Is focal spinal cord "atrophy" an evidence of chronic spinal instability?

In the event of chronic spinal instability, a number of natural events occur that have an influence on stalling or delaying the neurological manifestations. These protective measures include a range of musculoskeletal and neural alterations.[1] It is important to realize that several of these secondary natural measures may appear abnormal or pathological by themselves, but actually are "divine" interventions aimed at preservation of neural function and quality of life. Conventionally, such secondary manifestations have been considered to be abnormal and some related to embryological dysgenesis.

Atlantoaxial joint is the most mobile joint of the body. Its flat and round articular surfaces are architectured to provide circumferential movements. However, the joint design makes it most susceptible to the development of instability. Our clinical experience suggests that atlantoaxial joint instability is under-recognized and undertreated clinical entity. While acute atlantoaxial dislocation results in dramatic neurological dysfunction, chronic, or subtle atlantoaxial dislocations is compatible with long-term marginal symptomatic or even symptom-free existence.

In our earlier publications, it was identified that musculoskeletal events such as short neck, torticollis, Klippel-Feil abnormalities, assimilation of atlas, C2-3 fusion, bifid posterior arch of atlas, and platybasia are secondary natural responses to chronic or longstanding atlantoaxial dislocation.[1-3] Chiari malformation and syringomyelia are secondary neural events related to atlantoaxial instability.[4-6] Several previous publications identify all these musculoskeletal and neural events "pathological" and related to disordered formation during embryonic period. The surgical treatment has been directed "against" these entities and attempts of various kinds have been made to rectify such errors.

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Our results identified that a single act of atlantoaxial instability has a potential of reversal of all these secondary musculoskeletal and neural manifestations.[1] Some of these reversals can be identified in the immediate postoperative period.[7] It appeared that all these alterations are protective in nature and effectively stall or delay the development of neurological deficits. It was suggested that the term craniovertebral alterations or formations was better suited to describe these entities rather than the term craniovertebral junction abnormalities or anomalies.[8,9] The range of alterations is essentially determined by the extent of atlantoaxial instability and the time frame of its genesis. The exact cause of development of atlantoaxial instability varies, and more often it is impossible to determine the nodal point of pathogenesis.

The net effect of all the secondary alterations is to protect the neural structures from indenting pressure of odontoid process. The neural structures become elongated and thinned out and the skeletal cranial and spinal pathways shortened. We identified short neck, short head, and short spine in such a clinical situation.[10] The net effect is that the longer and thinner neural structures now extend over shorter length. The neural structures now have a more relaxed traverse over the indenting odontoid process, thus limiting the potential for injury. Chiari malformation simulates Nature's airbag and is strategically positioned to prevent neural structures from getting compressed between bones in the presence of manifest or potential atlantoaxial instability.[4,5] It appears that nature recognizes the instability before any radiological measures can be used for confirmation and initiates protective responses that develop and mature over the period.

Cerebrospinal fluid (CSF) alterations are the first to enter into the fray of protective forces. Excessive amount of CSF is seen inside the neural structures (syringomyelia, syringobulbia) and outside the neural structures (external syringomyelia

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and external syringobulbia).[11] This CSF cavitation provides a "solid" and noncompressible buffer that protects the neural structures. It is indeed astonishing to see how nature gets rid of more dispensable neural pathways and protects the more important ones in an attempt to reduce the girth of the craniocervical spinal cord while retaining important motor and sensory functions. It can be observed that in the presence of syringomyelia or external syringomyelia, the neural girth is remarkably reduced when clinically noticeable dysfunction may only be marginal.[12,13] Such self-neural destruction can also be observed in slow-growing benign spinal tumors such as neurinomas and meningiomas that occupy remarkable spinal canal space and reduce cord girth by multiple proportion and the clinical manifestation may only be marginal.

Spinal cord "atrophy" can frequently be observed in the craniocervical region in the event of atlantoaxial instability. Such atrophy of the cord is a natural protection wherein the more "essential" or important neural structures are retained and the dispensable neural structures are excluded. The aim of the natural events seems to be focused in reducing the girth of the neural structures to be able to tolerate bone pressure while retaining the neural functions.

A similar reduction in the spinal girth is identified in cases with chronic spinal compressive lesions as seen in degenerative spinal events. The spinal cord reduces in girth in the presence of osteophytes, ligamentous hypertrophy, and disc protrusions. Spinal cord may seem to be compressed or "atrophic" even when there is no adjoining compressing element. The spinal cord atrophy is a natural manifestation of potentially or manifestly unstable spinal segment. The spinal instability in subaxial spine is "vertical" instability and is manifested at the facets.[14] Despite the presence of a significant reduction in the cord size, neurological symptoms may be marginal. In the spinal cord, vertical spinal instability is probably the cause of such alterations in the cord, a process that does seem to be protective and in the interest of the human being. The natural events protect till the time the instability becomes overwhelming and symptoms become manifest, progressive, and disabling. The presence of focal spinal cord "atrophy" or when intraaxial spinal cord alterations are seen on sequences of magnetic resonance imaging should guide the clinician about the possibility of the presence of local spinal instability. When the symptoms are significant, stabilization of the spinal segment is warranted.

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