

Lumbar canal “stenosis:” Instability is the issue and stabilization is the treatment

The understanding of the clinical entity of “lumbar canal stenosis” has been based on the traditionally agreed upon and more than century, old concept that “old-age”-related disc degeneration, disc water content reduction, and disc space loss are the core pathogenetic issues that result in a cascade of “secondary” degenerative events such as osteophyte formation and ligamentum flavum hypertrophy that occupy space in the spinal canal, are neural compressive in nature and ultimately lead to the development of spinal canal and root canal stenosis. Alteration of spinal curvature and some degree of listhesis in the spinal segments are frequently observed, but in general, the lumbar spine is considered to be stable in lumbar canal stenosis. Although the issue of instability has been under discussion in various forms, its role as a primary pathogenetic factor has never been recognized or critically analyzed.

“Decompression” of compressed neural structures in a “stenotic” canal by laminectomy with or without foraminotomy is currently considered to be the gold standard treatment for lumbar canal stenosis. Whilst some surgeons prefer a relatively midline or small laminectomy, some prefer wide laminectomy that sometimes includes medial facetectomy to expose the dural tube and the nerve roots in the surgical field. Minimal invasive decompression using endoscope is currently popular. Some surgeons prefer simultaneous spinal fixation to avoid delayed iatrogenic spinal instability that could be a result of wide bone resection during laminectomy.

In 2013, for the first time in the literature, we proposed that the core issue in the pathogenesis of lumbar canal stenosis is “vertical” spinal instability. All the so-called degenerative changes that seem radiologically pathological and neural compressive in nature, are secondary and naturally “protective” or “adaptive” in nature, are the indicators of


segmental instability, and are reversible on stabilization. Our studies conclude that it is not neural compression or deformation that is the cause of symptoms, but it is instability-related micro-injury to neural structures that caused neurological symptoms and deficits.^[1,2] Our editorial on the subject states that the term lumbar canal “stenosis” is a misnomer and can lead to erroneous surgical treatment and should be changed to lumbar spinal “instability.”^[3] This concept has major clinical implications in that we propose that instability is the issue in the clinical entity of lumbar canal stenosis and stabilization is the treatment.^[2] Decompressive laminectomy that currently forms the primary and gold standard treatment has probably negative clinical implications in the presence of unstable spinal segments.

Of all the animals on the planet, human beings are unique in that they stand on two legs. The entire human musculature is geared to maintain activities that facilitate sitting, standing, walking, and running. All the major muscles are in the back of the spine or in the extensor compartment. Relatively, thin bands of muscles are in the anterior compartment or in the vicinity of the vertebral body and disc. All the major muscles in the extensor compartment of the spine have the fulcrum of their movements at the facet articulation. In our earlier articles, we observed that the intervertebral disc is like opera conductor who regulates the entire orchestra without having any musical instrument in his hands.^[4,5] Like the air in car tires, the disc is the seat of strength, the shape of the spine, and the center for movements. Whilst the car cannot run for even few meters without the air in the tires, it needs power in the form of petrol and motor that works as a pump. Whilst the disc is like a car tire and forms the brain of movements, muscles function like a pump and are the brawn or power of the movements.

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Muscles of the human back are large and strong. The facets in the lumbar spine are more vertically aligned when compared to the facets of the atlantoaxial joint that are more horizontal and cervical and dorsal facets that are more obliquely aligned.^[6,7] Weakness of the muscles leads to vertical facet instability or vertical facet listhesis.^[8] Such instability is initially manifested by reduction in the joint space, osteophyte formation around the articulation, reduction in the intervertebral disc space, buckling of the intervertebral ligaments, and osteophyte formation around the site of their attachment that includes anterior and posterior longitudinal ligament and ligamentum flavum. Our articles on the subject stress on the fact that all these secondary manifestations of primary spinal instability are naturally “protective” or “adaptive” in their function, indicate the presence of instability and are manifestly or potentially reversible after spinal segmental stabilization. The net effect of listhesis is the reduction in spinal and root canal dimensions. As the instability of the facets is lateral to the midline, vertical in nature, and away from neural structures, it is difficult to identify on static or dynamic imaging. However, the secondary manifestations are starkly evident on imaging, more related to their midline location and their neural compressive nature.

Vertical instability is a dynamic phenomenon and sets in or aggravates on walking for a distance when the weak muscles are unable to sustain further activity or movements and claudication pain and related symptoms set in.^[2,3] There are usually no symptoms when the muscles are appropriately rested and can function normally. Progressive weakness of the muscle reduces the claudication distance and in the more advanced stage of muscle weakness, the pain can even be at rest.^[9-11]

Long-standing symptoms are a hallmark of lumbar canal stenosis related to progressive muscle weakness in the elderly. Trauma at the beginning of initiation of symptoms is only a rare event. In younger patients, the symptoms are relatively acute, and trauma is a more frequent initiator of symptoms.^[12] Weakness of the muscles is more generalized, and consequently, instability of the lumbar spinal segments is generally multisegmental.^[13,14] In younger patients, the number of unstable spinal segments is usually less than in older patients.

It is crucial to identify the spinal segments that are unstable and that need stabilization. Apart from clinical and radiological parameters, direct operative manipulation of bones can help in the identification of unstable spinal segments. Observation of the open articular cavity, osteophytes in

the vicinity of facets, and excessive or abnormal mobility of the facets on manipulation are the indicators that suggest the level of spinal fixation that was indicated and performed. The presence of osteophytes, reduction in the disc space, osteophyte formation, and buckling of ligaments are indicators of spinal instability. Instability of the spinal segment can be present even in the absence of any indication of neural compression on radiological imaging. In general, multisegmental spinal stabilization is necessary in the treatment. L5-S1 segment should be included in the fixation construct. The rostral level of fixation usually extends up to L2-3 or even L1-2 level. We prefer direct stabilization of the facets by Camille’s technique of transarticular fixation and find it safe, simple, and strong.^[11] Facetal bones being largely cortical provides a strong purchase environment for the screws.^[7] The technique involves the implantation of screws in the strongest component of the spinal segment and at the point of the fulcrum of spinal movements. The technique provides a “zero” movement environment that is most conducive to bone arthrodesis. Double insurance technique that involves the implantation of 2 screws or triple insurance technique that involves the implantation of 3 screws at each facetal articulation adds strength and stability to the fixation construct.^[7]

CONCLUSIONS

Instability related to chronic muscle weakness forms the nodal point of genesis of lumbar canal stenosis. Compression seen on imaging is secondary to instability, is a naturally protective phenomenon, and is reversible following spinal stabilization. Identifying the unstable spinal segments and their stabilization forms the cornerstone of surgical treatment.

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