

Editorial

Muscle Weakness as the Genesis of Spinal Disease: Paradigm Shift in Understanding Spinal Degeneration

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The understanding of spinal disorders has undergone remarkable evolution over the past century. Traditionally, cervical and lumbar spondylosis, ossification of the posterior longitudinal ligament (OPLL), Hirschmann disease, degenerative spinal deformity, and several other spinal disorders have been viewed as distinct pathological entities, each characterized by unique radiological abnormalities and treated primarily by decompressive surgery. Disc degeneration, osteophyte formation, ligamentous hypertrophy, ligament ossification, and canal stenosis have conventionally been regarded as the primary pathological processes responsible for neurological dysfunction.

Over the last four decades, however, Goel has advanced a fundamentally different 'instability concept' that challenges this long-established paradigm. Through an extensive series of clinical observations, surgical innovations, and conceptual publications, it has been proposed that chronic weakness of the spinal extensor musculature is the initiating event in the pathogenesis of most degenerative and several non-degenerative spinal disorders.¹⁻³ According to this hypothesis, instability – not degeneration – is the primary disease, and the structural abnormalities identified on imaging represent secondary protective adaptations.

This concept represents one of the most comprehensive attempts to redefine spinal pathology from a biomechanical perspective.

The normal spine is maintained not merely by its bones, discs, and ligaments but primarily

by the continuous activity of powerful paraspinal muscles. These muscles function as the principal dynamic stabilizers throughout life and partake in affecting the unique human standing on two legs posture. Goel proposed that aging, repetitive microtrauma, altered posture, sedentary lifestyle, chronic overuse, and physiological muscle fatigue gradually diminish muscular competence.¹⁻³ As muscular support weakens, the burden of maintaining spinal stability shifts to passive structures, resulting in subtle but progressive instability.

The earliest manifestation of this instability occurs at the facet joints, the true articulations of the spine. Vertical facet instability or "telescoping" develops before disc collapse becomes radiologically evident.⁴ Progressive overriding of the facets leads to abnormal spinal mechanics and initiates a cascade of clinical manifestations and an adaptive structural response.

Within this framework, disc space reduction is no longer viewed as the initiating event. Instead, it is considered a secondary consequence of altered spinal loading. Ligamentum flavum buckling, posterior longitudinal ligament infolding, osteophyte formation, disc bulging, facet hypertrophy, and eventual canal stenosis are interpreted as biological attempts to stabilize an unstable spinal segment.¹ These changes increase spinal rigidity but eventually compromise neural structures, producing pain, myelopathy, or radiculopathy.

This reversal of the traditional sequence – from degeneration causing instability to



instability causing degeneration—represents an important conceptual contribution to spinal surgery.

An equally important aspect of ‘instability concept’ has been the development of surgical strategies derived directly from this pathogenetic understanding. If instability is the primary pathology, stabilization should constitute definitive treatment. Over the last three decades, Goel has consistently demonstrated that fixation alone, without decompression in selected patients, can produce remarkable neurological recovery in cervical and lumbar degenerative disease.^{1,2,5} Restoration of spinal stability permits reversal of ligamentous buckling, remodelling of soft tissues, and gradual resolution of neural compression without the need for removal of bone, ligament, osteophytes or disc.⁶

Few concepts have generated as much discussion as the interpretation of ossification of the posterior longitudinal ligament (OPLL). Traditionally regarded as a primary disorder of abnormal ossification with genetic and metabolic determinants, OPLL has generally been treated by direct decompression. Goel proposed that ossification represents an exaggerated stabilizing response to longstanding instability.⁷ In this model, ectopic bone formation is not the disease itself but an adaptive attempt by the body to reinforce mechanically compromised spinal segments. This hypothesis provides a biomechanical explanation for the characteristic distribution of OPLL and offers a rationale for treating selected patients with stabilization alone.

Goel has similarly extended the instability hypothesis to Hirayama disease, a disorder traditionally attributed to disproportionate growth of the vertebral column and dural sac during adolescence.⁸ While dynamic flexion MRI demonstrates forward displacement of the posterior dura, it is suggested that cervical instability resulting from muscular insufficiency may precede these radiological findings. Dynamic instability permits repetitive injury to the lower cervical spinal cord during neck flexion, whereas stabilization interrupts this pathological process. This interpretation transforms Hirayama disease from a purely developmental abnormality into a biomechanical disorder.

Perhaps the greatest strength of ‘instability concept’ lies in its ability to unify numerous

apparently unrelated clinical conditions under a common mechanistic framework. Over the years, it has been proposed that instability is the principal pathogenetic mechanism not only in cervical and lumbar spondylosis but also in basilar invagination, Chiari formation, syringomyelia, retro-odontoid pseudotumor, degenerative cervical kyphosis, spinal deformities, Klippel-Feil alterations, and several congenital craniovertebral junction anomalies.^{9,10} Although these disorders differ markedly in their radiological appearance, Goel has argued that each represents a distinct adaptive response to chronic mechanical instability generated by failure of muscular support.

Central to this philosophy is the recognition that radiological abnormalities should not automatically be interpreted as the primary disease. Osteophytes, hypertrophied ligaments, disc protrusions, and ligament ossification may instead represent the body’s attempts to increase stability. Their removal does not necessarily address the underlying pathology. Stabilization, rather than decompression alone, therefore becomes the logical therapeutic objective.

Importantly, this hypothesis has also redirected attention toward prevention. If chronic muscle weakness initiates instability, preserving paraspinal muscle function assumes critical importance. Strategies aimed at maintaining muscle strength, endurance, posture, and neuromuscular coordination may delay or prevent many structural changes currently accepted as inevitable consequences of aging.

Like every major paradigm shift, Goel’s concepts remain the subject of vigorous scientific discussion. Many investigators continue to emphasize the importance of genetics, aging, inflammation, metabolic factors, and developmental abnormalities in disorders such as OPLL and Hirayama disease. These mechanisms are not necessarily incompatible with ‘instability concept’; rather, they may influence susceptibility while mechanical instability serves as the final common pathway leading to structural adaptation.

History repeatedly demonstrates that transformative ideas often begin by questioning accepted assumptions. These contributions have challenged the long-

held belief that degeneration is the primary disease of the spine. The proposal that muscle weakness initiates instability, instability initiates degeneration, and stabilization addresses the root pathology, can be one of the most influential biomechanical concepts in contemporary spinal surgery. Whether every aspect of this hypothesis ultimately withstands scientific scrutiny is less important than the fact that it has fundamentally changed how many surgeons think about spinal disease.

Future advances in spinal research should therefore extend beyond imaging of bones and discs to include objective assessment of paraspinal muscle function, muscle quality, dynamic spinal biomechanics, and the biological mechanisms linking chronic instability with adaptive tissue remodelling. Such investigations may ultimately determine whether Goel's muscle-centered hypothesis represents not merely an alternative explanation but a unifying principle underlying a broad spectrum of spinal disorders.

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