

# We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

185,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index  
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?  
Contact [book.department@intechopen.com](mailto:book.department@intechopen.com)

Numbers displayed above are based on latest data collected.  
For more information visit [www.intechopen.com](http://www.intechopen.com)



---

# Lumbar Spinal Stenosis, Clinical Presentation, Diagnosis, and Treatment

---

Luiz Cláudio Lacerda Rodrigues

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/63920>

---

## Abstract

Lumbar spinal stenosis (LSS), a disease that mainly affects people over 50 years old, may have a dramatic presentation with pain, difficulty in walking, changes in urinary functions in addition to root symptoms, such as numbness, burning, and feeling of heaviness in the legs. The treatment is very varied with several non-surgical and surgical possibilities. With an aging population, this disease becomes increasingly preoccupant for their uncertain evolution and without a well-defined correlation with imaging tests, treatment, and outcome may be troublesome. Moreover, LSS frequently affects patients who have associated comorbidity that can hinder the treatment.

**Keywords:** lumbar stenosis, outcomes, degenerative disc, pathophysiology, surgical treatment, non-surgical treatment

---

## 1. Introduction

Lumbar canal stenosis was first described by Antoine Portal in 1803. However, Verbiest [1, 2] was the first to associate changes in the diameter of the vertebral canal with the clinical features and neurogenic claudication. The reduced canal diameter was only correlated to the disc degenerative process by Kirkaldy-Willis, when the authors demonstrated that disc degeneration was directly related to the changes that lead to the physiopathology of reduced vertebral canal diameter [3].

Based on a study of dissection of 50 cadavers, Kirkaldy and Willis described how changes in the zygapophyseal joints and disc degeneration may lead to root impingement and, consequently, all the set of symptoms, which will be discussed in depth later [4].

According to Farfan [5], the degenerative process starts with minor trauma, which, repeated over several years, leads to spondylosis. A few years later, Farfan et al. [6] described how each segment of the lumbar spine is composed of a complex triad: two zygapophyseal joints and the disc. Because those three joints work in tandem, any disease that affects the disc will eventually compromise the joint and vice versa. The chief lesion mechanisms are torsional forces and compression overload [7].

Farfan also describes how the degenerative process starts between the fourth and fifth lumbar vertebrae and that after that level is compromised, based on the three-joint theory, the degeneration progresses to the proximal and distal adjacent levels. Thus, it becomes a diffuse disease that affects multiple levels of the lumbar spine. The anatomic changes are described next.

The zygapophyseal joints are diarthrodial, having an articular surface, a synovial membrane, and a capsule made of collagen; they are filled with synovial fluid [8]. Their degenerative process follows a sequence described by Lewin in 1964 [9]: it starts with a synovial reaction, followed by fibrillation of the joint surface, gross degeneration of the cartilage, osteophyte formation, joint process fracture, and finally loss of the joint's natural shape, leading to instability.

The third component of this complex joint is the intervertebral disc, the largest nonvascular tissue in the human body [10], which comprises three structures: the nucleus pulposus, the annulus fibrosus, and the terminal plates [3]. Each one of these structures has its own anatomy and unique constitution, and considerable importance [10]. The annulus fibrosus is made of type I collagen, distributed in circular layers, and resistant to traction forces. The nucleus pulposus is made basically of proteoglycans, water, and type II collagen, as well as countless elastin fibers [11]. Nutrition of the disc cells occurs through diffusion, in which vessels in the subchondral space, adjacent to the terminal plate's hyaline cartilage, carry oxygen, glucose, and small molecules, thus maintaining the disc's homeostasis [12]. Such homeostasis allows the nucleus pulposus to withstand compressive forces without collapsing and forces to be homogeneously transferred to the annulus fibrosus in all spine movements [13].

Another anatomic area that may go unnoticed is the lateral region, including the intervertebral foramen. Lee et al. [14] subdivided this region into three zones: the afferent zone, located in the subarticular region, medially to the pedicles; the intermediate or middle zone, located below the pars interarticularis; and finally, the efferent zone, comprising the intervertebral foramen. The latter is very important in surgical cases, because a lack of identification may lead to incorrect decompression and persistence of symptoms after surgery [15]. The foramen is a relatively large orifice, which often contains the dorsal root ganglion, coated with a layer of fat for the protection of neural structures. It is delimited anteriorly by the posterior vertebral wall, proximally by the inferior edge of the superior pedicle, inferiorly by the superior edge of the inferior pedicle, and posteriorly by the zygapophyseal joints and the yellow ligament.

## 2. Historical Background

The degenerative process can also be observed in this region, where diffuse disc bulging can also be seen, associated with loss of height—all leading to a reduced diameter of the vertebral canal. The zygapophyseal joints are also directly associated with foramen stenosis, because their hypertrophy may or may not be associated with the presence of osteophytes, thus causing radicular compression. In this case, sciatica may be observed, mimicking the symptoms of disc herniation [16].

The progression of the degenerative disease still remains truly unknown [17]. However, many concepts have already been postulated. The first one concerns the definition of instability; it is defined as “excessive mobility, neural compression, or deformity.” The presence of instability may be associated with a variety of clinical and anatomic manifestations [18].

Kirkaldy and Willis described the degenerative process in terms of evolution and divided it into three phases. However, the duration of each stage is unknown. The first phase was described as a dysfunction in which the disc exhibits chiefly biochemical changes. The second phase was called instability in which degenerative processes in the disc lead to an increase in the segment’s motion; this is when disc herniation can occur. Finally, there is the stabilization phase in which disc height reduction, facet hypertrophy, and changes in the yellow ligament occur [18]. This phase of disc degeneration is the most important for the development of the present study, because the aforementioned changes lead to a reduction of the vertebral canal diameter and to narrow lumbar spinal canal syndrome—the disease that is the object of this study.

As was described by Kirkaldy and Willis, the cascading degeneration does not have a definite phase, but in the stabilization phase, lumbar canal stenosis can be observed. It may or may not cause symptoms, but if symptoms do occur, this is commonly observed in patients above 50 years of age. Currently, the most commonly performed type of spine surgery in patients over 65 years old in the United States is decompression of cauda equina roots [19].

Because the population is aging and life expectancy is increasing, we were motivated to conduct this project.

Narrow lumbar spinal canal syndrome comprises a number of symptoms and varied clinical features [20], which is further discussed below.

Vertebral canal stenosis, as defined by Verbiest [2], corresponds to narrowing of the vertebral canal, the lateral recess, and the intervertebral foramen, causing compression of neural elements. Vertebral canal stenosis can be divided into two main groups: congenital and acquired [21]. These main groups were further subdivided: congenital stenosis into idiopathic and achondroplastic, and acquired stenosis into degenerative, combined, spondylotic, iatrogenic, post-traumatic, and metabolic [21].

Narrow lumbar spinal canal syndrome may be confused with many other diseases. Such diseases must always be considered, and a detailed clinical examination may make all the difference. Among the conditions that should be investigated are disc herniation, vascular

claudication, tumors, peripheral neuropathy, arthrosis of the hip or knee, and compressive insufficiency fractures [16].

Hall et al. [22] described symptomatic canal stenosis in detail. They described that patients complain of progressive lumbar pain, associated with an incipient pain and numbness in the distal extremities. Neurogenic claudication, the commonest symptom, is characterized by pain and weakness starting in the buttocks and thighs that becomes gradually worse in the orthostatic position and during walking, but improves after sitting down or leaning forward. Less often, one can find unilateral radiculopathy [23]. Symptoms become more acute with the disease's natural progression [24].

The progression of the disease is uncertain: according to Johnson, 70% of patients remain stable for a 4-year period, 15% improve, and 15% tend to become worse. The progression to cauda equina syndrome is extremely rare, but must always be investigated, particularly because of the possibility of other causes, but also because it is an absolute indication for urgent surgery [25].

Elderly patients may present a clinical condition very similar to neurogenic claudication, an entity called vascular claudication, associated with atherosclerosis. The pain following a walk is very similar to that in neurogenic claudication. Physical examination then becomes essential, because in a detailed examination, one can observe impotence in men, dystrophic skin, loss of hair, nail dystrophy, cyanosis, and reduced peripheral pulse. Such symptoms may be essential for the latter diagnosis [22].

### 3. Physical Examination

The best diagnostic test to distinguish both syndromes was described by Van Gelderen [26]. He had patients riding a stationary bicycle. Patients with lumbar canal stenosis tolerate the exercise, because the forward-leaning position causes symptoms to improve, whereas patients with vascular claudication do not tolerate the exercise, because the hypoxia caused by the underlying disease causes pain and peripheral cyanosis. Another very relevant sign in narrow canal syndrome is improvement when walking uphill and worsening when walking downhill, always associated with the flexion or extension of the trunk [27, 28]. It is postulated that the improvement associated with flexion and extension is directly related to stretching or folding of the yellow ligament. Trunk flexion causes tension in this ligament, thus increasing its diameter, whereas trunk extension causes it to fold into the spinal canal, thus further narrowing the canal that is already narrowed by the degenerative process [29, 30].

The physical examination of a patient with lumbar canal stenosis starts with the careful observation, followed by a very thorough physical examination. One must always consider the differential diagnosis from the other above-mentioned conditions; however, when compared to disc herniation, there are some subtle differences, such as age above 50 years, insidious onset, improvement with trunk flexion and worsening with its extension, and localized motor weakness. Signs of dura mater tension and muscular contraction are rarely

found. Typically, a trunk flexion position is observed, due to the increased canal diameter in that position. The presence of a reduced arc of movement is associated with the joint's degenerative process and not directly with the lumbar canal stenosis. Analogous to Phalen's test, Kemp's test is described in the literature, in which the patient is kept in trunk extension for 30 s and claudication symptoms appear [31].

#### 4. Diagnostic

Radiological diagnosis includes several examinations: common radiography, computed tomography (CT), and magnetic resonance imaging (MRI). In selected cases, myelography or myelotomography may be necessary [32].

The study of neural function and conduction speed can be performed either by electroneuromyography or by sensitive-motor evoked potentials [32].

Radiographs must be obtained in four incidences: frontal, orthostatic lateral, flexion, and extension. Then one must look for degenerative changes, such as reduced disc space, sclerosis of vertebral plateaus, sclerosis and hypertrophy of articular facets, closeness of spinous processes, and the diameter of the intervertebral foramina. In dynamic radiography, one can notice the presence of anteroposterior translation [33].

Computed tomography is a very important advance in the diagnosis of vertebral stenosis, because it shows important bone details, including the central canal, the lateral recess, the foramen, the joint facets, and their degree of degeneration [34]. CT is, however, criticized for its high rate of radiologic findings without correlation to the patient's symptoms [33].

MRI provides images of soft tissue with excellent quality, including ligaments, neural tissue, and the intervertebral discs. It is more sensitive for diagnosing lumbar stenosis than tomography. MRI findings include signal weakening at T2, with dehydration and rupture of the annulus in multiple discs; changes in terminal plates; void signal; enlarged yellow ligaments; and reduced vertebral canal [35].

For many years, myelography was the gold-standard exam for diagnosing lumbar stenosis, but although today's water-soluble contrast is less toxic, patients still have nausea, vomiting, headache, and dizziness. Myelography is an invasive exam, although it shows the dimensions of the dural sac and the neural roots in detail. Myelography findings include the partial or total interruption of contrast flow, and the dynamic examination may reveal a dynamic compression of neural structures [36]. It should be noted that electromyography is not routinely used in lumbar stenosis, because 80% of symptomatic patients have changes in one or both legs, making it necessary only for differential diagnosis, particularly to distinguish it from diseases that affect peripheral nerves [37].

The canal's diameter may be calculated by several different techniques. We used Hamanishi's technique, widely used [38], on which the calculation to determine the presence of stenosis is based. That is, Hamanishi considers a diameter of less than 100 mm<sup>2</sup> to define stenosis in patients with clinical symptoms and characteristic images [39].



The treatment of lumbar canal stenosis may be divided into two main types: clinical or conservative and surgical [40], each of them comprising several different modalities.

When a thorough clinical examination has been performed and there is confirmation from imaging exams, electrodiagnosis is not needed, as results are often inconclusive and, when positive, do not have an influence on either the clinical or the surgical treatment [41].

Generally, clinical treatment is preferred by over 50% of patients [42], and they mostly evolve satisfactorily. However, a small fraction suffers a more severe progression, with more unfavorable natural history and serious, limiting symptoms [43].

Many lumbar canal stenosis patients have symptoms of unilateral radiculopathy. In such cases, the most likely cause is herniation, which may affect a root in an already stenotic canal. When this happens, treatment should be more focused on the disc herniation. Despite the large number of articles in the literature, there is no consensus about when to operate such patients and, if surgery is performed, what the best technique would be [44].

## 5. Treatment Options

Drug treatment does not offer many possibilities. The indiscriminate and frequent use of anti-inflammatory medications for chronic lumbar pain does not have a proven satisfactory response [45] and may be associated with gastrointestinal and renal complications. Its use should be very restricted and avoided in elderly patients with narrow lumbar spinal canal syndrome [45].

Simple painkillers, muscle relaxants, and opioids may be of value. They are indicated for treating and controlling the pain but have no effect on the treatment of neurogenic claudication [45]. Gabapentin has been shown to be a safe medication; it may be taken orally and has a positive effect on patients with neurogenic claudication and the sensory alterations, which are very common in these patients [46].

Corticosteroids are also used indiscriminately. The idea is that there is an inflammatory process associated with the mechanical compression that could benefit from the medication, but this theory was not proven by Natour's study [47].

Physiotherapy, or more broadly rehabilitation, is a second non-surgical approach. Manual therapy, stretching, and muscular strengthening play an important role, in addition to the exercises. Patients who suffer from canal stenosis have, in addition to pain, a significant muscle loss, which severely limits their activities and progressively worsens their clinical condition, which leads to further impairments [48, 49].

The recommended activities include manual therapy, strengthening, and walking training, as well as exercises that improves proprioception. In addition, weight loss is important, because obese patients have been described to have a worse prognosis [47]. Cycling is a very much recommended activity, not only because patients tolerate it well, but it also allows them to

improve their conditioning and does not impact other joints that may also be degenerated, such as the hip and the knee [50].

Zarife et al. [51], in a study comparing two types of conservative treatments—physiotherapy and peridural corticosteroids—concluded that, in a 6-month follow-up, both methods were effective in improving the patients' condition and ability to walk, which suggested that clinical treatment is important and effective in these delicate and active patients.

Peridural corticosteroids are another type of non-surgical treatment for narrow lumbar spinal canal syndrome, as opposed to oral corticosteroids, which were shown to be ineffective for this condition [47]. Peridural corticosteroids have some advantages, which are discussed below. There are several possibilities for their administration, with or without radiography, as well as several techniques: interlaminar, caudal, and transforaminal. Despite their limited benefits, their use may have lasting efficacy in many patients [52].

Cosgrove et al. [53] published an article in 2011 in which the efficacy of peridural corticosteroids was evaluated and showed that women obtained greater benefits than men and that clinical results were not related to MR findings, which was also found in Natour's study [47]. Although Cosgrove et al. [53] observed better results among women, as per the general literature, women are normally affected compared to men.

Similarly to the above-mentioned article, Charles et al. [54] showed that peridural corticosteroids produce a satisfactory response in lumbar stenosis. The results of the study showed that patients with associated radiculopathy have a better response than do patients with claudication and that 25% of patients respond more favorably up to 2 years after the procedure.

However, we also found some articles in which the use of peridural corticosteroids did not deliver the expected satisfaction, in addition to causing complications such as meningitis, arachnoiditis, aseptic meningitis, and increased serum corticosteroids [55]. Fukusaki et al. [56] compared the use of analgesics in isolation and in combination with peridural corticosteroids and reported no complications; however, the results after 3 months were unsatisfactory, with all symptoms returning.

Surgical treatment is considered the last resort for patients with treating lumbar canal stenosis. Because surgery is performed in patients over 65 years of age, there is significant morbidity and mortality, which increase with associated diseases and patient age, making it mandatory to assess the risks and benefit of the surgery [57].

Airaksinen's study [58] showed that patients over 50 years old who underwent decompression and arthrodesis evolved with a significantly reduced ability to return to work. That reduction was even greater in older patients.

There are articles that report surgical results, with conflicting results. Hurri et al. [59], in a 12-year follow-up study, did not find any differences between surgical and non-surgical results, showing that regardless of surgery, the outcome is the same. Another study comparing operated and non-operated patients was the Maine Lumbar Spine Study [60, 61], in which operated patients were followed up for a period from 4 to 10 years. Results showed that operated patients had better postoperative results than non-operated patients, with an average



of 72% satisfaction among the former and 52% among the latter at 4 years of follow-up. The same comparison made at 10 years showed inferior results, but operated patients still had a perception of improvement.

Turner et al. [62] performed a meta-analysis and found that 64% of patients showed good results after surgical treatment, for a period varying from 3 to 6 years.

Surgical treatment is indicated when clinical treatment fails or neurological symptoms worsen [63]. Today, there are several different surgical techniques. The classical technique is laminectomy, performed by an incision along the midline followed by decompression, removing up to 50% of facets. In addition, there are minimally invasive techniques, such as opening and decompressing only one side of the lamina, which is called recalibration [64]. Interspinous spacers have been recently included in the surgical arsenal for canal stenosis, but studies are still under way, and there are no studies yet evaluating for an adequate follow-up period. For this reason, the actual benefit of this kind of surgery is not yet well established. However, it is known that it does offer some advantages, such as short hospitalization periods and limited bleeding. Its indication takes into account that by tensioning the yellow ligament, the canal diameter is increased [65].

The median approach with broad exposure of the spine has the advantage of satisfactorily exposing the spinal canal, which allows the intervertebral foramina to be viewed, broad decompression to happen up to the efferent zone with direct view, and roots to be evaluated. However, care must be taken to preserve half of the facets; otherwise, postoperative instability will occur as an iatrogenic complication that may compromise the results for the patient. The main problem with this broad approach is blood loss, which may be large or even catastrophic in some cases, because muscular lesion leads to large arterial and venous bleeding [63].

The indication for instrumentation and fusion varies in the literature, with some authors indicating fusion in the presence of degenerative spondylolisthesis or if there is a translation greater than 5 mm in dynamic X-rays. Instrumentation may also be indicated in cases of degenerative scoliosis in which the neural foramen is compressed on the concave side of the curvature and resection of more than 50% of the articular facet is needed in order to decompress the root stuck inside [66, 67].

The minimally invasive approach in spinal canal stenosis associated with foraminal stenosis may be indicated for patients with lumbar and radicular pain associated with stenosis in imaging exams, but its main contraindication is the presence of instability in X-rays, associated with a scoliosis of more than 10° in X-rays, in the orthostatic position. The main complication is recurring symptoms, in approximately 20% of cases, with reoperation being necessary, with broad exposure of the spine [68].

Interspinous spacers are a new generation of implants. Their mechanism of action is by blocking extension, as well as tensioning the stenosis level, which theoretically increases the spinal canal diameter. Studies have shown that such an increase may reach 20% of the initial diameter [69], but these studies are questioned due to the possibility of commercial interests. They are indicated for lumbar canal stenosis patients with two levels of stenosis, but they are

not used in the L5-S1 level and are contraindicated for patients with degenerative spondylolisthesis or radiological signs of instability [70].

Postoperative care of lumbar canal stenosis patients may vary slightly, but basically, patients are instructed to walk on the first day after surgery. Longer rest is indicated for patients with incidental durotomy, in which case the recommendation is at least 2 days rest. Deambulation with the aid and training by a physiotherapist is very important [71]. Rehabilitation exercises must include stretching the posterior muscles of the thighs and legs, training trunk flexibility, and strengthening the abdominal and paravertebral muscles. Improving cardiopulmonary capacity is also a target of rehabilitation, always respecting the patients' limits [72].

The need for orthosis is very much relative. Their use is generally not indicated. In osteoporotic patients, when there is the risk of an acute failure of implants, their use may be indicated for a period of up to 6 weeks, but overall, the literature is highly controversial about this subject [73].

The complications observed in surgery for lumbar canal stenosis may be divided into complications in the operated area and systemic complications. The most commonly observed systemic complications are urinary retention, worsening of heart failure in previously affected patients, delirium, and thoracic pain. Such symptoms are usually temporary, but they increase hospitalization time [74].

Surgical complications vary according to the series. Jolles et al. [75] report sensorial and motor deficit, dura mater lesions with cerebrospinal fluid fistula, surgical site hematoma, and superficial and deep infection.

Epidemiologically, surgery for lumbar canal stenosis has the same incidence of complications as knee arthroplasties, but greater than hip arthroplasties [76]. Mortality is currently at an average of 10%, but it increases with patient age. Clinical complications vary from 3 to 31% [77]. However, the most common complication observed in lumbar canal stenosis surgery is incidental durotomy, with an average incidence of 16%, increasing in case of reoperation [78]. Cerebrospinal fluid fistula, with leakage of cerebrospinal fluid, is the chief cause of reoperation in the first 2 days after surgery [79].

## 6. Conclusion

In conclusion, lumbar canal stenosis is a complex syndrome, which comprises degenerative processes in the lumbar spine. This degeneration may lead to a painful and limiting clinical condition, which must always be investigated through an exhaustive study of imaging examinations. Even though treatment is varied, with a large number of possibilities found in the literature, studies usually compare different techniques, either surgical or conservative, to find the most effective one. Apparently, the surgical approach with decompression, either associated with arthrodesis or not, has provided not only the best clinical results but also a greater incidence of complications and mortality, which must always be weighed together with the patient before surgery.

## Author details

Luiz Cláudio Lacerda Rodrigues

Address all correspondence to: [luizclaudiolacerdarodrigues@gmail.com](mailto:luizclaudiolacerdarodrigues@gmail.com)

1 Santa Marcelina Medical School, São Paulo, Brazil

2 Oswaldo Cruz German Hospital, Rua Treze de Maio, São Paulo - SP, Brazil

## References

- [1] Verbiest H. A radicular syndrome from developmental narrowing of the lumbar vertebral canal. *J Bone Joint Surg Br*;36:230–237, 1954.
- [2] Verbiest H. The significance and principles of computerized axial tomography in idiopathic developmental stenosis of the bony lumbar vertebral canal. *Spine*;4:369–378, 1979.
- [3] Kirkaldy-Willis WH, Paine KW, Cauchoix J, McIvor G. Lumbar spinal stenosis. *Clin Orthop*;99:30–50, 1974.
- [4] Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, Reilly J. Pathology and pathogenesis of lumbar spondylosis and stenosis. *Spine*;3(4):319–328, 1978.
- [5] Farfan HF. Effects of torsion on the intervertebral joints. *Can J Surg*;12:336–341, 1969.
- [6] Farfan HF, Sullivan JB. The relation of facet orientation to intervertebral disc failure. *Can J Surg*;10:179–18, 1967.
- [7] Yong-Hing K, Reilly J, Kirkaldy-Willis WH. The ligamentum flavum. *Spine*;1:226–234, 1976.
- [8] Sullivan JD, Farfan HF, Kahn DS. Pathological changes with intervertebral joint rotational instability in the rabbit. *Can J Surg*;13:71–79, 1971.
- [9] Lewin T. Osteoarthritis in lumbar synovial joints. *Acta Orthop Scand Suppl*;73:1–112, 1964.
- [10] Yong-soo C. Pathophysiology of degenerative disc disease. *Asian Spine J*;3(1):39–44, 2009.
- [11] Borgesen SE, Vang PS. Herniation of the lumbar intervertebral disk in children and adolescents. *Acta Orthop Scand*;45:540–549, 1974.
- [12] Key JA. Intervertebral disc lesions in children and adolescents. *J Bone Joint Surg Am*; 32:97–102, 1950.

- [13] Parisini P, Di Silvestre M, Greggi T, Miglietta A, Paderni S. Lumbar disc excision in children and adolescents. *Spine*;26:1997–2000, 2001.
- [14] Lee CX, Ranching W, Glenn W. Lateral lumbar spinal canal stenosis. Classification, pathology anatomy and surgical decompression. *Spine*;13:313–320, 1980.
- [15] Haward S, Glover MJ. Lumbar spinal stenosis: historical perspectives, classification, and pathoanatomy. *Semin Spine Surg*;6(2):69–77, 1994.
- [16] Thomas SA. Spinal stenosis: history and physical examination. *Phys Med Rehabil Clin N Am*;14:29–39, 2003.
- [17] Inoue Nozomu MD, Orias AE. Biomechanics of intervertebral disc degeneration. *Orthop Clin North Am*;42(4):487–499, 2011.
- [18] Kirkaldy-Willis WH, Farfan HF. Instability of the lumbar spine. *Clin Orthop Relat Res*. 1982 May;(165):110-23.
- [19] Deyo RA, Gray DT, Kreuter W et al. United States trends in lumbar fusion surgery for degenerative conditions. *Spine*;30:1441–1445, 2005.
- [20] Epstein NE, Maldonado VC, Cusik JF. Symptomatic lumbar spinal stenosis. *Surg Neurol*;50:3–10, 1998.
- [21] Arnoldi CC, Brodsky AE, Cauchoix J et al. Lumbar spinal stenosis and nerve root entrapment: syndromes, definition and classification. *Clin Orthop*;115:4–5, 1976.
- [22] Hall S, Bartleson JD, Onoforio BM et al. Lumbar spinal stenosis. Clinical features, diagnostic procedures and results of surgical treatment in 68 patients. *Ann Intern Med*; 103(2):271–275, 1985.
- [23] Naylor A. Factors in the development of the spina stenosis syndrome. *J Bone Joint Surg Br*;61(3):319–328, 1978
- [24] Blau JN, Logue V. Intermittent claudication of the cauda equina. *Lancet*;1:1081–1086, 1961.
- [25] Johnsson KE, Rosen I, Uden A. The natural course of lumbar spinal stenosis. *Clin Orthop*;279:82–86, 1992.
- [26] Dyck P, Doyle JB. Bicycle test of Van Gelderen in diagnosis of intermittent cauda equina compression syndrome. *J Neurosurg*;46:667–670, 1997.
- [27] Denn HG, Zimmerman RS, Lyons MK et al. Measurement of exercise tolerance on the treadmill test in patients with symptomatic lumbar spinal stenosis: a useful indicator of functional status and surgical outcome. *J Neurosurg*;83:27–30, 1995.
- [28] Fritz JM, Erhard RE, Delitto A et al. Preliminary results of the two-stage treadmill test as a clinical diagnostic tool in the differential diagnosis of the lumbar spinal stenosis. *J Spinal Disord*;10:410–416, 1997.

- [29] Olszewski AD, Yaszemski MJ, White AA III. The anatomy of the human lumbar ligamentum flavum: new observations and their surgical importance. *Spine*;21:2307–2312, 1996.
- [30] Sairyo K, Biyani A, Goel V et al. Pathomechanism of ligamentum flavum hypertrophy: a multidisciplinary investigation based on clinical, biomechanical, histologic, and biologic assessments. *Spine*;23:2649–2656, 2005.
- [31] Katz JN, Dagas M, Stucki G et al. Degenerative lumbar spinal stenosis: diagnostic value of the history and physical examination. *Arthrit Rheum*;38(9):1236–1241, 1995.
- [32] Spengler DM. Current concepts review. Degenerative stenosis of the lumbar spine. *J Bone Joint Surg*;69-A:305–308, 1987.
- [33] Dyck P, Doyle JP Jr. “Bicycle Test” of Van Gelderen in diagnosis of intermittent cauda equina compression syndrome. *Case Report/Neurosurgery*;46:667–670, 1977.
- [34] Arnoldi CC, Brodsky AE, Cauchoix J, et al. Lumbar spinal stenosis and nerve root entrapment syndromes. Definition and classification. *Clin Orthop Relat Res*. 1976 Mar-Apr;(115):4–5.
- [35] Frymoyer JW, Newberg A, Pope MH, Wilder DG, Clements J, Macpherson B. Spine radiographs in patients with low-back pain. An epidemiological study in men. *J Bone Joint Surg Am*;66(7):1048–1055, 1984.
- [36] Herkowitz HN, Garfin SR, Bell GR, Bumphrey F, Rothman RH. The use of computerized tomography in evaluating non-visualized vertebral levels caudad to a complete block on a lumbar myelogram. A review of thirty-two cases. *J Bone Joint Surg Am*;69(2):218–224, 1987.
- [37] Czervionke LF. Lumbar intervertebral disc disease. *Neuroimaging Clin N Am*;3:465–485, 1993.
- [38] Hamanishi C, Matukura N, Fujita M, Tomihara M, Tanaka S. Crosssectional area of the stenotic lumbar dural tube measured from the transverse views of MRI. *J Spinal Dis*;7:388–393, 1994.
- [39] Owlia MB, Salimzadeh A, Alishiri G, Haghighi A. Comparison of two doses of corticosteroid in epidural steroid injection for lumbar radicular pain. *Singapore Med J*;48(3):241–245, 2007.
- [40] Amundsen T, Weber H, NOrdal HJ, Magnaes B et al. Lumbar spinal stenosis: conservative or surgical management? A prospective 10 year study. *Spine*;25(11):1424–1435, 2000.
- [41] Haig AJ, Tong HC, Yamakawa KS, Quint DJ et al. The sensitivity and specificity of eletrodiagnostic testing for the clinical syndrome of lumbar stenosis. *Spine*;30(23):2667–2676, 2005.



- [42] Atlas SJ, Kelle RB, Wu YA et al. Long-term outcomes of surgical and nonsurgical management of lumbar spinal stenosis: 8 to 10 years results from the Maine Lumbar Spine Study. *Spine*;30(8):936–943, 2005.
- [43] Waters WC, Bono CM, Gilbert TJ et al. An evidence based clinical guideline for the diagnosis and treatment of degenerative lumbar spondylolistheses. *Spine J*;8(2):305–310, 2008.
- [44] Chou R, Qaseem A, Snow V et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American college of physicians and the American Pain Society. *Ann Intern Med*;147(7):478–491, 2007 Oct 2.
- [45] Stephane G, Steven JA . Lumbar spinal stenosis. *Best Pract Res Clin Rheumatol*;24(2): 253–265, 2010 April.
- [46] Yaksi A, Ozgonenel L, Ozgonenel B. The efficiency of gabapentin in patients with lumbar spinal stenosis. *Spine*;32(9):939–942, 2007.
- [47] Rodrigues LC, Natour J. A double-blind, randomized controlled, prospective trial assessing the effectiveness of oral corticoids in the treatment of symptomatic lumbar canal stenosis. *J Negat Results Bioemed*;13:13, 2014.
- [48] Rittenberg JD, Ross AE. Functional rehabilitation for degenerative lumbar spina stenosis. *Phys Med Rehabil Clin N Am* (14):111–120, 2003.
- [49] Whrtiman JM. Flynn TW, Frotz JM. Nonsurgical management of patients with lumbar spine stenosis: a literature review and a case series of three patients managed with physical therapy. *Phys Med Rehabil Clin N Am*;14(1):77–101, 2003.
- [50] Whitman JM, Flynn TW, Chids JD, Wainer RS et al. A comparassion between two physical therapy treatment programs for patients with lumbar spinal stenosis: a randomized clinical trial. *Spine*;31(2):2541–2549, 2006.
- [51] Zarife K, Suheda O, Kncuy S et al. Effectiveness of physical therapy and epidural steroid injections in lumbar spinal stenosis. *Spine*;34(10):985–989, 2009.
- [52] Parr AT, Diwan S, Adbdi S. Lumbar interlaminar epidural injections in managing chronic low back and lower extremity pain: a systematic review. *Pain Physician*;12(1): 163–188, 2009 Jan–Feb.
- [53] Cosgrove JL, Berlote M, Chase SL et al. Epidural steroid injections in the treatement of lumbar spinal stenosis efficacy and predictability of successful response. *Am J Phys Med Rehabil*;90:1050–1055, 2011.
- [54] Charles DR, Neil K, Ralph B, Kathy V. A retrospective analysis of the efficacy of epidural steroid injections. *Clin Orthopaed Relat Res*;228:270–273, 1998.
- [55] Cuckler JM, Bernini PA, Wiesel SW et al. The use of epidural steroids in the treatment of lumbar radicular pain. *J Bone Joint Surg Am*;67:63–66, 1995.

- [56] Fukusaki M, Kobayashi I, Tetsuya H Sumikawa K. Symptoms of spinal stenosis do not improve after epidural steroid injection. *Clin J Pain*;14:148–151, 1988.
- [57] Shabat S, Arizon Z, Folman Y et al. Long-term outcomes of decompressive surgery for lumbar spinal stenosis in octogenarians. *Eur Spine J*;17:193–198, 2008.
- [58] Airaksinen O, Hermo A, Saari T. Surgical treatment of lumbar spinal stenosis: patients postoperative disability and working capacity. *Eur Spine J*;3:261–264, 1994.
- [59] Hurri H, Slati P, Soini J et al. Lumbar spinal stenosis: assessment of long-term outcome 12 years after operative and conservative treatment. *J Spinal Disord*;11:110–115, 1998.
- [60] Atlas SJ, Keller RB, Robson D et al. Surgical and nonsurgical management of lumbar spinal stenosis: four-years outcomes from the Maine lumbar spine study. *Spine*;25:556–562, 2000.
- [61] Atlas SJ, Keller RB, Wu YA et al. Long-term outcomes of surgical and nonsurgical management of lumbar spine study. *Spine*;30:936–943, 2005.
- [62] Turner JA, Ersek M, Herron L, Deyo R. Surgery for spinal stenosis: attempt meta-analysis of the literature. *Spine*;17:1–8, 1992.
- [63] Ra'kerry KR, Douglas DN, Daniel EG, Kornelis AP, Steven CL. Lumbar spinal stenosis. *Curr Orthop Pract*;19(4):351–356, 2008.
- [64] Asgarzadie F, Khoo LT. Minimally invasive operative management for lumbar spinal stenosis: overview of early and long-term outcomes. *Orthop Clin N Am*;38:387–399, 2007.
- [65] Zucherman JF, Hsu KY, Hartjen CA, et al. A prospective randomized multicenter study for the treatment of lumbar spinal stenosis with the X stop interspinous implant: 1-year results. *Eur Spine J*;13:22–31, 2004.
- [66] Deburg A, Bex M, Lassale B, Bitan F. Technique chirurgicale dans le traitement des Stenoses du canal Lombar. *Acta Orthop Belg*;53:412–419, 1987.
- [67] Spinavak J. Degenerative lumbar spinal stenosis. *J Bone Joint Surg (Am)*;80-A:1053–1066, 1998.
- [68] Yamada K, Matsuda H, Nabeta M et al. Clinical outcomes of microscopic decompression for degenerative lumbar foraminal stenosis: a comparison between patients with and without degenerative lumbar scoliosis. *Eur Spine J*;20:947–953, 2011.
- [69] Richards JC, Manjubar S, Lindsey DP. The treatment mechanism of an interspinous process implant for lumbar neurogenic intermittent claudication. *Spine*;30:744–749, 2005.
- [70] Siddiqui M, Nicol M, Karadimas E et al. The positional magnetic resonance imaging changes in the lumbar spine following insertion of a novel interspinous process distraction device. *Spine*;30:2677–2682, 2005.

- [71] Turner J, Ersek M, Herron L et al. Patient outcomes after lumbar spinal fussions. JAMA; 268:907–911, 1992.
- [72] Fairbank J, Frost H, Macdonald W, Yu L et al. Randomised controled trial to compare surgical stabilization of the lumbar spine with an intensive rehabilitation programme for patients with chronico low back pain: the MRC spine stabilization trial. Br Med J. 2005 May 28;1–7 330(7502): 1233.
- [73] Herkowitz HN, Garfin SR. Indications and surgical results of arthrodesis following spinal stenosis Spinal Stenosis. Philadelphia: Ed. Sauders. The Spine 1999;p. 806M-T. 132.
- [74] Fredmann B, Arizon Z, Zohar E et al. Observations on the safety and efficacy of surgical decompression for lumbar spinal stenosis in geriatric patients. Eur Spine J;11:571–574, 2002.
- [75] Jolles BM, Porchet F, Theumann N. Surgical treatment of lumbar spinal stenosis. J Bone Joint Surg (Br);83:949–953, 2001.
- [76] Alto TJ, Mallmivaara A, Kovacs F et al. Preoperative predictors for postoperative clinical outcome in lumbar spinal stenosis: systematic review. Spine;31(18):E648–E663, 2006.
- [77] Genevay S, Atlas SJ. Lumbar spinal stenosis. Best Pract Res Clin Rheumatol;24(2);253–265, 2010.
- [78] Eismont FJ, Wiesel SW, Rothman RH. Treatment of dural tears associated with spinal surgery. J Bone Joint Surg Am;63:1132–1136, 1981.
- [79] Sin AH, Caldito G, Smith D et al. Predictive factors for dural tear and cerebrospinal leakage in patients undergoing lumbar surgery. J Neurosurg Spine;5:224–227, 2006.

