

Editorial

Atlantoaxial instability: Analyzing and reflecting on the Nature’s reparative games

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“Look deep into nature, and then you will understand everything better.”

— Albert Einstein

“The Glory of God is to conceal a thing, but the Glory of a King is to find it out: As if according to that innocent and affectionate play of Children, the Divine Majesty took delight to hide his works, to the end to have them found out; and as if Kings could not obtain greater Honour, than to be God’s play-fellows in that game.”

— Sir Francis Bacon

“In Nature’s infinite book of secrecy, A little I can read.”

— William Shakespeare

As we develop a more mature and more subtle understanding of craniovertebral junction region, its pathology and its surgical treatment, we begin to realize that instability of the atlantoaxial joint forms a common primary pathological entity, and stabilization of the atlantoaxial joint is the principal mode of treatment.^[1,2] The extensive movements that occur in the atlantoaxial joint necessitate a flat and circular joint orientation, as ball and socket joints, hinge joints, and curved articulations are restrictive. The circumferential nature of movements at the atlantoaxial joint necessarily involves soft and supple yet firm and strong ligaments. The consequence of harboring the luxury of most diverse form of movements is the possibility of

an error of formation or in nurturing. The most mobile joint is the most likely candidate to become unstable. It is like the cells that multiply most rapidly are most likely to become cancerous by multiplying abnormally. Atlantoaxial instability is the most common joint instability and majority of pathological lesions seen in the craniovertebral junction are a consequence of reparative Natural efforts that attempt to stall or delay the neurological symptoms and deficits that can jeopardize the human existence.

Nature that designs and articulates the most complex and diverse life forms, knows all too well the techniques and methods of repair when their own creation is in danger.^[3,4] In the eventuality of atlantoaxial instability, the reparative mechanisms reach their prime and the entire musculoskeletal structures and neuronal tree play their roles in tiding over the crisis that threaten the very existence and survival. The Nature’s play starts and progresses over several months or years without the owner of the body realizing the fact that he is harboring an unstable critical joint that has the potential of threatening his existence. When the natural “games” reach a climax, the clinical manifestations signal the threat. During these harrowing times, the subtle beauty of the Nature must be acknowledged with a sense of wonder and awe.^[3,4]

In the event of an acute atlantoaxial dislocation, the stability of the neck is taken over by the muscles that develop spasm,

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stiffness, and pain. The purpose of these natural maneuvers is to stabilize the spine and to restrict all movements.^[5] The neck loses its curvature and movements. During this period, analgesic drugs may help in relieving the pain, but essentially are against the natural protective designs. In longstanding instability, the neck muscle spasms persist for long periods and eventually musculoskeletal alterations result in short neck and frequently torticollis. Shortening of musculoskeletal structures does not translate to neural shortening. Essentially, short neck results in reduction of stretch over the spinal cord that now traverses in a more relaxed fashion over the bump of the odontoid process and is best suited to wither off the potential or manifest compressive effects.

Shortening of the neck is a result of series of changes that take place in the bones and in the soft tissues. The first thing that comes into play whenever there is a danger of compression or of space reduction is that the fluids make way for the intruder or aggressor. In the case of brain or spinal cord tumor, cerebrospinal fluid (CSF) is the first to get out of the cranial or spinal compartment. In the case where the neck needs to become short, the biggest fluid compartment of the spine, namely the intervertebral discs, gradually reduces in thickness. The reduction in the disc space height in this condition mimics the reduction seen in degenerative cervical spondylosis. Reduction of disc space height results in buckling of the posterior longitudinal ligaments and secondary osteophyte formation, an event frequently seen in cases with shortening of neck.^[6-11] The most effective shortening of the neck occurs in the upper cervical spine, close to the craniovertebral junction. High cervical disc space reduction and osteophyte formation is more often a consequence of shortening of upper neck. Longstanding reduction in disc space ultimately results in vertebral body fusions. The more frequently encountered fusions occur in the regions close to the tip of the odontoid process. Assimilation of the atlas and C2-3 fusions are commonly seen in cases with short neck. Klippel-Feil abnormality or cervical spinal vertebral body fusions participate in the reduction of neck height.^[5] In addition to reduction in the neck height, there is also reduction in clivus height or size. Platybasia or a more horizontal lay of the clivus and a reduction of its size appear to be the cranial component of reduction of length of the highway designed for brainstem-spinal cord. Reduction in the neck and cranial size are net effects of longstanding atlantoaxial instability.

The shortening in the neck/cranial size results in alterations of movements. The neck flexion movement is most affected and reduced. On the other hand, even hyperextension of the neck is possible.^[12,13] These alterations in neck movement effectively result in reduction of movements that are most likely to cause stretch of the spinal cord over the tip of the odontoid process.

The net result of reduction in the neck (and cranial) size is to provide more vertical space to the spinal cord. The Nature also strives to provide horizontal space to the cord. The spinal canal dimension that is maximum at the level of atlas

(not at foramen magnum) is the high potential site to get compromised in the eventuality of atlantoaxial instability. Bifid anterior and posterior arches of atlas are frequent associations of longstanding atlantoaxial instability. Bifid anterior and posterior arches widen the horizontal space at the level of arch of atlas.^[14] The segments of the split arches of atlas and the facets are displaced laterally to widen the circle of atlas. The bifid arches have a dynamic character, the posterior bifid opens up on neck flexion when more space is needed to avoid cord compromise and closes on neck extension. The bifid nature of posterior arch also prevents the critical spinal cord being directly compressed between bones and is a form of natural decompression or laminectomy.

We recently identified that osteophyte formation and ossification of the posterior longitudinal ligament are natural events that are secondary to instability of the cervical spine.^[15-17] The abnormal bone formation in both these situations is a Nature's mechanism that attempts to introduce measures that could additionally support the spinal pillar. Retro-odontoid and paraodontoid ossification are similar attempts at stabilizing an unstable atlantoaxial joint.^[18,19] Such protective ossifications are similar to crust-like formations that surround the base of an unstable tooth.

The entire neural tree also gradually reforms itself to wither the potential storm of atlantoaxial instability. Various neuronal alterations occur in the event of potential or manifest atlantoaxial instability. The ingenious games of the Nature and almost divine play of "fluids" can be appreciated in the circumstance. CSF that is present outside the spinal cord is also present within its substance and around each neuronal fibers that swim freely within their watery pool. In an acute stage, CSF escapes out reducing both the extraspinal and intraspinal cord content.^[20,21] The spinal cord appears "atrophic" and "shriveled" and is deprived of its cozy habitat only for larger aim to protect life by providing additional space for the potential or real bony intrusion. In longstanding or chronically unstable situations, there is excessive CSF formation both inside (syringomyelia) and outside the spinal cord (external syringomyelia) and inside the brainstem (syringobulbia) and outside the brainstem (external syringobulbia) and within the cerebellar hemisphere that becomes atrophic. The cerebellar tonsils herniate down into the spinal canal only to provide a cushion-like support to the spinal cord at the level of atlas, an event commonly recognized as Chiari 1 malformation.^[22,23] The watery and soft tissue supports are introduced as protective measures. It is fascinating to realize that the spinal cord may contain a large amount of syrinx fluid, but the neurological dysfunction occurs late and is proportionately miniscule or insignificant.

It does appear that the reparative or protective natural measures are just as "divine" as the methods that come into play when a human body is "manufactured" from two innocuous looking cells. It is clinically and therapeutically important to evaluate and understand these ways of the Nature. Or else, one can call them errors in development or relate them to embryonic

dysgenesis and treat these protective miracles as pathological. Essentially, it means that short neck, torticollis, platybasia, Klippel-Feil abnormality, secondary spinal osteophytic changes, retro-odontoid bone formation, syringomyelia, Chiari 1 malformation and several similar alterations are secondary to a single pathological issue of atlantoaxial instability. Stabilization of atlantoaxial joint is the treatment of choice. All the mentioned "secondary" and "protective" natural events are most likely to eventually reverse after stabilization is achieved.

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