

Editorial



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Basilar Invagination: Instability Is the Cause and Stabilization Is the Treatment

Basilar invagination was for long considered to be a radiological curiosity rather than a surgically treatable clinical entity. Subsequently, for several years, including in our article dedicated to the subject in 1998, basilar invagination was associated with 'fixed' or 'irreducible' atlantoaxial joint and decompression was considered to be the treatment.¹ Depending on the direction of neural compression, anterior transoral odontoidectomy (group 1 basilar invagination) or posterior foramen magnum bone and/or dural decompression (group 2 basilar invagination) were the modalities of surgical treatment.¹

In 2004, the possibility of reducing basilar invagination by atlantoaxial facetal distraction and achieving craniovertebral junction realignment revolutionized the treatment for basilar invagination.² The concept was that basilar invagination (group A) is a result of 'vertical' spinal instability and the atlantoaxial facetal listhesis that eventually results in basilar invagination mimics lumbosacral listhesis.3 It was observed that in the entity of 'fixed' or 'irreducible' atlantoaxial dislocation, atlantoaxial joint is actually not stable or fused but is an unstable spinal segment and the joint in such cases is not only mobile but is pathologically hypermobile and can be reduced by manual distraction of the facets. Since the introduction of this concept, a number of authors have resorted to attempts at reducing atlantoaxial instability and realigning craniovertebral junction and the authors recommending 'decompression' as a primary modality of surgical treatment is gradually but definitely reducing all over the world. Our studies identify muscle weakness related to disuse or injury as a nodal point of genesis of atlantoaxial instability and subsequent development of basilar invagination. Ligamentous 'stretch' has not been identified to have a role in the pathogenesis of atlantoaxial instability or of basilar invagination and anterior and/or posterior 'release' of 'tension bands' ligaments or muscle groups by sectioning has not been identified to have a place in the surgical treatment.

Since 1988, our technique of atlantoaxial fixation involves opening of the atlantoaxial joint, denuding the articular cartilage and introduction of bone graft within the articular cavity prior to lateral mass instrumentation. All these principle surgical steps are essential in achieving strong atlantoaxial arthrodesis. In 2004, we introduced intra-articular metal spacers to facilitate and sustain facetal distraction in cases with basilar invagination, rotatory dislocation, and irreducible and even reducible atlantoaxial dislocation. As the experience has increased, it is now realized that distraction of the facets of atlas and axis and packing of bone graft in the articular cavity can lay a ground for solid bone arthrodesis and the reliance on intra-articular facetal implant can be reduced if not entirely avoided. Whilst the metal implants provides for stability, bone graft provides for fusion of the facets and also sustains stability-immobility and distraction. As the experience in surgically managing

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basilar invagination has increased, it now appears that more than 'realignment' of the craniovertebral junction, it is firm stabilization of the atlantoaxial segment that is necessary.¹⁰⁻¹²

Chronic or longstanding atlantoaxial instability is associated with a host of 'secondary' musculoskeletal and neural 'alterations.' Musculoskeletal alterations include a number of morphological changes that include platybasia, Klippel-Feil abnormality, assimilation of atlas, bifid arch of atlas and C2-3 fusion and neural alterations that include Chiari formation and syringomyelia.¹³⁻¹⁵ The term 'basilar invagination' in general is an umbrella term that includes a range of alterations. Short neck, torticollis, short spine and dorsal kyphoscoliosis are external manifestations of basilar invagination. All the secondary manifestations are natural protective maneuvers. More importantly, it was identified that all the secondary musculoskeletal and neural alterations when present in a consort or in isolation indicate the presence of atlantoaxial instability and are reversible following atlantoaxial fixation. The fact that there can be dislocation of the atlantoaxial joint even in the absence of any alteration in atlantodental interval or any dural or neural compression by the odontoid process promises a fresh understanding of the entire subject.¹⁶ Such form of dislocation was labeled as 'central' or 'axial' atlantoaxial dislocation and was diagnosed on the basis of alignment of facets on sagittal imaging with the head in neutral position and was confirmed on direct bone manipulation during the surgical procedure.¹⁷ Atlantoaxial stabilization forms the basis of treatment. Although our articles were amongst the first that suggested suitability of occipital screws, we have currently abandoned inclusion of occipital bone in the fixation construct and identify segmental atlantoaxial fixationarthrodesis as optimum surgical treatment.^{3,18}

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