Editorial

Can spinal instability by itself result in neurological symptoms and neurological deficits? An analysis

Though how nature works is way beyond man's ability to comprehend, I have found that observing how nature works offers innumerable lessons that can help us understand the realities that affect us.

–Ray Dalio

Over the years, we have observed that "only" spinal fixation, even without any direct or indirect neural "decompression" could result in relief from neurological symptoms and in the reversal of neurological deficits in a range of clinical conditions. Our several related articles present such observations.[1-16] Patients presenting with gross or manifest spinal instability or spinal segmental malalignment that can be identified on static or dynamic radiological imaging are excluded from the analysis. Chiari formation, "idiopathic" syringomyelia, basilar invagination, myelopathy related to degenerative spinal disease, and ossification of the posterior longitudinal ligament, Hirayama disease, and several other so-called "pathological" clinical conditions are included in this cohort.[1-18] Only spinal stabilization in cases with spinal trauma without radiological evidence of neural compression or deformation can result in neurological recovery.[19] Major neurological symptoms in the absence of direct proportional neural compression and recovery following spinal stabilization without any kind of decompression raise several questions. Is instability by itself a cause of neurological symptoms and deficits?[20] Is neural deformation and compression not the cause of neurological symptoms and/or deficits?[21] Are the neural tissues easily deformable and stretchable like rubber or rubber band? Can twisting or buckling of the cord during abnormal spine movements be the cause of symptoms? Is transient compromise of blood supply to the cord possible during abnormal spinal movements? Do the facets have a role in initiating and propelling symptoms? Does microinstability cause repeated trauma to the cord and result in symptoms? Is "vertical instability" that results in buckling of the spinal cord the cause of symptoms?[22] Are neurological symptoms akin to symptoms related to inflammation wherein pain, fever, swelling, and reduction in body movements are all secondary to a primary infection or injury? Is the process similar to the pathogenesis of hydrocephalus or edema that is secondary to a primary brain tumor or infection?[23]

Standing human position makes humans unique. However, it entails lifelong stress on the extensor muscles located on the back of the spine. In 2010, we proposed that acute or chronic weakness of these muscles secondary to disuse, abuse, or injury leads to telescoping of the spinal segments related to subtle or manifest listhesis at the facetal articulation.[1,2,24,25] Instability or abnormality in movements might not be identified on dynamic imaging of the spine. We labeled such instability as "vertical" spinal instability.[22] It was observed that vertical instability forms the nodal point of genesis of spinal "spondylotic" alterations. In the craniovertebral junction, such instability that is initially manifested at the facetal articulation was labeled as central or axial atlantoaxial instability (CAAD).[17,26-28]

The majority of muscles of the spine are located in the extensor compartment or back of the spine and focus their activity on the facets. Facetal articulation forms the fulcrum point of the muscle pulley. Flexion of the spine and body is essentially a passive activity, and relatively, only a few strands of muscles are located in the vicinity of vertebral body and the disc. The intervertebral disc forms the basis or "backbone" of movement of the spine or is the brain of all movements. We earlier likened the disc to opera conductor who regulates all music without holding any instrument in hand. Our articles

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observe remarkable similarities between intervertebral disc and the odontoid process.[29,30]

Muscle disuse, abuse, or injury can lead to abnormal spinal alignments and instability that begins at the facets. Reduction in the facetal articulation space and retrolisthesis of the facets is the initial manifestations of muscle weakness. Reduction in the joint space, buckling of intervertebral ligaments that includes ligamentum flavum and posterior longitudinal ligament, bulging of the disc into the spinal canal, osteophyte formation around the bony edges of vertebral bodies and facets, and eventual reduction in the spinal and neural canal dimensions are consequences or manifestations of muscle weakness and segmental spinal instability.[6,24,25] We related cord atrophy and signal changes as seen on magnetic resonance imaging to the vertical reduction in the length of the spinal cord. Such a complex of secondary manifestations is grouped under the generally agreed term of spinal spondylosis or spinal/neural canal stenosis. The listhesis at the facets can be subtle and instability can be difficult or impossible to identify on modern dynamic imaging. Essentially, instability at the facets is the primary issue and the spondylotic "alterations" are secondary, naturally protective, or adaptive and are potentially reversible following spinal stabilization. Our articles observe that stabilization of the spinal segments is the treatment and any kind of bone, soft tissue, or disc resection for "decompression" is unnecessary and probably a counter-effective surgical exercise. Clinical recovery following only fixation and without any kind of alteration of neural "compressive" or deforming structures identifies the role of instability and questions the significance of compression in the generation or propulsion of neurological symptoms. The extent of compression by one or all the secondary spinal alterations can quantify the extent of segmental spinal instability.

We speculated that acute disc herniation is a result of sudden onset instability following lifting heavy weight or following an acute blunt or sharp injury to the spine or chronic instability by itself causes disc herniation.[31] It was observed that pain related to disc herniation is a natural protective or adaptive response to unstable spine and functions to avoid excessive movements that can permanently injure the neural structures. Even symptoms such as foot drop and leg weakness and urinary incontinence appear to be natural phenomenon that attempts to reduce body movements that could adversely affect neural structures in an unstable spine. Stabilization of the spinal segment relieves the symptom of pain instantly and initiates a process of recovery in neural dysfunction even when the neural compression and deformation by the herniated disc are not directly addressed. As soon as the spinal segment is stabilized, the process of natural resorption of the herniated disc is initiated. The role of neural compression and deformation in initiating the pain and related symptoms needs to be evaluated.

Relief from symptoms following spinal stabilization in cases with myelopathy related to ossification of the posterior longitudinal ligament is dramatic and is observed in the immediate postoperative period. The fact that fixation alone and without any attempt to "decompress" the neural structures produces relief from symptoms questions the role of neural compression.[10-13]

It was observed that in cases with spinal trauma that, stabilization by itself and without handling of neural structures could result in improvement of clinical symptoms and recovery from neurological deficits. We observed such clinical improvement not only in "older" patient group, wherein some "degenerative" spinal alterations are present but also in younger patients where the spinal canal was not intruded by any ligament, osteophyte or fractured bone, or disc segment.[19]

CAAD was identified to be the nodal point of the pathogenesis of a range of clinical conditions.[17] Conditions such as Chiari formation, syringomyelia and external syringomyelia, basilar invagination, C2–3 fusion, assimilation of atlas, platybasia, Klippel–Feil abnormality, bifid C1 and C2, os odontoideum, short neck, torticollis, short spine, dorsal kyphoscoliosis, and a range of other clinical conditions can have their basis of origin in atlantoaxial instability. These conditions appear to be neural compressive and have conventionally been labeled as "pathological." We speculated that all these conditions are secondary to segmental spinal instability, are naturally protective or adaptive, and are manifestly or potentially reversible following atlantoaxial stabilization. Neurological deficits can be present even in the absence of direct radiological evidence of neural compression. Remarkable recovery in the symptoms and deficits following atlantoaxial stabilization without any attempt toward decompression of neural structures is suggestive of the role of instability in the generation of symptoms. CAAD by itself without any other associated spinal bone or soft-tissue alteration can result in significant neurological symptoms. An element of rotatory atlantoaxial instability can be a generator of a host of the brain- and spine-related neurological symptoms and deficits. Clinical recovery following stabilization points toward the role of instability in the generation of neurological symptoms.

"Only-spinal fixation" in cases with Hirayama disease was seen to result in recovery from clinical symptoms.[18]

Facet injections with steroids and with similar drugs have been identified to result in relief from neurological symptoms. The role of steroids in initiating spinal stabilization by fibrosis or other similar mechanism/s can only be speculated.

The spinal cord and other neural structures have remarkable resilience, elasticity, and plasticity and ability to accommodate and thrive, particularly when the compression is chronic or longstanding. Tolerance of neural structures in general and spinal cord in particular in a variety of clinical conditions is well-known. Patients with large benign spinal tumors and cysts and with huge syringomyelia presenting with only minimal or marginal neurological deficits or symptoms is a common observation.[32]

Like symptoms related to inflammation can be the primary manifestation of infection, they are essentially protective in nature. Similarly, brain edema and hydrocephalus can be the cause of symptoms when they are essentially secondary and protective in their function. Vasospasm following aneurysmal bleed is a protective natural act in the presence of rupture of an aneurysm.[33] While one can relieve the person of symptoms related to inflammation or that due to brain edema and hydrocephalus by drugs or surgery, it is essential to treat the primary pathogenetic factor. Moreover, the treatment of secondary manifestation rather than the primary issue can have a negative clinical implication or connotation.

Our studies suggest that chronic or long-standing instability initiates multiple natural processes that are adaptive and aimed at protection of neural structures from potential injury that could threaten the existence of life. Even processes of self-bone and self-neural destruction such as syringomyelia, os odontoideum, bifid arches of atlas, and bone fusions and new bone formation have a protective role and are potentially reversible following stabilization of the affected spinal segment.[34-37] Musculoskeletal abnormalities such as short neck, torticollis, and dorsal kyphoscoliosis do not always signify abnormality or pathology but could be an outcome of divine intervention.

It is clear that stabilization results in clinical recovery. In our entire series, we have resorted to facetal fixation or stabilization. It is unclear as to how facetal fixation alone can cause recovery from neurological symptoms, when the entire surgical procedure is done away from the neural structures. It is difficult to speculate as to how facetal instability can initiate a general body response. It is equally difficult to speculate as to how only fixation results in "dramatic" clinical improvement.

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REFERENCES

1. Goel A, Shah A. Facetal distraction as treatment for single- and multilevel cervical spondylotic radiculopathy and myelopathy: A preliminary report. J Neurosurg Spine 2011;14:689-96.
2. Goel A, Shah A, Jadhav M, Nama S. Distraction of facets with intraarticular spacers as treatment for lumbar canal stenosis: Report on a preliminary experience with 21 cases. J Neurosurg Spine 2013;19:672-7.
3. Goel A. Role of subaxial spinal and atlantoaxial instability in multisegmental cervical spondylotic myelopathy. Acta Neurochir Suppl 2019;125:71-8.
4. Goel A. 'Only fixation' as rationale treatment for spinal canal stenosis. J Craniovertebr Junction Spine 2011;2:55-6.
5. Goel A. Only fixation for cervical spondylosis: Report of early results with a preliminary experience with 6 cases. J Craniovertebr Junction Spine 2013;4:64-8.
6. Goel A, Dandpat S, Shah A, Rai S, Vutha R. Muscle weakness-related spinal instability is the cause of cervical spinal degeneration and spinal stabilization is the treatment: An experience with 215 cases surgically treated over 7 years. World Neurosurg 2020;140:614-21.
7. Goel A, Dharurkar P, Shah A, Gore S, More S, Ranjan S. Only spinal fixation as treatment of prolapsed cervical intervertebral disc in patients presenting with myelopathy. J Craniovertebr Junction Spine 2017;8:305-10.
8. Goel A, Dharurkar P, Shah A, Gore S, Bakale N, Vaja T. Facetal fixation arthrodesis as treatment of cervical radiculopathy. World Neurosurg 2019;121:e875-81.
9. Goel A, Vutha R, Shah A, Patil A, Dhar A, Prasad A. Cervical spondylosis in patients presenting with "severe" myelopathy: Analysis of treatment by multisegmental spinal fixation – A case series. J Craniovertebr Junction Spine 2019;10:144-51.
10. Goel A, Nadkarni T, Shah A, Rai S, Rangarajan V, Kulkarni A. Is only stabilization the ideal treatment for ossified posterior longitudinal ligament? Report of early results with a preliminary experience in 14 patients. World Neurosurg 2015;84:813-9.
11. Goel A. Is atlantoaxial instability the cause of "high" cervical ossified posterior longitudinal ligament? Analysis on the basis of surgical treatment of seven patients. J Craniovertebr Junction Spine 2016;7:20-5.
12. Goel A. Ossification of the posterior longitudinal ligament: Analysis of the role of craniovertebral and spinal instability. Acta Neurochir Suppl 2019;125:63-70.
13. Goel A, Grasso G, Shah A, Rai S, Dandpat S, Vaja T, *et al*. "Only spinal fixation" as surgical treatment of cervical myelopathy related to ossified posterior longitudinal ligament: Review of 52 cases. World Neurosurg 2020;140:556-63.
14. Goel A, Biswas C, Shah A, Rai S, Dandpat S, Bhambere S. Report of an eight-year experience with Camille's transarticular technique of cervical spinal stabilization. J Clin Neurosci 2022;95:9-19.
15. Goel A, Vaja T, Shah A, Rai S, Dandpat S, Vutha R, *et al*. Outcome of osteophytes after only-fixation as treatment for multilevel cervical

spondylosis-a minimum of 12 months follow-up. World Neurosurg 2021;146:e876-87.
16. Goel A, Ranjan S, Shah A, Patil A, Vutha R. Lumbar canal stenosis: Analyzing the role of stabilization and the futility of decompression as treatment. Neurosurg Focus 2019;46:E7.
17. Shah A, Vutha R, Prasad A, Goel A. Central or axial atlantoaxial dislocation and craniovertebral junction alterations: A review of 393 patients treated over 12 years. Neurosurg Focus 2023;54:E13.
18. Goel A, Dhar A, Shah A. Multilevel spinal stabilization as a treatment for hirayama disease: Report of an experience with five cases. World Neurosurg 2017;99:186-91.
19. Goel A, Lunawat A, Shah A, Dandpat S, Hawaldar A, Darji H, *et al*. Clinical outcome following multisegmental cervical spinal fixation in patients who recovered partially following injury. J Craniovertebr Junction Spine 2021;12:216-22.
20. Goel A. Is instability the nodal point of pathogenesis for both cervical spondylotic myelopathy and ossified posterior longitudinal ligament? Neurol India 2016;64:837-8.
21. Goel A. Not neural deformation or compression but instability is the cause of symptoms in degenerative spinal disease. J Craniovertebr Junction Spine 2014;5:141-2.
22. Goel A. Vertical facetal instability: Is it the point of genesis of spinal spondylotic disease? J Craniovertebr Junction Spine 2015;6:47-8.
23. Goel A. Tumour induced hydrocephalus and oedema: Pathology or natural defence. J Postgrad Med 2002;48:153-4.
24. Goel A. Facet distraction spacers for treatment of degenerative disease of the spine: Rationale and an alternative hypothesis of spinal degeneration. J Craniovertebr Junction Spine 2010;1:65-6.
25. Goel A. Facet distraction-arthrodesis technique: Can it revolutionize spinal stabilization methods? J Craniovertebr Junction Spine 2011;2:1-2.
26. Goel A. Goel's classification of atlantoaxial "facetal" dislocation. J Craniovertebr Junction Spine 2014;5:3-8.
27. Goel A. Central or axial atlantoaxial instability: Expanding understanding of craniovertebral junction. J Craniovertebr Junction Spine 2016;7:1-3.
28. Goel A. A review of a new clinical entity of 'central atlantoaxial instability': Expanding horizons of craniovertebral junction surgery. Neurospine 2019;16:186-94.
29. Goel A. Odontoid process and intervertebral disc: Do they have the same function? J Craniovertebr Junction Spine 2020;11:59-60.
30. Kothari M, Goel A. The so-called intervertebral disc: A 4-D reverie. Neurol India 2007;55:97-8.
31. Goel A. Is disc herniation "secondary" to spinal instability? Is it a protective natural response? J Craniovertebr Junction Spine 2021;12:213-5.
32. Goel A, Shah AH, Vutha R, Goel A. External syringomyelia in longstanding benign foramen magnum tumors. Surg Neurol Int 2020;11:92.
33. Goel A. Nature's wisdom during aneurysmal subarachnoid hemorrhage. Neurol India 2003;51:165-6.
34. Goel A. Is syringomyelia pathology or a natural protective phenomenon? J Postgrad Med 2001;47:87-8.
35. Goel A, Patil A, Shah A, Dandpat S, Rai S, Ranjan S. Os odontoideum: Analysis of 190 surgically treated cases. World Neurosurg 2020;134:e512-23.
36. Goel A, Nadkarni T, Shah A, Ramdasi R, Patni N. Bifid anterior and posterior arches of atlas: Surgical implication and analysis of 70 cases. Neurosurgery 2015;77:296-305.
37. Goel A. Is evidence of bone "formation" and "fusion" in the spinal segment an evidence of segmental spinal instability? J Craniovertebr Junction Spine 2022;13:365-7.