Instability is the cause, and stabilization is the treatment for single-or multi-level spinal degeneration. Our several articles refer to this issue and suggest that for patients presenting with cervical radiculopathy or myelopathy in cases with single-or multi-level spinal degeneration with or without ossification of posterior longitudinal ligament (OPLL) decompression by removal of bone, ligament, disc or bone component of OPLL is unnecessary and “only-stabilization” is the treatment.[1-18] Our hypothesis is based on the concept that weakness of the muscles of the back of the spine, particularly those involved in maintaining standing human posture, leads to “vertical” spinal instability that is first manifested at the facets in the form of listhesis of the inferior facet of the rostral vertebral over the superior facet of the caudal vertebra.[15] Due to the subtle nature of dislocation and lateral location of the facets that is away from neural structures, radiological identification of instability or dislocation is difficult or impossible.[19] Our studies conclude that the term “spinal canal stenosis” is a misnomer and directs to erroneous treatment and should be changed to single- or multi-level “spinal instability.”[20] Spinal instability is also a primary issue in cases with prolapsed and herniated disc. The instability is either the cause of disc herniation or disc herniation leads to spinal instability. Essentially, our hypothesis suggests that spinal instability is the primary issue and all the so-called “pathological alterations” are secondary in nature and “protective” in function.

Buckling of the interspinous ligaments that include ligamentum flavum and OPLL, osteophyte formation, reduction of the intervertebral disc space and all the other known pathological features of degenerative spondylosis are secondary consequences of vertical spinal instability. Essentially, these secondary features are radiological musculoskeletal evidence of spinal instability. The net effect is reduction in the spinal and neural canal dimensions. Radiating pain, weakness, or paresthesiae in affected dermatomes are clinical evidence of the level of spinal instability. Radiological evidence of neural compression indicate subtle, manifest, or potential spinal instability even in the absence of clinical manifestations. On the other hand, clinical features, even in the absence of radiological evidence can suggest spinal instability and indicate the need for surgical treatment. Spinal medullary cord changes without any compressive neural factor indicate vertical spinal instability and longitudinal buckling of the spinal cord. The cause of symptoms is spinal instability and not physical neural compression.[18] It is important to note that there may be spinal instability even in the absence of clinical and/or radiological evidence. Ignoring neighboring unstable spinal segments and treatment of only the spinal level having radiological evidence is an important cause of the surgical failure or delayed “adjacent segment disease.” Spinal stabilization is usually necessary in more segments than that demonstrated by radiological images and evidence of neural compression and clinical parameters.

Atlantoaxial joint is the most mobile joint of the body and is the most likely joint to develop spinal instability. Our studies suggest that atlantoaxial instability has been traditionally ignored as a likely site for spinal degeneration. Significant or severe symptoms related to cervical myelopathy are more often than not associated with atlantoaxial instability. Atlantoaxial instability is generally associated in cases with multi-level spinal degeneration that is more often seen in “old-age” patients.[12,13] Atlantoaxial instability can be present even without any neural compression in the region or any radiological evidence of spinal instability on dynamic imaging.

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Direct inspection of the facetal articulation by exposing the segments in the vicinity of those suggested by clinical and radiological guides is crucial to identify the unstable spinal levels. Excessive or abnormal movements on spinal manipulation, identification of osteophytes in the proximity of facets, and unusually open articular cavity can indicate spinal instability.

Our recent classification divides atlantoaxial instability into three types depending on the facet alignment on lateral profile imaging with the head in neutral position. Type 1 atlantoaxial instability is when the facet of atlas is dislocated anterior to the facet. Type 2 atlantoaxial instability is when the facet of atlas is dislocated posterior to the facet of axis. Type 3 atlantoaxial instability is when the facets of atlas and axis are in alignment and atlantoaxial instability is diagnosed based on corroborative evidence. As there may not be any atlantodental interval disturbance and there may not be any neural or dural compression adjacent to the odontoid process, Types 2 and 3 atlantoaxial instability are referred to as central or axial atlantoaxial instability. Such central atlantoaxial dislocation is usually associated with chronic or longstanding instability. Direct observation of the facetal articulation and excessive or abnormal movements of the joint on bone manipulation is the final or confirmatory evidence of atlantoaxial instability. Essentially it means that there can be atlantoaxial instability even in the absence of any evidence of bone abnormality or any kind of neural compression. Multi-level spinal degeneration is more often associated with central or axial atlantoaxial instability.

Direct facetal fixation is biomechanically the most stable as it fixes the point of fulcrum of spinal movements. For subaxial spine, Camille’s technique of transarticular fixation is quick, safe, strong and relatively “easy.” Two or even three transarticular screw insertion is possible to provide “double-insurance” or “triple insurance” fixation. Goel technique of atlantoaxial fixation has been identified to be biomechanically the most stable fixation technique. Recently, we described an alternative atlantoaxial fixation technique by fixing C2-3 articulation by transarticular fixation and sectioning the muscles attached to the C2 spinous process. The technique disables movements of the odontoid process and retains transverse or rotatory movements executed by the muscles attached to the transverse process of the atlas. Even though direct atlantoaxial fixation, as described in our technique is an ideal form of surgical treatment in cases with multi-level cervical spinal degeneration, the alternative facetal technique can be used as an option as it preserves rotatory neck movements.

Inclusion of the C2 spinal segment in the fixation construct with the aim to provide selective atlantoaxial stabilization is crucial and mandatory in a “large” number of cases with multi-level spinal degeneration. Such stabilization is particularly essential in cases where the myelopathy is moderately or significantly severe and in old age patients. Ignorance of association of atlantoaxial instability and ignoring the inclusion of the C2 spinal segment appears to be a major cause of failure of surgical treatment.

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