore, with a CT scan performed after myelography (postmyelographic CT), which compensates for these limitations (Figure 5). Myelography combined with postmyelographic CT might be indicated for preoperative surgical planning, so as to assess the thecal sac and the bony status of the surgical area.

In summary, recent studies have shown that the diagnostic and predictive values of conventional myelography, CT myelography and MRI are not markedly different.[54] One important point to consider when assessing imaging methods is that the radiological degree of LSS, both before and after surgery, does not necessarily correlate with the degree of the clinical signs and symptoms.[32,54–58]

Additional diagnostics

Selective diagnostic injections can be useful in some patients to estimate the contribution of different pain components to the patient's overall health, especially against the background of pain psychology in chronic pain. If vascular genesis of the symptoms is suspected, noninvasive diagnostic techniques include determination of the ratio of the systolic blood pressure of the ankles to that of the arms (ankle–brachial index), which can be considered to be pathological when the value is <0.5. In addition, routine duplex Doppler angiography, contrast-enhanced MR angiography and—in rare cases before intervention—digital subtraction angiography can also be employed to determine the involvement of vascular genesis in causing pain. Given the low practical importance of classical electromyograghy and nerve conduction studies in diagnosing LSS, an electrophysiological examination is only recommended to exclude other disorders, especially if the distribution of pain and numbness is unusual (for example, suspicion of peripheral polyneuropathy or myopathy, which might both occur concomitantly with LSS).[55,56] Walking on a treadmill is an appropriate provocation test for such examinations, although this technique is not yet common in daily practice.[55,56] Routine laboratory tests can be used to detect comorbidities, such as diabetes or diabetic polyneuropathy (by detection of glucose and HbA1c), and infections such as spondylodiscitis (by measurement of C-reactive protein).

Therapy

The progressive nature of degenerative LSS makes fully curing the condition unlikely, so the primary objective of each treatment is to reduce the severity of the symptoms (Box 2). Recent interventional strategies have mostly focused on pain (bothersome indices) and physical function as primary end points.[57] The indications for intervention are not absolute in the majority of patients. Cauda equina syndrome or relevant paresis are, however, imperative indications for intervention. Given the considerable pathological and clinical heterogeneity of LSS, the lack of therapeutic recommendations and the large number of distinct therapies, the selection of an appropriate procedure is difficult.[4] Prospective, randomized studies comparing the various therapies are urgently required.[58,59] The need for efficient therapy for LSS is reflected by the substantial economic burden of low back pain, which is estimated to exceed US$100 billion, with lost productivity at work representing the majority of the overall costs.[60]

Natural disease course

LSS is a degenerative condition that develops slowly over time, and for much of the clinical course of the disease the neurological deficits are only subtle. For these reasons, LSS is usually diagnosed in patients over the age of 50 years. There are, however, no prospective long-term studies that document the natural symptomatic changes

over time.[31,58] This makes the initiation and choice of a specific therapy difficult, as such decisions ideally require an estimate of the natural course of the condition.[6] Limited information is available from the subgroups of untreated patients in some intervention trials. The Spine Patient Outcomes Research Trial (SPORT) reported that there was no worsening of symptoms over 2 years in most patients in the conservatively treated (see below) control group.[57] Another study reported an increase in the severity of symptoms in ~20% of the untreated cases,[31] whereas a further trial focusing on pain development over almost 5 years found that the clinical symptoms of 70% patients reached a plateau, 15% experienced pain exacerbation and 15% spontaneously improved.[11,59] Given that long-term clinical stability is common in LSS, the acute exacerbations of symptoms should not be confused with a change in the patient's trajectory.

Conservative therapy

The conservative treatment of LSS comprises a wide variety of methods, such as physical therapy, ergotherapy, behavioral therapy, delordosing orthopedic devices, girdles, acupuncture, manual therapy and pharmacological intervention. Few studies have been conducted to demonstrate the effectiveness of conservative therapy in treating LSS, although those that reported the use of such an approach had success rates of up to 70%.[61–63] None of the available studies, however, provide sufficient data to support the superiority, or even the effectiveness, of any one of the wide range of conservative treatments.[6,64] In the absence of evidence-based clinical guidelines, an intensified, multidisciplinary approach should be given preference over a singular therapy.[64,65] The objectives of applicable physiotherapy and manual therapy approaches are flexion, distraction, neural mobilization and relief of the affected segments, as well as improvements in paravertebral muscle tone through the use of stabilizing exercises.[61,66] There is wide agreement between clinicians that bed rest is not recommended in the therapy of chronic and acute pain.[64] Advice tailored to individual patients is of central importance in LSS, particularly in cases with mild symptoms, because the simple modification of an individual's behavior can be sufficient to stabilize or improve the condition.

The pharmacological component of conservative therapy aims to relieve painful nerve-root pathologies and is identical to the medication given for a disc prolapse (herniation). Agents used to treat LSS include NSAIDs, other peripheral analgesics, steroids, muscle relaxants, opioids, antidepressants and, in very severe cases where quality of life is impaired, neuroleptics. Besides oral medication, weekly therapeutic injections can offer short-term to medium-term alleviation. Frequently, steroids are used in combination with local anesthetics in epidural, deep paravertebral, pararadicular and facet joint injections. Such invasive procedures are, however, associated with a risk of developing infections.

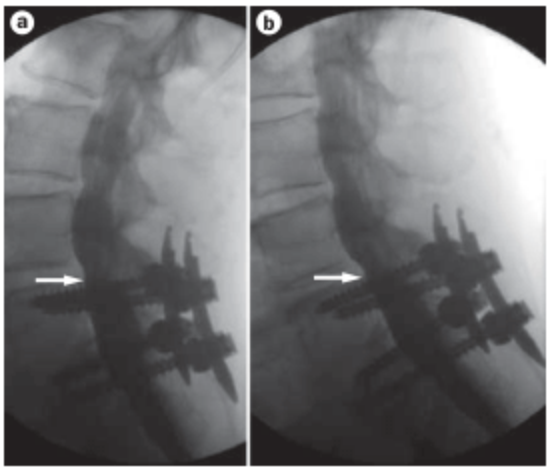

Figure 6 | Conventional myelography in a patient with a posterior lumbal intervertebral fusion and positional back pain. A reactive hypermobility adjacent to the fused segment is viewed in **a** | a reclined position and **b** | an inclined position. A moderate ventral slipping is evident in the inclined position. No substantial positional effect on the sagittal diameter of the thecal sac can be observed.

As for all conservative treatment strategies, the effectiveness of drug regimens has only been investigated in a few studies. Evidence-based recommendations cannot, therefore, be made for long-term administration of NSAIDs and muscle relaxants, or for the use of steroids, antidepressants and long-acting opioids.[6,65] Likewise, the evidence for the efficacy of therapeutic injections for LSS has not been confirmed.[67–69]

Surgery

If a diagnosis of LSS has been established with consistent results from clinical history taking, physical examination and radiological assessment, conservative treatment should be applied for 3–6 months, with the aim of achieving satisfactory improvement of the symptoms. In patients in whom severe symptoms persist and functional impairment develops, surgery is the recommended option, unless this approach is contraindicated for other reasons. Clinicians should also consider that some patients simply do not want to have surgery, despite meeting these criteria, whereas many others have unrealistic expectations of what can be achieved with surgical procedures.[70]

All surgical procedures used in LSS aim to decompress the entrapped neural elements, without disrupting the stability of the segment. Such decompression surgery usually leads to spontaneous relief of pain in the legs, and, to a lesser degree, of low back pain.[71] The speed and extent of recovery is, however, unpredictable, even if pressure on nerve roots, dura and blood vessels is sufficiently eliminated. Decompressive surgical procedures include laminectomy and hemilaminectomy, hemilaminotomy, fenestration, foraminotomy and the implantation of interspinous distraction devices.[11,69,72,73] The complication rates for decompression surgery (during and after the surgical

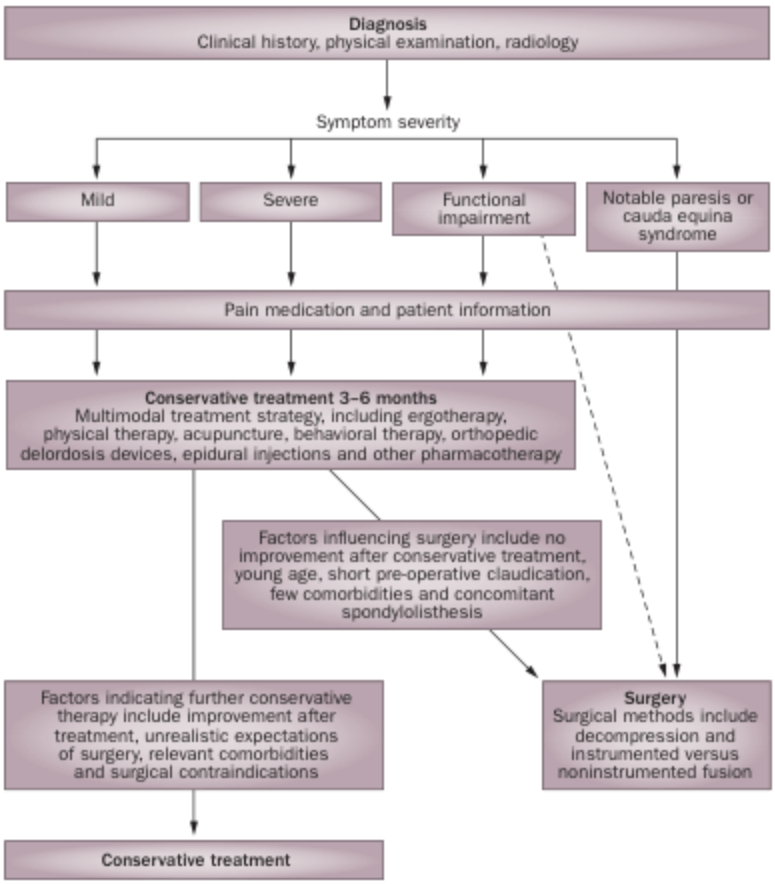

Figure 7 | Proposed treatment algorithm for symptomatic LSS. The diagnosis of LSS is made on the basis of consistent results from clinical history taking, a physical examination and radiological observations. Given the considerable pathological and clinical heterogeneity of LSS, the lack of therapeutic recommendations and the large number of distinct therapies, the selection of an appropriate procedure is difficult. In general, conservative treatment should be applied for 3–6 months, except in cases of cauda equina syndrome or relevant paresis—both of these conditions are absolute indications for surgical intervention. In patients who are refractory to treatment, with persisting severe symptoms and functional impairment, surgery is a recommended option, provided that there are no contraindications that increase the risks of surgical procedures. Importantly, there is no sharp border between conservative and surgical regimens in every symptom group, which reflects the lack of evidence-based recommendations. Moreover, there are insufficient evidence-based data to support the superiority of any individual treatment over the array of conservative therapies and surgery options. Abbreviation: LSS, lumbar spinal stenosis.

procedure) range from 14%[57] to 35% or more.[74–76] Fusion surgery, which is a more invasive procedure than decompression surgery and is used in cases of instability, is associated with higher complication rates. Typical complications of both decompression and fusion surgery include dura vessel lacerations, epidural hematomas, inadequate decompression with significant residual stenosis, instability, and reossification. All of these complications result in renewed nerve compression.[58,59,75–78] The 10-year reoperation rates after decompressive surgical procedures are reported to range from 10–23%.[71,79] Additional fusion surgery lowered the rate of reoperation in one study.[79] Additive fusion surgery might be needed in cases of instability (rotational or vertical mobility of the vertebral body >3 mm), spondylolisthesis (>5 mm forward movement of a lumbar vertebra relative to one below)[80] or scoliosis (lateral curvature of the spine) >20°,[81] because instability can foster spinal root congestion. Success rates for decompression surgery in cases of LSS range from 40–90% in the literature and depend on a wide variety of factors such as type of decompression, duration of follow-up, age of patients and comorbidities.[77,82–88]

Results from one study showed that patients who underwent laminotomy were more likely to show a marked improvement in lumbago than patients who underwent laminectomy.[87] A randomized trial revealed that bilateral laminotomy conferred greater clinical benefit than unilateral laminotomy or laminectomy in patients with LSS at 1 year post-surgery.[76] Another study compared the 1-year results after tissue sparing—so-called undercutting decompression—with those after the more-invasive laminectomy procedure and found no statistically significant difference between the two procedures.[89] Of patients who underwent unilateral foraminotomy for degenerative foraminal stenosis, 91% reported an improvement of leg pain, although there was a concomitant increase in lumbago in one-third of patients.[90] Resection of the pars interarticularis does not seem to result in segment instability, but might cause an increase in the frequency of lumbalgias.[91] Laminoplasty is recommended for central LSS as two-thirds of patients show improvement after 6 years.[92] Even in cases of mild degenerative spondylolistheses (complex stenosis), laminoplasty can produce good results, which are similar to those obtained with additive fusion.[86] To date, there is no agreement as to whether only the symptomatic level and side should be decompressed, or whether additional non-symptomatic—but evidently confirmed—neighbouring stenoses should also be decompressed.[58,59,93–96] In general, as for the preoperative diagnosis, the paraclinical assessment of a successful surgical intervention is hampered by the fact that the imaging data do not correlate well with the clinical presentation.

Conclusions

LSS has become an increasingly prevalent diagnosis, which is handled in a heterogeneous manner by clinicians. Decisions on treatments are made on the basis of clinical experience and emerging guidance from trial-based evidence, which is starting to meet rigorous evidence-based medicine criteria. Surgery is commonly recommended for cases of severe LSS with progressive neurological deficits and severe neurogenic claudication, but the decision to operate is influenced more by clinical experience than by proven evidence.[64] Recommending patients with only the most serious cases of LSS for surgery has also biased the available studies, making the decision between conservative therapy and surgery more difficult.[58,97]

Several meta-analyses have attempted to compare the success of conservative therapy with that of surgery.[6] Until recently, meta-analyses (including Cochrane reviews) of

surgical treatments for spinal stenosis concluded that there is still insufficient evidence to support surgery over non-surgical treaments.[6,58,98] The relevance of careful follow-up became evident with the publication of the Maine Lumbar Spine Study, which reported the 8–10-year outcome results for conservative versus surgical LSS therapy.[71] Short-term (1-year) to mid-term (4-year) results suggested that surgery was more beneficial than conservative treatment for patients with LSS.[99,100] After 8–10 years, approximately half of the patients reported an improvement in low back pain compared with baseline, regardless of the initial therapy method. One criticism of the Maine Lumbar Spine Study is that they used nonrandomly assigned patients, which affects the level of evidence generated by trials. A prospective study detected a better clinical outcome following surgery than in a control group receiving conservative therapy after both 4 and 10 years of follow-up.[93] This trial, however, had methodological restrictions, as it was only partly randomized (31 out of 100 patients) and 20% of the enrolled patients were lost to follow-up; thus, the results can only be considered as level 2b evidence. Since there was no difference in the clinical outcome between patients who were operated on shortly after being diagnosed and those who underwent surgery after initially receiving physiotherapy, the authors recommended conservative treatment in the first instance. By contrast, a pair-matched study demonstrated no statistically significant difference in clinical outcome between surgically decompressed and conservatively treated patients after a 4-year follow-up period.[96] In a further follow-up study 5–10 years after treatment there was no longer a significant difference between the two groups with regard to lumbago and patient satisfaction with their condition, although differences in leg pain and functional status were still detectable.[97]

Two prospective trials indicated that surgical decompression is superior to conservative therapy.[57,101] However, the differences in pain relief and improvement in functional status narrowed during the 2-year follow-up period in the Weinstein *et al.* study.[57] Aside from the short period to follow-up, other limitations of the study were the use of only one type of operation and the high rate of crossover from surgery to conservative therapy and vice versa. Moreover, a later meta-analysis was unable to provide evidence for the effectiveness of surgery in patients with LSS.[65] Nevertheless, the SPORT provides level 1b evidence, which is higher than the other evidence provided from previously discussed studies, and the mid-term and long-term follow-up results of the trial will be eagerly awaited, as they could heavily influence future treatment recommendations.

On the basis of the aforementioned studies, the provision of clear evidence-based recommendations in favor of surgery or conservative treatment for LSS is difficult. To provide guidance for clinical decision-making, we have proposed a treatment algorithm (Figure 7). Nevertheless, an individualized choice of treatment remains crucial. For example, the degree of pain in patients with LSS might make it impossible for them to perform their daily activities, so in those individuals surgical treatment is a reasonable proposition.[6] Such nonpaternalistic treatment decisions involve informed patients who have a full history of their symptoms and understand the underlying risks of both nonsurgical and surgical interventions. As decompression can still be performed successfully after the failure of conservative therapy,[93] initial conservative treatment should be applied for at least 3–6 months. If the symptoms do not improve satisfactorily, surgery might be indicated if there are no contraindications (for example, unacceptable anesthesia risk or marked psychosocial symptoms), and if the symptoms reported by the patient are consistent with the results of imaging, history, clinical results and physical examination.[57] The limited trial-based evidence means that the identification of subgroups of patients with LSS who are most likely to benefit from surgery will be imperative for future studies. So far, only a few prognostic signs, such as young age,[102] short preoperative duration of claudication (the absence of sphincter dysfunction and atrophy), symptom relief with lumbar flexion and a limited number or absence of comorbidities (for example, musculovascular and cardiovascular disorders), predict a favorable outcome after surgery.[84,103,104] In addition, in the case of concomitant degenerative spondylolisthesis, the clinical results are better after surgery than after conservative therapy.[105] The extent of radiological findings are generally of little help for the identification of a surgery indication.

In summary, there has been a longstanding need for evidence-based data on which to base treatment decisions for LSS, and this need has become increasingly important as the frequency of diagnosis has increased. The first randomized, prospective studies have provided class 1b evidence that in the short term, decompressive surgery produces a faster and more profound decline of symptoms than conservative therapy. However, in view of the narrowing of this effect during follow-up and in the absence of a valid paraclinical technical (surrogate) marker, more mid-term and long-term evidence-based data are needed to identify patients for whom the benefits of surgery would outweigh the risk of developing complications. Likewise, future studies should compare the effectiveness of the various nonsurgical, conservative treatments for LSS, as there is little supporting evidence for the current methods.

Review criteria

PubMed was searched using Entrez without date restrictions for articles, including early release publications. Search terms included "lumbar spinal stenosis", "intervention", and "outcome". The abstracts of retrieved citations were reviewed for relevant content. Full articles were obtained and references were checked for additional material when appropriate.

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Acknowledgments

E. Siebert and H. Prüss contributed equally to this article. J. M. Schwab receives support from the Berlin–Brandenburg Center for Regenerative Therapies, NeuroCure (together with K. M. Einhäupl), the Wings for Life Spinal Cord Research Foundation (Travelgrant) and the International Foundation for Research in Paraplegia.

Charles P. Vega, University of California, Irvine, CA, is the author of and is solely responsible for the content of the learning objectives, questions and answers of the MedscapeCME-accredited continuing medical education activity associated with this article.