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EDITORIAL

New Treatment for Lumbar Canal Stenosis: 'Only Fixation-no Decompression'

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Lumbar canal stenosis is a common clinical diagnosis, more particularly in the 'elderly'. Approximately 20% of population above the age of 60 years develop symptoms related to lumbar canal stenosis.[1]Surgical treatment for lumbar canal stenosis is the most frequently performed spinal operation throughout the world. Chronic or long standing backache and muscle spasm that worsens on walking or exercising is the most frequent presenting symptom. 'Claudication' pain or pain that starts on walking for a distance and is relieved on resting is diagnostic of lumbar canal stenosis. The distance at which claudication pain starts is called 'claudication distance'. Claudication pain is frequently associated with radiating pain to both legs, spasms of the muscles, tingling paresthesiae and numbness of the legs. The symptoms are constant, for long duration of time, progressive and ultimately disabling. Claudication distance progressively reduces, and the pain and leg symptoms can be constant and even at rest. The clinical story is more of symptoms than of neurological deficits. Weakness of the legs and symptoms at rest are indicators of severe canal stenosis and is rarely seen in cases where there has been neglect of clinical symptoms. Multi-segmental lumbar spinal dural canal compression, bulging discs from anterior and thickened ligaments from posterior are frequent radiological observations. Reduction in the disc space, intervertebral osteophyte formation, new bone formation and bulging ligaments lead to reduction in the spinal canal and neural foraminal dimension.

Myelography, CT scan and MRI show multiple spinal level 'hour-glass' appearance of the spinal canal demonstrating 'compression' of the dural tube and neural foramina. As the radiological evidence of neural compression and spinal canal 'stenosis' are stark, the clinical entity has long been referred to as lumbar canal stenosis. As stenosis of the canal and 'compression' of the neural structures has been the basis of understanding of the entity of lumbar canal 'stenosis', decompression of the spinal and neural canal and decompression of the neural structures has been the treatment for several decades. Several variations in the techniques have been described, but essentially the aim of surgical treatment that is focussed on 'wide' decompression of the neural structures has remained constant over the years.[1] Stabilization of the spinal segments has never

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been advocated, as instability has not been considered a primary issue in the pathogenesis. Some surgeons have recently identified the possibility of delayed iatrogenic instability following wide spinal decompression that involves resection of the laminae, ligaments and part of the facets for decompression and have advocated the need for spinal stabilization. In general, gratifying clinical results have been demonstrated following surgery that involves decompression.

In 2010, we identified for the first time in the literature the entity of 'vertical' spinal instability.[2] Standing on two legs, a position unique to humans, lays lifelong stress on the extensor muscles located on the posterior aspect of the spine. Majority of muscle bulk of the spine is in the extensor compartment and their activity is focussed on the facets that forms the fulcrum of all movements. Only thin muscle groups are in relationship to the vertebral bodies and the intervertebral discs. Our articles identify disc spaces to 'air' in the tyres and muscles as pump and motor of the running car.[3-5] We also observed that discs (and odontoid process) are the brain of the spinal movements and muscle pump is the heart of its activity. Our articles identify intervertebral discs as 'divine' a structure that can 'never go wrong'. [3-5]

Weakness of the muscles of the spine related to their disuse, abuse or injury leads to subtle listhesis of the facet of the rostral vertebra over the facets of caudal vertebra.[2] Such listhesis is a chronic process and progressively worsens with the increasing weakness of the muscles. Facetallisthesis or vertical spinal instability that is the nodal point of genesis of the entire process of spinal degeneration is difficult or impossible to identify on dynamic imaging at least in the initial stages and has therefore been entirely ignored in the understanding of the pathogenesis of spinal degeneration. In our articles on the subject, we have observed that vertical spinal instability leads to a cascade of events that eventually lead to spinal canal stenosis. Chronic pain and muscle spasm that is naturally aimed at reduction in spinal movements and protection of the neural structures in the face of an unstable spinal segment is the basis of secondary spinal alterations that include bulging of the discs, buckling of the ligaments, reduction in the intervertebral disc dimensions and osteophyte formation in the intervertebral spaces that include vertebral bodies and facets. The eventual outcome of the secondary spinal alterations is reduction in the spinal and neural canal dimensions.[6] Essentially, it is the muscle weakness that leads to instability that is initially focussed on the facets that is the primary clinical event and all the so-called pathological processes are secondary natural alterations that are protective or adaptive in their function. We identified that claudication pain is a result of weak muscles giving way after a period of activity and initiate the process of canal compromise.

Our clinical studies identify the validity of stabilizing the unstable spine as the primary focus of surgical treatment of lumbar canal stenosis.[1,7,8] Decompression of the spinal canal by resection of bones/soft tissues/ osteophytes/ligaments and discs in the presence of an unstable spine can only be a negative surgical strategy that can exacerbate the instability and can have long standing negative clinical consequence.

Identification of the unstable spinal segments that will need stabilization is the key issue in the surgical treatment of spinal degeneration in general and lumbar spinal canal stenosis in particular. The number of spinal segments involved in 'older' patients is usually more than in younger patients wherein the weakness of the muscles is not generalised but is focal and more often due to injury or stretch related to focussed hyperactivity.[8] The levels of secondary alterations in the spinal canal on imaging, duration and intensity of clinical symptoms also guide the surgeon regarding the number of spinal segments that will need stabilization.

Earlier in our experience, we resorted to insertion of intra-articular spacers or facet distractors that aimed at secondary neural decompression, reduction in the facetallisthesis and stabilization and eventually led to arthrodesis of the involved spinal segment.[9] As we graduated in our experience, we have realized that both direct decompression by bone/soft tissue resection and indirect decompression of the spinal neural structures by intra-articular spacers is unnecessary and only spinal fixation is needed for these cases.[1,10] Accordingly, we now resort to only fixation of the spinal segments using Camille's transarticular fixation technique.[11] We use 2 and

sometimes 3 screws at each facetal articulation to provide double or triple insurance fixation and find this technique remarkably stable, safe and effective.[1,10,11]

It does appear that the use of nomenclature of 'stenosis' is a misnomer and leads to erroneous form of surgical treatment. We suggest that the clinical entity should be labelled as spinal instability rather that spinal stenosis.[12] All the secondary alterations like osteophyte formation, ligamentumflavum buckling and disc space reduction and bulging are secondary alterations that have a protective or adaptive function are indicators of unstable segments and are reversible following segmental spinal stabilization.

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