Editorial

Basilar invagination, spinal "degeneration," and "lumbosacral" spondylolisthesis: Instability is the cause and stabilization is the treatment

In the year 1998, we identified atlantoaxial facetal listhesis as the initial point of pathogenesis of basilar invagination.[1-3] Subaxial spinal "degeneration" was also identified to have its origin in facetal listhesis. Standing human position and "lifelong" stress on muscles facilitating this posture form the basis of facetal listhesis. Muscle abuse, disuse, and injury propel "vertical" spinal instability that manifests initially in the form of reduction in the interfacetal space and subsequently listhesis.[4]

Telescoping of the spinal segments as a result of muscle weakness forms the basis of basilar invagination in the craniovertebral junction and spinal degeneration in the subaxial spine. The atlantoaxial instability is more often chronic or long standing. Our articles refer to a number of natural protective maneuvers in the face of chronic atlantoaxial instability that lead to musculoskeletal and neural alterations.[5-14] These alterations include short neck, short head, short spine, torticollis, dorsal kyphoscoliosis, Klippel-Feil alteration, C2–3 fusion, assimilation of atlas, bifid arches of atlas and axis, platybasia, Chiari formation, and syringomyelia.[5-14] Despite the fact that all the mentioned secondary manifestations appear to be compressing or deforming the neural structures, our analysis suggests that they are all secondary to vertical spinal instability, are protective natural events, and are reversible following atlantoaxial stabilization.[15] Essentially, the clinical issue in basilar invagination is spinal instability and stabilization is the treatment.[16] It is not neural deformation or compression that is the cause of symptoms, but it is muscle weakness-related spinal instability that initiates, propels, and establishes secondary manifestations and results in clinical symptoms.[17-19] Restoration or realignment of the musculoskeletal and neural structures can eventually happen, either in the early or delayed postoperative period. It has to be clearly understood that the aim of surgery is spinal segmental stabilization and not any direct surgical action against any of the secondary manifestations.[16] Secondary manifestations and neural compression that might persist after stabilization need only observation and no direct surgical action. Even when the realignment is incomplete and there is persistent radiological evidence of "compression," there may not be any need for "decompression" of the neural structures. Resection of bone in an unstable spinal segment for "decompression" may actually be harmful in the long run as the bone available for arthrodesis is consequently reduced.

In degenerative spinal disease, all the known so-called "pathological" entities such as osteophyte formation, ligamentum flavum buckling, disc space reduction, and disc bulging into spinal canal and several similar issues that ultimately result in reduction in neural foramina and spinal canal are related to primary issue of vertical spinal instability.[4] Instability at the facets is related to compromise of the strength or turgor of the muscles that facilitate the movements and standing human posture. Despite the fact that all the secondary events have been incriminated to be the cause of clinical symptoms, our observations suggest that symptoms are related more to instability and its secondary effects. As in the craniovertebral junction, all the secondary manifestations have a naturally protective role and are reversible following stabilization. "Only stabilization" of the affected spinal segment/s is the treatment.

Lumbosacral (and other spinal segment) spondylolisthesis is a result of incompetence of the muscles and related spinal

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instability. Spondylolisthesis is actually a manifestation of more "severe" form of spinal segmental instability or spinal degeneration. The fracture of pars interarticularis in cases with lumbosacral listhesis simulates the fracture of pedicle of C2 in cases of Hangman's fracture. The clinical symptoms are related to spinal instability and not neural deformation or compression as is observed on radiological imaging. Like in basilar invagination, wherein there is a need to stabilize the atlantoaxial joint and any form of bone decompression can be avoided, in lumbosacral spondylolisthesis, it is essential to firmly stabilize the affected spinal segment with the aim of arthrodesis or fusion and avoid any form of bone decompression. Moreover, although preferable, it is unnecessary to directly aim at realignment. This concept suggests that any form of laminectomy, discoidectomy, or distraction–realignment for decompression and realignment can be avoided in cases of lumbosacral spondylolisthesis.

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