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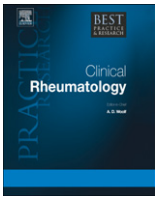

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Lumbar Spinal Stenosis

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Lumbar spinal stenosis (LSS) is most commonly due to degenerative changes in older individuals. LSS is being more commonly diagnosed and may relate to better access to advanced imaging and to an ageing population. This review focusses on radicular symptoms related to degenerative central and lateral stenosis and updates knowledge of LSS pathophysiology, diagnosis and management. Since patients with anatomic LSS can range from asymptomatic to severely disabled, the clinical diagnosis focusses on symptoms and examination findings associated with LSS. Imaging findings are helpful for patients with persistent, bothersome symptoms in whom invasive treatments are being considered. There is limited information from high-quality studies about the relative merits and demerits of commonly used treatments. Interpreting and comparing results of available research are limited by a lack of consensus about the definition of LSS. Nevertheless, evidence supports decompressive laminectomy for patients with persistent and bothersome symptoms. Recommendations favour a shared decision-making approach due to important trade-offs between alternative therapies and differences among patients in their preferences and values.

Lumbar spinal stenosis (LSS) is commonly used to describe patients with symptoms related to anatomical reduction of the lumbar spinal size. The challenge to this anatomically based definition is that while necessary for the diagnosis of LSS, it is not sufficient to determine the severity of symptoms

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and functional impairment that leads a patient to seek treatment. Indeed, even severe anatomical spinal stenosis may be present in asymptomatic patients. This review focusses on the clinical management of degenerative LSS including the aetiology and natural history, symptoms and physical findings, diagnostic testing and treatment options. Degenerative spinal stenosis can occur with other conditions including degenerative spondylolisthesis or degenerative scoliosis. Although many studies of degenerative LSS include individuals with these conditions, they are beyond the scope of this review.

The first clinical description of LSS is attributed to Verbiest in 1954 [1], although earlier descriptions are available [2]. More than 50 years later, there are still no widely accepted diagnostic or classification criteria for the diagnosis of LSS and, as a consequence, studies use widely differing eligibility criteria that limit the generalisability of reported findings [3]. Among older individuals, LSS can be a highly disabling condition [4], and is the most common reason for spinal surgery [5,6]. Though LSS is a growing concern with the ageing of the population, few studies have examined how its prevalence or incidence is changing [5].

Aetiology and pathophysiology

Spinal stenosis is most commonly classified as either primary, caused by congenital abnormalities, or a disorder of postnatal development [7], or secondary (acquired stenosis) resulting from degenerative changes or as consequences of local infection, trauma or surgery. The focus of this review is on the most common cause, a slowly progressive degenerative process that predominates at the three lower lumbar levels [8]. The natural history of spinal stenosis remains poorly understood with studies reporting about a half of patients remaining clinically stable, with a quarter worsening or improving [9]. For any individual patient, the course can be unpredictable with flares and stable periods over time [10].

Degenerative LSS anatomically can involve the central canal, lateral recess, foramina or any combination of these locations (Fig. 1). Central canal stenosis may result from a decrease in the antero-posterior, transversal or combined diameter secondary to loss of disc height with or without bulging of the intervertebral disc, and hypertrophy of the facet joints and the ligamentum flavum. Fibrosis is the main cause of ligamentum flavum hypertrophy and is caused by accumulated of mechanical stress, especially along the dorsal aspect of the ligamentum flavum. Transforming growth factor (TGF)-β released by the endothelial cells may stimulate fibrosis, especially during the early phase of hypertrophy [11]. The same processes, including decreased disc height, facet joint hypertrophy (with or without spondylolisthesis) and/or vertebral endplate osteophytosis can also result in lateral recess stenosis. Foraminal stenosis can be either antero-posterior resulting from a combination of disc-space

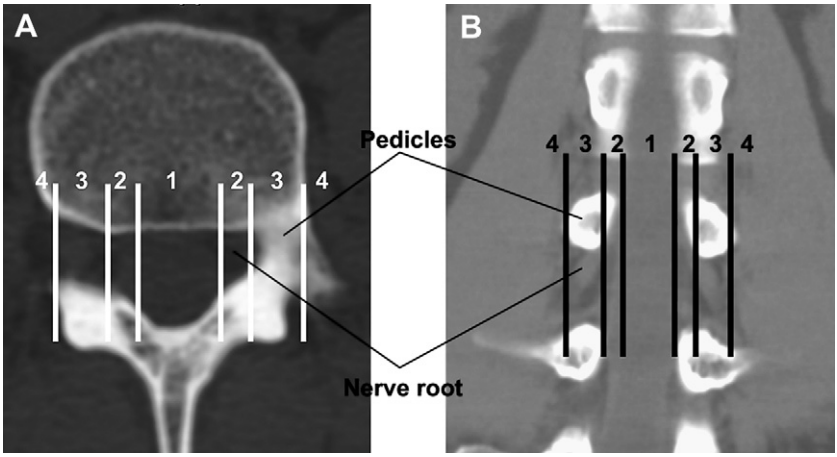

Fig. 1. Lumbar vertebrae. Potential regions of contact with nerve roots as described by Jenis(12): 1 central; 2 lateral recess; 3 foramen; 4 extraforaminal.