

For residents

General considerations in lumbar spinal stenosis

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“The secret of being a bore is to tell everything”
Voltaire - *Sept Discours en Vers sur l'Homme*, 1738

Definition

Lumbar spinal stenosis (LSS) is a common and often disabling, well-recognized spinal disorder that generally occurs in the sixth or seventh decade of life, although it can uncommonly occur in younger individuals, congenital or acquired, focal or diffuse (multilevel), defining a osteo-ligamentous narrowing (congenital) or shrink (secondary – acquired) of the lumbar spinal canal, a conflict between the lumbar spinal canal with vertebral body osteophytes, hypertrophy of the ligamentum flavum, zygapophyseal joint, lumbar disc hernia or a combination of these and the content represented by the cauda quina roots, lumbar spinal roots and ganglia, generating a complex set of symptoms of which the hallmark is neurogenic claudication, physical findings and radiological abnormalities.

Lumbar spinal stenosis or “loss of epidural reserve” could affect the central lumbar spinal canal ± lateral recess ± the neuroforamen or any combination of these, causing nervous impingement and vascular structures

compression (1-4). It can coexist with cervical stenosis too.

It's important to underline that diagnosis of LSS should be defined by symptoms and clinical findings that must be supported by neuro-imagistic evidences. Computed tomography and magnetic resonance imaging are often non-specific and there may be discrepancies between clinical symptoms and imaging findings in cases of LSS. Example: for central stenosis according to the sagittal diameter of the lumbar spinal canal there are absolute lumbar spinal stenosis <10 mm and relative lumbar spinal stenosis 10 to 14 mm (5-7).

Spinal stenosis was first described only sixty five years ago by Verbiest (8): “It is characterized by narrowing of the spinal canal, the nerve root canals or the neural foramina and can produce a debilitating condition in older adults”

History (1-3) (9-18)

1803 – the first medical report of spinal stenosis: Portal in France postulated that back and leg pain could be caused by bone impingement on the nerves (1-3) (9-11).

1893 – first decompressive laminectomy: Lane in England did a to relieve a woman of cauda equina syndrome caused by spinal stenosis (1-3) (9-11)

1900 - connexion between lumbo-sciatalgic pain and nerve compression was first remarked by Sachs B and Fraenkel J. (12)

1945 Sarpyener M.A. reveal congenital stricture of the spinal canal (14)

1948 Van Gelderen C. suggest that ligamentum flavum hypertrophy could be a possible cause of lumbar spinal stenosis referring to two clinical observations. (1-3) (10-11)

1954 –Verbiest H.A. A. – a holland surgeon, defined first the clinical syndrome of lumbar stenosis in seven patients who had bilateral radicular pain and motor and sensory disturbances in the legs, caused by standing and walking and made first the difference between “narrow” and “shrink”.(15). He also remarked clinical signs in congenital lumbar spinal stenosis justified by mechanical nerve compression at sacroradiculography (16). (16)(115)(16)

1955 - Schlesinger (cited by 11) and 1972 - Epstein et al. (17) reported some cases and described the clinical and radiographic findings of facet syndrome. They pointed out the importance of the height of the intervertebral foramen on plain radiographs in narrow lateral recess cases. Since then several authors recognized and described lateral recess stenosis and lateral entrapment syndrome (18-20).

In the 1960s and 1970s spinal stenosis began to be recognized as an impairing condition, also during the 1970s and 1980s, many case reports showed successful surgical

treatment rates, but these were based on subjective assessment by surgeons.

1974 Kirkaldy-Willis P. Paine K, Cauchoix J, et al. precise the anathomo-pathological aspects in LSS and made correlation with clinical signs (21).

1978 Porter and colleagues discovered that individuals who experience back pain and other symptoms are likely to have smaller spinal canals than those who are asymptomatic (22)

1982 Rothman reported that a normal sized lumbar canal is rarely encountered in persons with either disc disease or those requiring a de-roofing (laminectomy) procedure (23).

1992, Johnsson, Rosén and Udén described the natural history of LSS (24) and concluded that a reasonable treatment option for lumbar stenosis is observation. Another investigators appreciate that significant neurologic deterioration is rare: 70% of patients reported no significant change in symptoms, 15% showed significant improvement, whereas 15% showed some deterioration (25)(26).

Frequency

LSS are frequent, the incidence of this condition has been reported to be 8–11% of population, generally occurs in the sixth or seventh decade of life, except congenital; in the 4th decade to men in central lumbar stenosis, in the 5-6 th decades to women in lateral stenosis. LSS is more frequent to woman: ratio woman/men: 3/1 to 5/1400,000 (30). 400,000 Americans are estimated to have spinal stenosis with increased incidence with the expansion of life-expectancy (3).

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Generally LSS are 5 times less frequent as lumbar disc hernia. No clear correlation exists between the symptoms of stenosis and race, occupation, sex or body type; the anvergence of the degenerative process which cannot be prevented by diet, exercise or lifestyle (1-3).

The anvergence of the problem is expanding: americans are estimated to have spinal stenosis with increased incidence with the expansion of life-expectancy, especially by increasing place of neuroimagistic procedures; prevalence 1 case at 1000 persons (27). Ubiquitous degeneration associated with aging may lead to stenosis of the spinal canal, especially along the lumbar spine. As a result of the changing societal age structure, the incidence of symptomatic lumbar spinal stenosis is increasing exponentially. Inpatients older than 60, lumbar spinal stenosis is found on magnetic resonance imaging in more than 20% of cases (28). Older patients' desires for mobility and functionality, combined with improved perioperative management, have resulted in a situation where surgical intervention is being increasingly considered. In the United States, the incidence of surgery has increased eight-fold from 1979 to 1992 (29) and in 2007 37,598 people were treated surgically for lumbar stenosis in the US, totally a bill of 1.65 billion dollars (25) (26).

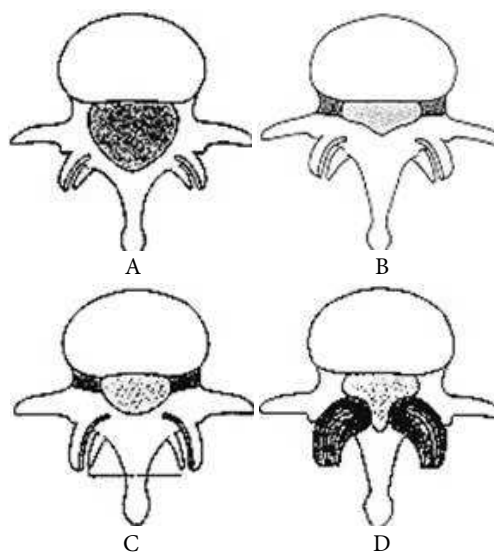
LSS - classifications

Anatomical classification (1-3) (31-33):

LSS are defined by the anterior-posterior diameter less than 15 mm (normal value 15-25 mm) and lateral recess less than 3 mm. It's to be remarked that adult cross section lumbar spinal area (av. 194 sq mm) are similar to those

seen of 4 years old. LSS could be (figure 1): segmentary (2 levels 45%, 1 level 37%, 3 levels 17%) or diffuse;

- central, 10% due to hypertrophic spurring, bony projection or ligamentum flavum/laminar thickening
- clover leaf ("fleur de lis"): laminar thickening with subsequent postero-lateral bulging
- medial - secondary to IAP hypertrophy
- lateral recess and foraminal stenosis 45%, secondary to SAP hypertrophy
- mixed 45%



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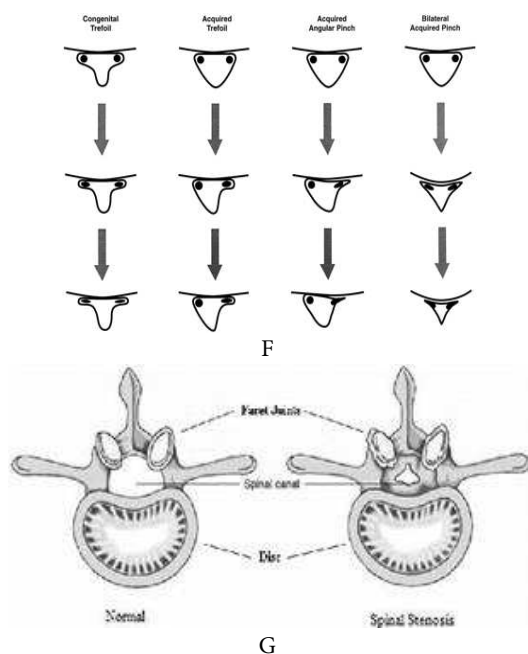


Figure 1 - LSS - modified after (1)(32): A normal aspect, B short pedicles reducing the antero-posterior diameter, C central stenosis, articular process are disposed in sagittal plane, D clover leaf central stenosis, E severe stenosis macroscopic aspect, F different shapes of spinal canal in spinal stenosis, G didactic schema in central stenosis

Aetiological classification (1-3) (10) (11) (21) (22) (30)

1. Congenital stenosis (narrowed lumbar channel) – described by Sarpyener at childrens (14), Verbiest in adults (8), are rare, occurring in only 5-9 % of cases. Congenital lumbar stenosis could be idiopathic or concomitant with anatomical variants: partial or total LSS; unilateral asymmetric, with discontinuity in stenosis; normal lumbar segment between 2 stenotic segments; normal central lumbar canal with lateral recess stenosis or associated with sacral stenosis. Anatomically lumbar constitutional stenosis are related to short

pedicles, and short, thick, vertical lamae; sometimes medial articular part are in contact with spinous process, short interspinous and interlamae spaces, the antero-posterior diameter is ≤ 15 mm and interpedicular diameter has a normal dimension or $<$ of 25 mm. Generally congenital stenosis are insufficient to generate clinical symptoms, but able to decompensate by secondary lesions (35) (36):

- dysplastic lesions: achondroplastic, acromesomelic, arterio-hepatic, diastrophic, osteo glophonic nanism, hypochondroplastic, thanatophoric, Smith-McCort syndrome, Weill-Marchesani syndrome, Gordon syndrome – all spinal canal could be stenotic
- dysostotic lesions: cheiro-lumbar, Gorlin baso cellular naevomatose, Wyers & Thier syndrome, acrodysostose

A rare cause of lumbar canal stenosis are idiopathic lumbar epidural lipomatosis (37).

2. Secondary (acquired) stenosis (shrink lumbar channel):

- nonspecific: degenerative changes more frequent in aged persons related to hypertrophic frequent asymmetric articular process; include central canal and lateral recess stenosis from posterior disc protrusion, zygapophyseal joint and ligamentum flavum hypertrophy/calcification, degenerative spondylolisthesis – characterized by forward displacement of a vertebra due to disc and facet degeneration (38), synovial cysts, lumbar epidural varices (39), lumbo-sacral transitional vertebra; degenerative process stages responsible for dysfunction, instability
- specific:
 - traumatic

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- systemic processes include: Paget disease, fluorosis, acromegaly, ankylosing spondylitis, renal osteodystrophy, neoplastic, infectious, lipomatous epiduritis of Cushing disease, tabetic arteriopathy

- iatrogenic changes result following surgical procedures such as laminectomy, fusion, discectomy, malpositioned osseous grafts used for osteosynthesis

3. Combined congenital and degenerative - in several cases generally degenerative stages made symptomatic a constitutional lumbar stenosis.

Pathophysiology of LSS

LSS is a slow, fluctuant progression disease, occur in all affected individuals. LSS is more frequent on L3-L4 and L4-L5, segments; several patho-physiological mechanisms could be related to this condition. No clear correlation exists between the symptoms of stenosis and race, occupation, sex, body type. The degenerative process cannot be prevented by diet, exercise or lifestyle. Acute cauda equina syndrome is rare. Degenerative process stages in LSS are: dysfunction, instability, stabilization and three main factors are responsible for the development of this spinal disorder (1-3) (40):

1. Disk protrusion as a consequence of disk degeneration of a motion segment lead to height segment loss, lateral recess, neural foramen and central canal narrowing. As a consequence it effects ligamentous laxity with increased segmental mobility, with additional strain especially on the facet joints. This subclinical instability of the segment generate

osseous hypertrophy, especially the facet joints, fibrotic hypertrophy of the ligamentum flavum, in addition to folding inwards subsequent to height loss. If these reactive processes do not succeed in stabilizing the segment, disk degeneration, laxity of capsules and ligaments may result in the manifest instability of spondylolisthesis.

2. The position of the spine may generate nerve root compression: the strain imposed by standing is in itself sufficient to result in hyperlordosis of the affected segment, with further protrusion of the ligamentum flavum into the spinal canal.

3. Both arterial ischemia as well as venous factors are implied in mechanical nervous roots compressions. Experimental studies have been made looking on lombar roots during walking, on normal asymptomatic patients and to those patients with claudication by LSS, using microendoscopic techniques, under local anesthesia (41).

During walking oxygen roots needs are demanding a supplementary arterial supply. The strain and weight imposed by walking results in decompensation of the vascular flow to the spinal nerves, which is mostly sufficient during rest.

If patient is bending anteriorly (figure 2), both the canal diameter and arterial supply is bigger. If that is not enough the patient need to sit down to reestablish the normal metabolic situation.

Also mechanical pressure on cauda equine is generating venous stasis affecting nervous influx by local demyelination and axonal degenerescence.

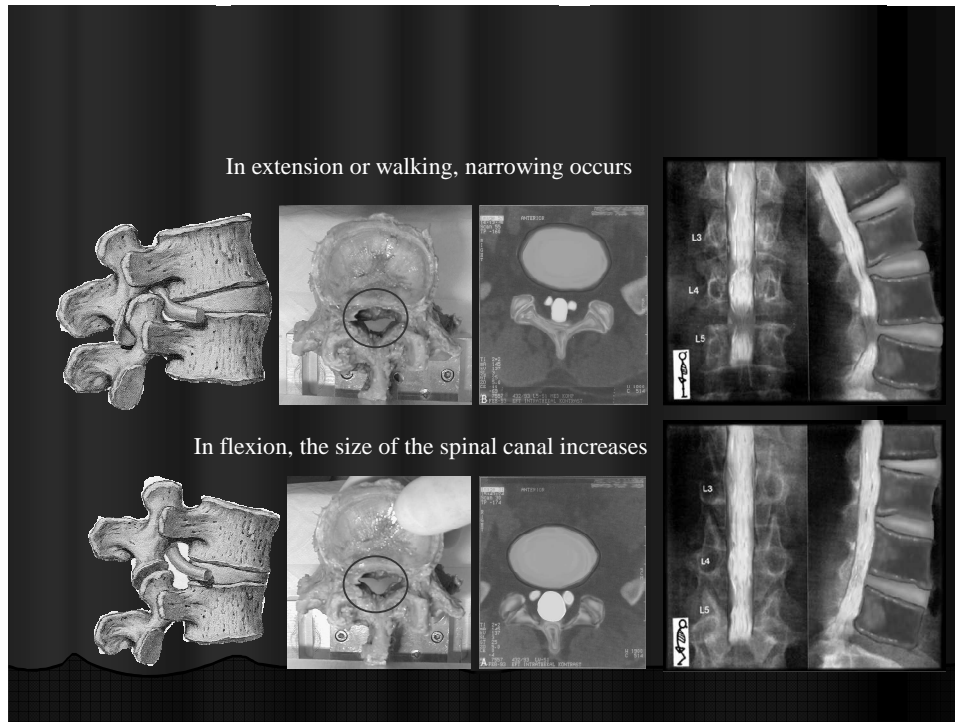


Figure 2 - LSS – modified after (1) (32): patients walk in flexion and prefer to sit and lean forward

In synthesis, in LSS the mechanisms explaining symptomatology are

Neural compression:

Anterior:

- disk protrusion or herniation
- osteotic overgrowth: osteophytes

Posterior:

- lamina & ligamentous hypertrophy (the role of TGF-β1, TIMP-1 and TIMP-2 on hypertrophy of the ligamentum flavum in spinal stenosis patients is a local phenomenon, not systemic)(42)

- facet hypertrophy, synovial cyst
- spinal lipomatosis, etc

Pain mechanisms may be due to:

- mechanical: chronic compression of the nerve root in LSS causes compromise of blood

flow leading to venous congestion, ischaemia, +/-microcirculation anomalies, intraneural edema. This then leads to the development of periradicular fibrosis. Neurogenic claudication likely arises from increased metabolic demands of the nerve root in the presence of vascular compromise and traction on the adhered nerve root when lower extremity movement occurs during walking

- instability
- biochemical component especially by cytokines when disc is involved - nucleus pulposus
- inflammatory–arthritis

Instability:

Defined by White and Panjabi (43): “the loss of the spine’s ability to maintain its

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patterns of displacement, under physiologic loads so there is no initial or additional neurologic deficit, no major deformity and no incapacitating pain”

- mechanical instability without clinical signs

- symptomatic instability: intermittent pain + clinical signs of LSS

Instability mechanisms are leading and responsible for spondylolisthesis and scoliosis may be due to:

- loss of discal integrity
- loss of ligamentous integrity
- loss of facetal integrity
- loss of supportive integrity:
- paraspinal and abdominal muscle tone and power

Conclusion

Pathologic determinants that result in a stenotic canal are protruding discs, ligamentum flavum, facet and lamina hypertrophy, lysis, degenerative scoliosis and rotational deformities.

References

1. Iacob G, Craciun M - Lumbar spinal stenosis (LSS): indications and operative treatment – the “Bucharest view”, German-Romanian Course Cluj-Napoca 24-25 May 2011
2. Iacob G, Craciun M - Personal experience in lumbar spinal stenosis (LSS), Romanian Neurosurgery 2011, XVIII, 4, 400-411
3. Iacob G. - Neurochirurgia in managementul coloanei dureroase, Congresul Asociației Române pentru Studiul Durerii, “Managementul durerii acute pentru medicii din ambulator” Howard-Johnson Grand Plaza, Bucuresti, 14-15. 10. 2011

4. AWMF-Leitlinie: Leitlinien der Deutschen Gesellschaft für Neurochirurgie, „Lumbale Spinal kanal stenose“ 2005, <http://www.uni-duesseldorf.de/awmf/ll-na/008-022.htm>
5. deGraaf I, Prak A, et al. - Diagnosis of lumbar spinal stenosis: a systematic review of the accuracy of diagnostic tests. Spine 2006, 31, 1168-1176
6. Beattie PF, Meyers SP, et al. - Associations between patient report of symptoms and anatomic impairment visible on lumbar magnetic resonance imaging. Spine 2000, 25, 819-828
7. Amundsen T, Weber H, et al. - Lumbar spinal stenosis. Clinical and radiologic features, Spine 1995, 20, 1178-1186
8. Verbiest H. - Primary stenosis of the lumbar spinal canal in adults, a new syndrome, Ned Tijdschr Geneesk. 1950, 94, 2415-33
9. Spivak J. - Degenerative spinal stenosis, J Bone Joint Surg 1998, 80, 1053-1066
10. Katz J, Dalgas M, et al. - Diagnosis of lumbar canal stenosis, Rheum Dis Clin North Am 1994, 20, 471-483
11. Garfin S, et al. - Spinal stenosis, AAOS Instructional Course Lectures 2000, 49, 361-374
12. Arbit E, Pannulo S. - Lumbar stenosis-a clinical review, Clin Orthop 2001, 384, 137-143
13. Sachs B, Frankel V. - Progressive and kyphotic rigidity of the spine, J Nerv Ment Dis 1900, 27, 1-15
14. Sarpyener MA. - Congenital stricture of the spinal canal, J Bone Joint Surg 1945, 27, 70-9
15. Verbiest H. - A radicular syndrome from developmental narrowing of the lumbar vertebral canal. J Bone Joint Surg Br 1954, 36(2), 230-237
16. Verbiest H. - Further experiences on the pathological influence of a developmental narrowness of the bony lumbar vertebral canal. J Bone Joint Surg Br 1955, 37(4):576-583
17. Epstein JA, Epstein BS, et al. - Sciatica caused by nerve root entrapment in the lateral recess: the superior facet syndrome. J Neurosurg 1972, 36(5), 584-589
18. Shenouda EF, Gill SS - Laminal fenestration for the treatment of lumbar nerve root foraminal stenosis. Br J Neurosurg 2002, 16(5), 494-497
19. Ciric I, Mikhael MA, Tarkington JA, Vick NA - The lateral recess syndrome: a variant of spinal stenosis. J Neurosurg 1980, 53, 433-443

20. Lee CK, Raushning W, Glenn W - Lateral lumbar spinal canal stenosis: classification, pathologic anatomy and surgical decompression. *Spine* 1988, 13(3), 313-320
21. Kirkaldy-Willis WH, Paine KWE, et al.: Lumbar spinal stenosis. *Clin Orthop*. 1974, 99, 30-50 1974
22. Porter RW, et al. - The spinal canal in symptomatic lumbar disc lesions, *J Bone Joint Surg Br* 1978, 60-B (4), 485-487
23. Rothman RH., Semeone FA, - *The Spine*. Philadelphia: Saunders. 1982., 518. ISBN 0-7216-7718-5
24. Johnsson KE, Rosén I, Udén A - "The natural course of lumbar spinal stenosis", *Clin. Orthop. Relat. Res.* 1992, (279), 82-86
25. Djurasovic M, Glassman SD, et al. - Contemporary management of symptomatic lumbar spinal stenosis, *Orthop. Clin. North Am.* 2010, 41 (2), 183-191
26. Genevay S, Atlas SJ. - Lumbar spinal stenosis, *Best Pract Res Clin Rheumatol* 2010, 24 (2), 253-65
27. Lurie J.D. - LSS, *Spine* 2003, 28, 616-620
28. Boden SD, Davis DO, et al. - Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am* 1990, 72, 403-408
29. Ciol MA, Deyo RA, et al. - An assessment of surgery for spinal stenosis: time trends, geographic variations, complications and reoperations. *J Am Geriatr Soc* 1996, 44, 285-290
30. Bridwell K. - Lumbar spinal stenosis. Diagnosis, management and treatment. *Clin Geriatr Med* 1994, 10, 677-701
31. Keim HA, Hajdu M, et al. - LSS, *Spine* 1985 May, 10(4), 338-344
32. Iqbal J. - Surgery For Lumbar Canal Stenosis: Micro or Macro? 9th Dubai Spine Conference May 14th-15th, 2008
33. Mohamed El-Fiki - Spinal stenosis; Is there a place for limited procedures? 9th Dubai Spine Conference, May 14th-15th, 2008
34. Arnoldi C, Brodsky A, et al. - Definition and classification of lumbar spinal stenosis and nerve root entrapment syndromes, *Clin Orthop* 1976, 115, 4-5
35. Garfin SR, Rauschnig W. - Spinal stenosis, *Instr Course Lect.* 2001, 50, 145-52
36. Resnick D. - Degenerative disease of the spine, in: Resnick D(ed): *Diagnosis of Bone and Joint Disorders* (4th ed). Philadelphia: W.B. Saunders 2002, 1382-1475
37. Thiagarajan G., Dhandapani S. - Idiopathic lumbar epidural lipomatosis: A rare cause of lumbar canal stenosis, *Pan Arab Journal of Neurosurgery* 2010, 14, 2, 117-118
38. Herkowitz H. - Degenerative lumbar spondylolisthesis *Spine* 1995, 20, 1084-1090
39. Farrokhi M.R., Niknam H. - Lumbar radicular pain caused by epidural varices, *Pan Arab Journal of Neurosurgery* 2010, 14, 2, 115-116
40. Porter R.W. - Spinal stenosis and neurogenic claudication, *Spine* 1996, 21: 2046-2052
41. Yong-Hing K, Kirkaldy-Willis W.H - The pathophysiology of surgery for diseases of the lumbar spine. *Orthop Clin North Am* 1983, 14, 491-504
42. Kim H.J. et al. - Serum Levels of TGF- β 1, TIMP-1 and TIMP-2 in Patients with Lumbar Spinal Stenosis and Disc Herniation, *Asian Spine Journal* 2007, 1, 1, 8-11
43. White A.A., Panjabi M.M. - *Clinical biomechanics of the spine*, 2nd ed, Lippincott, Philadelphia, PA, 1990