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Editorial

Is inclusion of the occipital bone necessary/counter-effective for craniovertebral junction stabilization?

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The craniovertebral junction is a complex formation where bone, neural structures, and blood vessels are located in a compact junctional "bottleneck" zone. The bony structure is designed immaculately to perform the function of circumferential mobility while being the most stable of all joints in the body. More importantly it functions to provide safety and resistance free traverse to critical neural and vascular structures even while conducting all the complex movements. Standing human posture lays excessive and life-long stresses on the region.

In general, more mobile the region is, less stable it is. However, the complex of atlantoaxial and occipitoatlantal joints is most stable while being most mobile. Occipitoatlantal joints are like sacroiliac joints. Both these joints are the least mobile but are most stable. In the craniovertebral junction, complex atlantoaxial joints are the centers for mobility and occipitoatlantal joints are the centers for stability.

At the atlantoaxial region, all movements are initiated and completed at the facet joints. The odontoid process directs and participates in the conduct of the movements while the actual brunt of the activity is focused at the facets joints.[1] It may be that odontoid process is the brain while facets are the brawn of all movements. In the similar lines, in the subaxial spine, we hypothesized that discs are like an opera conductor for all the movements that are orchestrated and played at the facets.[2-4]

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While the atlantoaxial joint is the center for mobility, it is also the center of instability. Occipitoatlantal instability is a rare phenomenon that occurs in extreme trauma or in children having syndromic disorders that lead to generalized ligamental laxity. Tuberculosis, rheumatoid arthritis, degenerative arthritis, and tumors can only rarely affect the stability of the occipitoatlantal joint. It may not be an over-exaggeration to say that in general all craniovertebral junction region instability is equivalent to atlantoaxial instability. On similar lines, one can generalize that all craniovertebral junction stabilization should aim at atlantoaxial stabilization and for achieving segmental atlantoaxial arthrodesis.[5-7] The inclusion of the occipital bone and subaxial spinal vertebrae is not only not indicated but provides a suboptimal form of the fixation construct.[7] The other disadvantage of including the occipital bone in the fixation is a severe restriction of neck movements. Moreover, despite the external appearance of a firm occipitocervical implant, the instability at the atlantoaxial joint persists if the atlantoaxial facets are not included in the fixation construct.

Atlantoaxial dislocation has been classified into two types, mobile type, and irreducible or fixed variety. Basilar invagination was earlier associated with fixed variety of atlantoaxial dislocation. Basilar invagination is a manifestation of chronic or long-standing atlantoaxial instability. We identified that all dislocations are potentially reducible either

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on dynamic neck movements or by the direct distraction of the facets of atlas and axis.[8,9] The atlantoaxial facets in cases with "irreducible" atlantoaxial dislocations are abnormally mobile and can be reduced by manual manipulations.[10,11] While acute and mobile atlantoaxial dislocation is associated with pain and spasm of the nape of neck muscles, chronic instability is associated with a string of musculoskeletal and neural changes that are all secondary and naturally protective mechanisms that are aimed to reduce the effect of instability. Short neck results in a reduction in the length of the spinal cord and a reduction of a stretch of the cord over the indenting odontoid process. Shortening of the neck length is an event that is a result of chronic or long-standing spasm and an attempt by the body to reduce the flexion of the neck. However, the neck can perform extension movements, and even hyperextension is possible. Torticollis occurs when the instability is more unilateral. The most pronounced changes in the spine occur in proximity to the indentation at the odontoid tip, and assimilation of the atlas and C2-3 fusions are initial attempts to reduce the local length of the spine.[12] The entire craniovertebral angulations change to affect a smooth traverse of the cord over the indenting odontoid process. Platybasia and alteration in the angulation of the clivus are attempts in this direction. In the neck, shortening is affected by initial attempts at the reduction in the disc space height, an event that radiologically simulates degenerative spinal changes.[12] Secondary degenerative changes such as osteophyte formation and ligamentous buckling occurs frequently in such a situation. The spinal cord by itself can thin out to tolerate the chronic instability and potential injury to the spinal cord. The spinal cord dangles within "ocean" of fluid that is entirely around the cord or is within its substance in the form of syringomyelia. It appears that the excessive cerebrospinal fluid around the cord can be labeled as "external" syringomyelia. We recently hypothesized that Chiari malformation is like Nature's airbag that is placed into position to cushion the critical neural structures in the region and protect it against potential or manifest atlantoaxial instability.[13,14] In all these events, atlantoaxial instability forms the primary or nodal point of instability. All other joints, including the occipitoatlantal joint, are in a reparative or protective mode. Assimilation of the atlas is a frequently observed phenomenon in the event of chronic atlantoaxial instability and basilar invagination. Occipitoatlantal joint reacts in events of chronic atlantoaxial instability by reducing its movements further and ultimately ends up in the spontaneous fusion of the region.

In cases with chronic atlantoaxial instability and with basilar invagination, it is the atlantoaxial joint that is the functionally abnormal. For any fixation construct to be successful, the fixation has to be segmental and at the site of pathology. Direct atlantoaxial joint fixation by disabling the abnormally hyperactive joint and fixation of the facets has been known to be the biomechanically most effective method of fixation. Any fixation that is remote from the site of movement can provide less than optimum stabilization. The inclusion of the occipital bone and the subaxial spinal segments in the fixation construct adversely affect the stability and can severely restrict the neck movements.

Screw or wire fixation of the occipital end of the implant with the help of appropriately contoured loops or pins has been effectively used.[5] The stabilization of the occipital end of the implant can be less than optimum form of fixation, as the thickness of the occipital squamal bone can be a little too thin to provide a stable base for the screw or wire fixation. Turning of the neck can result in a relatively severe torque that can affect the stability of the implant. Exposure of the atlantoaxial joint is relatively difficult in cases with irreducible atlantoaxial dislocation and in cases with basilar invagination due to the rostral location of the atlantoaxial joint and facets. Short neck, torticollis and assimilation of the atlas make the exposure further difficult. Moreover, the vertebral artery course and its bone relationships can be altered making the exposure surgically challenging and complex. However, atlantoaxial joint exposure, opening of the joint cavity, denuding of the articular cartilage, introduction of the bone graft within the joint cavity, and direct fixation of the facets with screws and rods/plates provide the most optimal form of stabilization that is focused at the fulcrum of all movements in the region. With our increasing experience in surgically dealing with the region in general and particularly in exposing the atlantoaxial joints, we conclude that direct atlantaxial joint fixation form the key to treatment of atlantoaxial instability. The inclusion of the occipital bone not only forms a weak point of fixation but is mechanically incorrect and a suboptimal form of fixation. We believe that occipitoaxial fixation that does not include atlantoaxial joint manipulation and direct fixation of facets of atlas and axis can seldom if ever result in a successful stabilization. Occipitoaxial fixation can mechanically affect the atlantoaxial region that can result in neurological deterioration. Stabilization of the most mobile joint of the body can be most effective when limited spinal segments are incorporated in the fixation process.

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