Lumbar canal stenosis: can “only fixation” really make decompression futile?

TO THE EDITOR: All the articles in the May 2019 issue of Neurosurgical Focus, focusing on a challenging global problem of lumbar spinal stenosis, were very intriguing. Of those, one article by Goel et al. caught my interest (Goel A, Ranjan S, Shah A, et al: Lumbar canal stenosis: analyzing the role of stabilization and the futility of decompression as treatment. Neurosurg Focus 46(5):E7, 2019). Goel has been airing his philosophy for a long time, which we welcomed previously with some reservations. 1,2

A universally accepted surgical option for lumbar canal stenosis has yet to be decided. Posterior decompression in different forms is still the gold standard. We find it commendable—we have been practicing it for a long time.

Of the factors playing a role in unison in the development of spinal canal stenosis, thickening of the ligamentum flavum (LF) is one of the most critical ones, where buckling of the LF plays a more pivotal role than hypertrophy. 3 The other crucial components are bulging of degenerated disc, buckling of posterior longitudinal ligament (PLL), hypertrophy, and medial encroachment of the facet joints and osteophytes.

Targeting the remedy for lumbar spinal stenosis with only arthrodesis does not seem to be practical, because in this process none of the compressive elements are addressed directly. Goel’s hypothesis that the origin of all pathologies leading to canal stenosis is segmental vertical instability appears to be somewhat rational. However, the changes that take place over a long period of time cannot be solved instantaneously. The debuckling of the LF and PLL can reduce the bulk in the spinal canal, but the other changes, which are not addressed, are very unlikely to be reversed immediately. Goel et al. reported reversal of some pathological changes in their earlier publications without any time frame. 3,4 Stabilization by only transarticular screws does not seem to increase the height of disc space or neural foramina. That can only be possible if the facets are distracted and some spacer is given in the articular spaces, which the authors described earlier. But that was not done in this series. 3,4 Hence, their claim of immediate relief in this series is highly dubious. Additionally, fixation has the potential to jeopardize the adjacent levels, because there is more risk of microtrauma at the levels above and below the stabilized segments. 5

The biggest concern is that the “only fixation” theory of Goel still is not an evidence-based one. An intraoperative finding of instability does not withstand strongly because that can be biased by the surgeon, leading to unnecessary intervention. The vertical instability that Goel is hypothesizing may be confirmed by preoperative dynamic CT scans, which have been shown to be effective in demonstrating craniovertebral junction instability. 2 Furthermore, there is no postoperative CT or MRI sequence to show the changes in the degenerative process following fixation.

Only after more prospective evidence-based studies with a greater number of patients and longer follow-ups are published would it be prudent to promote Goel’s prodigious philosophy to revolutionize the concept of management in this field and to exhibit the futility of decompression as treatment.

Asifur Rahman, MS
Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh

References

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Correspondence
Asifur Rahman: bijoun14@yahoo.com.

Response
The basic tenet of spinal surgery is that compression and deformation of neural structures is the cause of symptoms and decompression is the treatment. We identified the fact that for a number of common spinal ailments, it is not neural compression or deformation but subtle instability related to repeated microinjuries that causes symptoms.3–5

The concept that the lumbar canal is “stenosed” and that the neural structures are compressed at multiple lumbar spinal levels and cause related symptoms has overwhelmed the psyche of all spinal surgeons for decades. Decompression of the compressed neural structures by laminectomy is the gold standard treatment. The spine is generally considered to be stable in this clinical condition and spinal stabilization has only rarely been recommended. I am not surprised by the response of Dr. Rahman to our entirely divergent view on the subject wherein we recommend that only fixation of spinal segments is necessary and decompression is futile and probably harmful.

Our several articles have stressed that lifelong standing human posture and muscle weakness related to their misuse, disuse, or injury leads to vertical spinal instability and telescoping of the facets, wherein there is listhesis of facets of rostral vertebra over the facets of caudal vertebra.2–6 Instability of the spine is the nodal point of pathogenesis that initiates a cascade of events that are grouped under the term spinal degeneration. Buckling of the intervertebral ligaments that includes LF and PLL, osteophyte formation, and disc space reduction that result in spinal and neural canal stenosis are all secondary events to primary spinal instability.6

Some authors have argued that the symptoms could be an outcome of both static compressive factors and dynamic mechanical factors. In our article we have discussed the fact that claudication pain occurs only after walking for a distance. It means that the spinal canal “stenosis” as seen on static imaging is not the cause of symptoms. We speculated that after walking for a distance the weak muscles give way and there is instability-related listhesis of facets that initiates and generates the symptoms.

Identification of the unstable spinal segments on the basis of clinical and radiological guides and by direct inspection of the unstable facet articulation by manual manipulation of bones and by evidence like osteophyte formation around the facet articulation, excessive facet movements, and open articular cavity is the key to successful stabilization. We frequently identified that levels where there were no osteophytes, LF hypertrophy, or any other evidence of dural compression were remarkably unstable on direct inspection and needed stabilization. Our earlier articles recommended facet distraction and secondary decompression.7,8 However, our current view is that only stabilization is the answer and no primary or secondary decompression is necessary.4,5 Obviously, stabilization will not result in reversal of secondary degenerative processes instantly, but these can be expected to regress over a period of time.

After our experience with this philosophy for more than a decade, it is clear to us that the time has now come for all those in spine surgery to review the subject in a different light. The remarkably gratifying immediate postoperative and long-term clinical outcome following such a surgical strategy will certainly put the concept of decompression for multilevel spinal degeneration at both the lumbar and cervical spine in the history books.3

Atul Goel, MCh
K.E.M. Hospital and Seth G.S. Medical College, Parel, Mumbai, India
Lilavati Hospital and Research Centre, Bandra, Mumbai, India

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