Spinal deformities form a formidable management and therapeutic challenge. Apart from cosmetic issues, deformities can be a cause of a variety of clinical symptoms and neurological dysfunction. Advances in spinal instrumentation, techniques, and technology have paved a way for restoration of spinal curvature and improvement in functional quality. It is important to identify if the deformity is the cause of clinical symptoms, in general, and neurological dysfunction, in particular. It is also important to analyze if deformity is “fixed” (stable) or “mobile” (unstable) and if the deformed bones are the cause of neural compression and related symptoms. It is crucial to analyze if the deformity is primary or structural or is secondary. From our analysis of the subject, we identify that a number of spinal deformities are not primary but are secondary to a discrete pathogenetic factor. In such cases, treatment of the primary etiological factor is necessary, and direct alteration of spinal deformity may not be indicated or may even be contraindicated. Fixation of a number of spinal segments to restore spinal curvatures can reduce or affect the suppleness of the spinal movements. It is mandatory that a surgeon attempts to analyze if a primary cause of deformity can be identified and treated and direct surgery for deformed spine can be avoided.

MUSCULOSKELETAL DEFORMITIES RELATED TO CHRONIC ATLANTOAXIAL INSTABILITY

Short neck and torticollis are frequent associations of chronic or longstanding atlantoaxial instability.[1,2] The musculoskeletal alterations in the presence of chronic atlantoaxial instability are grouped under the umbrella term “basilar invagination.”[1] Chiari formation and syringomyelia are neural alterations in the face of chronic or longstanding atlantoaxial instability.[3] Muscle spasm in the nape of the neck is associated with “acute” atlantoaxial instability. In chronic or longstanding atlantoaxial instability, chronic muscle spasm and natural attempts at limiting the neck movements result in shortening of the neck. Shortening of the neck is an outcome of reduction in the disc space height, disc bulges, intervertebral ligament (that includes posterior spinal ligament and ligamentum flavum) buckling, osteophyte formation, and bone fusions.[2] Bone fusions around the unstable atlantoaxial spinal segment, namely assimilation of atlas and C2–3 fusions, are more common. Klippel–Feil abnormalities and/or fusion of subaxial cervical vertebrae are also frequent associations.[4] Torticollis can be associated when the atlantoaxial instability is eccentric in nature and rostrally migrated or unstable odontoid process is directed off-midline.[5,6] Such neck deformities and bone and soft tissue alterations can by themselves be the indicators of the presence of atlantoaxial instability even when there or no other positive radiological indicators. It is critical to realize that atlantoaxial instability is the primary or the nodal point of genesis of the entire structural alteration. Atlantoaxial stabilization is the treatment.[7] Neck deformities can be restored toward normalcy as early as in the immediate postoperative period. Despite the radiological appearance of neural “compression” by bones, decompression by any kind of osteotomy is unnecessary. Direct attempt at restoration of neck size or neck deformity can only harm the interests of the patient.

Dorsal spinal kyphoscoliosis is “frequently” associated with syringomyelia and Chiari formation.[8–10] “Short spine” and dorsal kyphoscoliosis are consequences of chronic or longstanding atlantoaxial instability. Untreated rotatory atlantoaxial dislocation can result in dorsal kyphoscoliosis. On the basis of our experience, it seems that majority of cases of dorsal kyphoscoliosis are not primary structural alterations but are secondary manifestations of chronic atlantoaxial instability. The atlantoaxial instability is more
often of “central” or “axial” type.[8,11] There may not be any compression of the dural tube or the neural structures by the odontoid process, and there may not be any facet malalignment. Understanding the issues involved with central or axial atlantoaxial instability is crucial to identify such instability, as there may not be any malalignment or abnormality when assessed by validated radiological parameters. Our earlier articles have analyzed and suggested that such structural alterations are natural protective maneuvers and are secondary to primary atlantoaxial instability. Breathing disturbances, sleep apneas, and neurological dysfunction are secondary consequences of atlantoaxial instability and not related to direct neural “compression” by bones.[12] It is crucial and probably mandatory to realize that direct structural alteration of the spinal deformity is unnecessary and probably harms the interests of the patient. Reversal of spinal kyphoscoliosis following atlantoaxial stabilization surgery is possible as early as in the immediate postoperative period.[8] More importantly, the symptoms of pain in the deformed spinal region, breathing disturbances, and neurological symptoms reverse following atlantoaxial fixation. Although the spinal deformity may not entirely reverse to normal situation, the suppleness of the spinal movements is retained and improves following such surgery. Any kind of spinal bone osteotomy and direct spinal fixation is unnecessary in such cases. Direct manipulation of the spinal bones to restore spinal curvature can only have negative clinical implications.

MUSCULOSKELETAL DEFORMITIES RELATED TO SPINAL “DEGENERATION”

We earlier proposed that it is not disc space reduction or “old-age”–related disc degeneration, but it is the weakness of the muscles responsible for standing human position and the related spinal instability that is the primary point of pathogenesis of spondylotic spinal disease. Spinal instability results in telescoping of the spinal segments, a process that results in reduction in the disc space height, bulging of the disc into the spinal canal, buckling of the intervertebral ligaments that include posterior longitudinal ligament and ligamentum flavum, osteophyte formation, and multiple such so-called “pathological” processes that result in reduction in the spinal canal and intervertebral foraminal size reduction.[13] All these processes can reverse by surgery that involves “distraction” of the spinal segments.[14] We described facet distraction using Goel facet spacers as the treatment for single or multiple segments cervical and lumbar spinal radiculopathy and/or myelopathy.[14,15] Our recent concept is that instability is the cause and stabilization is the treatment for all forms and types of spinal “degeneration.”[16] Cervical spinal kyphosis can be associated with multisegmental spinal degeneration. We presented our philosophy of treatment of such cervical kyphosis. It was identified that multisegmental spinal instability that often includes atlantoaxial instability is the cause of cervical kyphosis.[17] Stabilization of the affected spinal segments is the treatment. Any attempt of resection of the bone or “kyphectomy” can only have negative implications.

We identified that the lumbar spinal deformity or “adult idiopathic de novo lumbar scoliosis” (AIDLS) is a consequence of multiple-level spinal instability.[18] The general understanding is that deformity eventually results in neural compression-related symptoms that are more pronounced on the concave side of the spinal curve. Clinical symptoms usually begin with the observation of deformity and progress to mimic those of lumbar canal stenosis. Decompression of the neural structures has historically formed the basis of surgical treatment. Our article on the subject suggests that for AIDLS, stabilization of the multiple spinal segments is necessary.[18] Resection of the bones for the purpose of “decompression” of the neural structures is unnecessary.

Essentially, it appears that instability of the spinal segments is the cause of majority of so-called “idiopathic” spinal deformities. Spine is generally considered to be “stable” in a majority of spinal conditions, such as spinal degeneration and spinal deformities, and decompression forms the basis of surgical treatment. Our studies on the subject suggest that identification of the cause, deciphering the unstable spinal segments and stabilization of these segments, forms the rational form treatment.[19] Resection of the bone for “decompression” of the neural tissue may not be necessary.

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