Abnormal Upper Cervical Joint Alignment and the Neurologic Component of the Atlas Subluxation Complex

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ABSTRACT

In several upper cervical theories it is hypothesized that an atlas vertebra misaligned out of a presumptive normal horizontal plane causes a change in the vertical orientation and center of gravity of the skull. Resultant altered weight bearing of the atlanto-occipital and atlanto-axial joints are suspected of stimulating joint mechanoreceptors and aberrant proprioceptive spinal input, which initiate spinal reflexes causing body distortion and a functional or contractured leg-leg length inequality.

Two case studies in which there was unusual joint alignment in the upper cervical spine - atlantoaxial rotatory fixation and a hypoplastic occipital condyle - are reviewed. This paper discusses how the clinical findings in these cases of joint alignment anomalies may alter hypotheses regarding the causation of mechanoreceptor stimulation, leg length inequality, concepts of normal atlas orientation, a medical counterpart of a chiropractic subluxation, and the necessity of routinely post-x-raying patients in an attempt to establish physical equilibrium.

Key words: Chiropractic, specific upper cervical, proprioceptive insult hypothesis

INTRODUCTION

In the Chiropractic techniques characterized as specific upper cervical, (Atlas Orthogonal, Chiropractic Biophysics, Chiropractic Orthospinology, Duff, Life Cervical, Mears, NUCCA, Spinal Biomechanics (Pettibon) and Wernsing (1)), the positioning of the atlas vertebra, as viewed by specific upper cervical x-ray, is a critical part of the vertebral subluxation complex. The subluxation hypothesis describes the manner in which displacement of the atlas from an idealized horizontal orientation beneath the skull is suspected of causing altered weight bearing resulting in detrimental biomechanical and neurological effects.

The biomechanical changes noted in the cervical spine are reasoned to be compensatory responses to an alteration in the vertical orientation and center of gravity of the skull. Displacement of the center of gravity of the skull is thought to produce asymmetrical weight bearing in the occipital condyles and lateral masses of the atlas. Pettibon and Harrison provided a formula for estimating the side-to-side weight differential transmitted to C2 and the lower cervical spine as the atlas/skull relationship is deviated from 90° (2). Common radiographic findings suspected of being related to altered weight bearing include lateral bending of the lower cervical spine and rotation of C2 (2, 3).

Neurological effects of altered weight bearing include hypothetical mechanisms by which misalignment of the atlas and axis result in a functional leg length inequality (LLI). One of these mechanisms is the so-called proprioceptive insult hypothesis in which stimulation of mechanoreceptors in the atlanto-occipital or atlantoaxial joints and/or surrounding tissues causes aberrant spinal cord input resulting in altered postural reflexes (4).

In one version of the proprioceptive insult hypothesis, Crowe and Kleinman postulate that "...physical disequilibrium and loss of physiologic motion involving a weight-bearing joint supplies irregular input from the primary sensory modality, and that through axo-axonal and axo-dendritic synapses this irregular supply spills over into the central nervous system feedback loops and potentially into all body systems" (5). In other words, this "physical disequilibrium," which I equate to altered weight bearing of the occipital-atlantoaxial joints, "supplies irregular input" that "spills over into the central nervous system" to affect the reticular formation. Further, they hypothesize that the effect on the reticular formation "physiologically explains the consistent clinical findings of the atlas subluxation complex..."(5).

This proposed mechanism dovetails with Dr. Ralph Gregory's hypothesis that, "...spinal and pelvic distortions from the normal or vertical axis are caused by the displacements of the C-1 subluxation affecting the reticular formation, causing imbalance between the facilitatory and the inhibitory tracts of the brain stem and producing over-innervation throughout the spinal cord, resulting in spastic contracture of the extensors" (6).

Technique systems that depend on a theoretically normal horizontal orientation of the atlas in the frontal plane (1, 2, 7, 8) seem also to require symmetry of the occipital condyles and atlas superior articulating facets. However, anatomical studies of the occipital condyles (9) and the superior articular facets of the atlas (10) have determined that asymmetry is the norm. Indeed, it has been stated that the craniovertebral junction is one of the most common sites of bony malformations (11). While methods exist to account for compensatory biomechanical rotations and bony malformations when analyzing the x-ray and deriving an adjusting vector (1), the effects of such abnormal joint structure and resultant misalignment in regards to the stimulation of joint mechanoreceptors and the production of nerve interference has not been addressed.

This paper presents two cases of abnormal upper cervical joint alignment, one acquired atlantoaxial and one congenital atlantooccipital, and discusses how these joint alignment anomalies may alter hypotheses regarding production of nerve interference, concepts of normal, and the necessity of routinely post x-raying patients to establish a physical equilibrium.

Case 1

An 11-year-old girl suffering from cervical pain, headache, cervical crepitus, and significant head tilt presented for examination and treatment. Her symptoms had begun immediately after an auto accident seven months ago. Prior treatment had included a regime of physical therapy and diversified lower cervical manipulation.

Exam revealed a 10° left head tilt with slight rotation to the right, often called the "cock robin" position. There was palpably increased muscle tone in the right sternocleidomastoid and trapezius, with no increased tension of the same muscles on the left side. There was a left functional short leg.

Active cervical range of motion in right lateral flexion and right rotation were limited to 5° and 10°, respectively; this was only slightly painful and did not elicit muscle spasm. Active attempts with passive assistance to move the head beyond the limit of right lateral flexion caused an apprehensive reaction in the patient, who would tilt her shoulders and thoracic spine to maintain her previous head/shoulder angle. Upon release of the head from forced lateral flexion, the head and neck returned to the left tilted position, in which it seemed to be locked. Motion palpation in right and left lateral flexion revealed asymmetric hypermobility of the C2 spinous process to the left, toward the tilt, and little movement to the right.

Cervical x-ray exams revealed persistent asymmetry of the atlanto-odontoid interval with specific upper cervical (Grostic) analysis showing atlas laterality right (1°) with posterior rotation (4°) and C2 spinous rotation left (5°).

The patient was diagnosed with atlantoaxial rotatory fixation (AARF) (for a complete work-up and explanation, see "Chiropractic Correction of Atlantoaxial Rotatory Fixation, Knutson G., *JMPT* 1996; 19(4): 268–272). The patient required four standard vectored upper cervical adjustments, the necessity based on supine leg check, and one special upper cervical adjustment (see Discussion) over 12 weeks. There was a complete recovery.

Case 2

A 33-year-old female presented for treatment of chronic, insidious, debilitating lower back pain. The pain was worse in the mornings and after periods of inactivity. Significant history included known congenital malformations: a bicornuate uterus and agenesis of a kidney.

The patient had seen a wide variety of health care providers seeking relief from her back pain. Interventions had included chiropractic manipulation, drug therapy, physical therapy, kinesiology, acupuncture, massage, bio-feedback, meditation, visualization, and energy movement. Any relief gained by these treatments had been temporary.

Physical examination revealed taut and painful lumbar erector muscles, pelvic unleveling, and a right functional leg length inequality. Lumbo-pelvic x-rays confirmed the pelvic tilt with slight compensatory lumbar curvature, but were otherwise negative. Cervical x-rays, reviewed by a DACBR, showed hypolordosis, a hypoplastic left occipital condyle and right atlas transverse process, and asymmetric C2 superior articulating facets. Upper cervical (Grostic) analysis found the atlas plane line 8° high on the left, left atlas laterality of 9°, right lower angle of 7°, and axis spinous left 6°. The unusual magnitude of the upper cervical misalignment was most likely due to the hypoplastic left occipital condyle, which produced a high atlas plane line on the left.

The patient was seen six times over a one-month period with atlas adjustments, based on LLI, delivered on the first and last visits. The patient was released from care with a 90+ % improvement in her symptoms, level pelvic crests, and elimination of LLI. In follow-up six months later, the patient was asymptomatic and had no neurological signs of upper cervical subluxation. Due to the unusual nature of the case, and with the patient's permission, a follow-up nasium x-ray was done at that time to assess the non-subluxated orientation of the atlas. X-ray analysis revealed an atlas plane line of 6°, upper angle left 6°, and lower angle right 4°.

DISCUSSION

In Case 1, normal vectored adjustment was successful in decreasing neck pain, muscle tension, and leg length inequality. However, the abnormal rotation of the skull/atlas on the superior articulations of C2 was still present, as evidenced by the continued fixed head tilt (cock-robin position).

I believe that the abnormal joint positioning in AARF is significant for two reasons. First, some upper cervical techniques hypothesize that subluxation and attendant joint mechanoreceptor stimulation at C1/C2 can produce or influence LLI (12). Yet in this case, no LLI was recorded with upper cervical style supine leg check procedures in eight attempts over four visits, despite continued abnormal C1/C2 joint mechanics. If the Crowe/Kleinman hypothesis of physical disequilibrium and presumptive mechanoreceptor stimulation is correct, the continued significant abnormal rotation at C1/C2 should have resulted in

LLI after correction of the atlas/occiput subluxation. It is possible the leg checks were in error and upper cervical subluxation still remained; however, all other symptoms had been alleviated, and only the cock-robin position of the head/neck remained. While only one case, this appears to be evidence that altered weight bearing at the level of C1/C2 may not, by itself, produce the mechanoreceptor stimulation and proprioceptive output hypothesized to produce a contractured leg. As such, it may be necessary to look for alternative neurologic mechanisms for how a C1/C2 subluxation may affect LLI.

Second, atlantoaxial rotatory fixation has been used as an example of a known medical diagnostic entity that is the equivalent of a chiropractic subluxation (13). I believe this is incorrect. In this case, the abnormal joint alignment found in AARF did not appear to produce neurologic insult, either in contribution to a functional LLI or in a segmental facilitatory sense. To label AARF as a chiropractic subluxation, which as a theoretical entity is characterized by a neurologic component (14, 15, 16), becomes problematical. This joint derangement seems better described using the medical definition of subluxation – less than a complete luxation. While this may seem to be merely semantic juggling, atlantoaxial rotatory fixation is a relatively uncommon phenomenon (17), and if taken as a model of a chiropractic subluxation, it would, in my view, incorrectly define and severely limit the understanding of the neurological ramifications of the chiropractic vertebral subluxation complex.

Finally, though the atlas subluxation with attendant neurological insult (LLI) was corrected, the rotatory fixation and head tilt remained. The right posterior contact on the atlas transverse process, derived from the specific upper cervical protocol for adjusting the atlas subluxation complex, corrected the neurological component of the subluxation, but left a significant biomechanical component of C1/C2 rotation.

By considering the auto accident that produced the patient's misalignment, it was ascertained that the force vector from the collision came from behind and to the right of the patient, causing an angular acceleration of the head to the right and posterior. Such an analysis has been shown to be of potential value in determining an adjusting vector for treatment (18). I hypothesized that the axis of rotation for the movement of the skull and atlas had been the right atlantoaxial articulation and that C1 rotation, in relation to C2, would then have occurred to the anterior on the left. An adjustment was performed using a left anterior atlas transverse contact and a height factor based only on the condylar and axial circle diameters, hoping to lever C0/C1 over the superior articulating surface of C2. The adjustment was effective; two days later on reexamination the head tilt was gone and has not returned.

Had this misalignment been the typical chiropractic atlas vertebral subluxation complex, switching the adjustment vector to the side opposite atlas laterality and rotation, as found with upper cervical x-ray protocol, might have been symptomatically detrimental to the patient (19). Such was not the case, giving further credence to the idea that AARF is not a chiropractic subluxation.

Case 2 featured a hypoplastic left occipital condyle, an upper cervical anomaly associated with genitourinary anomalies (20), also found in this patient. Since the condyles are ossified at birth (21), in infancy, as this patient began to hold her head erect, the atlas could not have laid in a horizontal plane. The resultant altered weight bearing seems to have created asymmetry in the superior articulating surfaces of C2. These changes might be explained by Heuter Volkman's Law, which postulates that pressure across an epiphyseal growth, plate controls its growth: increased pressure results in decreased growth and decreased pressure leads to increased growth (22). Developmental adaptations to decreased pressure on the surface of C2 might also explain the flatter articular surface on the side of the hypoplastic condyle.

Such physical disequilibrium, in the Crowe/Kleinman hypothesis, is believed to be responsible for "irregular [mechanoreceptor] input," leading to a contractured or functional short leg (5). If this is correct, the patient would have been expected, from birth, to have suffered from the ramifications of upper cervical subluxation, including body distortion and contractured leg. Yet during the patient's developmental years, as she began to hold her head upright and the joint surfaces reacted to the altered weight bearing, neither the patient nor her parents could recall any indications of head tilt, any structural problem (such as scoliosis), or head/neck/back pain. Indeed, until the patient began to experience lower back pain in her twenties, she had no health complaints. While the internal state of stress on the bones and the amount of physical disequilibrium this birth anomaly engendered is unknown, it is reasonable to suspect some sign or symptom of upper cervical subluxation would have been evident if the physical disequilibrium/altered weight bearing hypothesis of abnormal afferentation, body distortion, and leg length inequality is correct.

It is possible that the physical disequilibrium/altered weight bearing from birth did cause neurologic effects of an atlas subluxation complex until the reforming of the C2 superior articular surfaces was complete. Then, as the patient's "normal" upper cervical biomechanics were established, and the weight of the skull was more equally transmitted through the joints of the atlas and the reformed axis, the presumptive mechanoreceptor stimulation would have ceased. How long the joint reforming would take and concomitant atlas subluxation complex symptoms (pain, body distortion, LLI) would theoretically be evident is unknown. If the joint reforming via Heuter Volkman's Law happened rapidly, it is possible any signs and symptoms of mechanoreceptor stimulation could have been overlooked.

The scenario discussed above might explain how significant structural anomaly could be present without causing notable signs and symptoms. If this is correct, it raises the question – is the same weight bearing/joint reforming process taking place in every patient with an upper cervical subluxation complex? Does the body reform the upper cervical joints to compensate for physical disequilibrium/altered weight bearing in all subluxated patients? If so, then, mechanoreceptor stimulation would be expected to cease as well when remodeling is complete and the neurological signs of subluxation would disappear.

Based only on personal clinical observation, leg length inequality/contracture does not disappear with time, even in children. In my practice I have seen patients who were aware of LLI for years, some treated with heel lifts, yet whose contractured leg responded immediately to upper cervical adjustment. If LLI is the result of physical disequilibrium/altered weight bearing in the upper cervical spine, joint reforming via Heuter Volkman's law does not seem to eliminate the presumptive proprioceptive insult. As the previous case suggested, physical disequilibrium/

altered weight bearing may not be the primary source of the neurological afferentation that is hypothesized to produce body distortion and contractured leg.

While a vertical orientation of the skull is recognized as something the body tries to attain (23, 24, 25), in this case to the point of altering the curvature of the superior articulating surfaces of C2, such an orientation may result in an atlas plane line appearing slightly off a perfect horizontal plane even in the absence of neurological signs of subluxation. If a horizontal atlas and complete reduction of the upper angle is not, in all practicality, attainable, then a successful reduction of the upper cervical subluxation might better be assessed by the elimination of the neurologic component, the contractured or short leg, and not by seeking to have "...the C-1 subluxation zeroed out" (6) structurally. Given this, routine post-adjustment x-raying in absence of observable nerve interference (LLI) in order to promote physical equilibrium/equal weight bearing might not be necessary. This prompts a testable hypothesis: does the post x-ray provide information that leads to better patient outcome? An answer to this question could come from studying patient outcome and comparing patients who were post x-rayed with those who were not.

CONCLUSION

Two case studies of abnormal upper cervical joint alignment are presented, and the potential neurologic consequences are discussed. In the first case, significant axis (C2) rotation and alteration in normal upper cervical weight bearing was present after the neurologic component of atlas subluxation complex, leg length inequality, had been normalized. The inference that can be drawn is that physical disequilibrium/altered weight bearing causing presumptive mechanoreceptor stimulation of the atlantoaxial joints may not be associated with the production of leg length inequality.

In the second case, an upper cervical birth anomaly that undoubtedly was the cause of altered weight bearing, did not seem to cause any signs or symptoms of upper cervical vertebral subluxation complex. Eventually the atlantoaxial joints reformed to accommodate the altered weight bearing in accordance with Heuter Volkman's law. The inductive conclusion reached was that physical disequilibrium/altered weight bearing in the upper cervical spine may not be the primary source of the neurological afferentation that is hypothesized to produce body distortion and contractured leg.

Crowe and Kleinman hypothesized that "physical disequilibrium *and* (my emphasis) loss of physiologic motion" causes the "irregular supply [that] spills over into the central nervous system"; perhaps, given the tentative conclusions reached from the cases presented here, looking to the loss of physiologic motion as the source of the abnormal afferentation responsible for the putative reflexes causing the contractured leg may be more productive.

Given the findings in these cases and the anatomical asymmetry of both the atlas and the condyles, perhaps an ideal horizontal atlas, a 90° upper angle relationship – physical equilibrium, is not the goal for the specific upper cervical chiropractic adjuster to strive for. As such, the routine post x-raying of patients for the purpose of establishing physical equilibrium/equivalent weight bearing, in absence of nerve interference – body distortion/contractured leg – is brought into question, and a possible hypothesis for studying the necessity of the post x-ray procedure is suggested. It must be emphasized that these are just two cases, but they are unique with respect to upper cervical joint alignment. The cases give rise to critical speculation regarding a hypothetical mechanism for the clinical findings of specific upper cervical techniques and invite further explanation, clarification, and research. ◆

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