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Editorial

Instability and basilar invagination

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Basilar invagination is significantly common in India and in the Indian subcontinent. Even in India, there is a disproportionately high incidence in north-western belt of the country. No genetic factor has been identified that could explain the discrepancy in incidence. Our understanding in the subject suggests that muscular weakness of the neck due to protein-calorie mal-nutrition might be the key initiating factor in providing a foundation of instability that leads to formation of abnormality of basilar invagination. Poor delivery practices and excessive use of forceps may have a role in initial injury to the craniovertebral junction muscles and initiation of the process of basilar invagination. Degeneration of the spine in general and craniovertebral junction in particular can lead to instability and subsequently to basilar invagination. Some workers had related lifting of heavy weights on the head to spinal degeneration. Deficiency in specific vitamins has also been incriminated to be causative.

Basilar invagination and its associated physical and musculoskeletal changes seem to be secondary to the factor of instability and neural compression at the medullospinal level. Characteristic physical body features such as short neck, torticollis, platybasia, spondylotic cervical changes, bony fusions, and Klippel-Feil abnormalities are all secondary to primary cord compression.[1] Although, compression and distortion of the neural structures is a factor, micro-trauma due to repeated cord injuries due to instability seems to be the defining factor in the entire pathophysiology of basilar invagination.

Occipitoatlantal joint is the center for stability and atlanto-axial joint is the center for mobility. These two joints form the centers for stability and of mobility of the most stable and most mobile joints of the body. The general dictum in the formation of craniovertebral junction of any animal is that stability of craniovertebral junction is inversely related to mobility. More is the mobilityof the neck less stable it is and vice versa. Atlanto-axial joint is the center for mobility and also the center for instability. All instability of the craniovertebral junction can generally be related to atlanto-axial joint. Stabilization of the atlanto-axial joint forms the basis of stabilization of the craniovertebral junction. Inclusion of the occipital condyle or occipital squama in the fixation construct appears to be a suboptimal form of stabilization.[2] Even in cases where there is assimilation of the atlas in the occipital bone, the fixation can be focused on the facetsof the atlas and axis.

Facets of atlas and axis and occipital condyles form the "V" of the spinal "Y." The role of facets in the spinal stability is relevant in the entire spine and more particularly in the craniovertebral junction. The role of the facets in the spinal stabilization in general seems to be most ill-understood and therapeutically the strength of the facets is least exploited. Our studies have emphasized the significance of facetal distraction and fixation for the entire spine, particularly of the craniovertebral junction.[3-5]

Standing human position brings in additional stresses on the muscles of the back. Muscle abuse and disuse leads to the so called "spinal degeneration" that ultimately leads to telescoping of the individual spinal elements.[5] Instability of the craniovertebraljunction leads to "listhesis" of facet of atlas over facet of axis, "retrolisthesis" of facets of the sub-axial cervical spine and dorsal spine and facetal overriding of the lumbar spine.[5,6] Basilar invagination can be renamed as listhesis of facets of atlas over axis.[6] The treatment of basilar invagination is basically treatment of listhesis. Facetal distraction and fixation of the atlanto-axial joint simulates treatment of listhesis of the sub-axial spine.

We observed that more severe the instability, more acute is the clinical presentation, more severe are the neurological deficits

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and relatively less severe are the physical and morphological musculoskeletal alterations. Essentially, it appears that the commonly observed physical changes such as short neck, torticollis, platybasia, bone fusions, and Klippel-Fiel abnormalities, secondary spondylotic spinal changes and neural changes like Chiari malformation and syringomyelia are all centered on the basic single issue of instability. Essentially the presence of complex of short neck, platybasia, and superior migration of the odontoid process are indicative of potential or manifest instability of the atlanto-axial joint. It appears that the entire complex of basilar invagination and related malformation are related to acquired instability of the atlanto-axial joint rather than being a congenital malformation. It may even be possible that the instability of the craniovertebral junction is present at the time of its formation in the fetus or at the time of its maturation in early infancy. The musculoskeletal and the neural changes that occur are secondary events and all are protective in nature and aim at minimizing the trauma to the neural structures.

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