Tonic Neck Reflexes, Leg Length Inequality, and Atlanto-Occipital Fat Pad Infringement: An Atlas Subluxation Complex Hypothesis

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ABSTRACT

This study presents a new theory to explain misalignment, relative fixation, and the neurologic manifestation of a contractured or functional leg length inequality in the atlas subluxation complex.

The proposed mechanism of fixation involves impingement/extrapment of the atlanto-occipital intra-articular fat pad causing nociception and reflexive guarding contraction of the suboccipital muscles. Stimulation of spindles in the suboccipital muscles are hypothesized to be involved in the initiation of tonic neck reflexes that alter global extensor muscle tone to achieve proper body balance in response to head movement. This altered muscle tone caused by the tonic neck reflexes is hypothesized to be responsible for the clinical finding of a contractured or functional leg length inequality associated with atlas subluxation complex.

Short reviews of other theories in which atlas subluxation complex results in leg length inequality – dentate ligament/cord distortion and proprioceptive/mechanoreceptor insult – are provided, clinical correlations with the new theory are noted, and potential methods by which to test this new theory are outlined.

INTRODUCTION

In the chiropractic profession, a long noted clinical association of functional leg length inequality (LLI) with upper cervical misalignment/subluxation complex exists. Although the clinical tests of leg length inequality suffer from subjectivity (1), researchers have a attempted to objectify the perceived phenomenon (2, 3). One case study has shown a significant statistical correlation between an objective measurement and the subjective supine leg check test (4). Given the emphasis on the use of leg checks in upper cervical practice, research is needed to establish a "gold standard" procedure exhibiting acceptable inter- and intra-examiner reliability.

Two competing theories address the relationship of putative atlas subluxation complex to LLI: the dentate ligament/cord distortion hypothesis, developed and championed by the late Dr. John Grostic, and the proprioceptive insult – or more accurately mechanoreceptor insult (5) – theories, which come with many differing hypotheses.

Although these theories provide a neurologic rationale for leg length inequality resulting from atlas subluxation complex, and both theories presume the atlas in subluxation to be relatively fixed, neither provides a hypothesis as to what causes or sustains the fixation.

This paper will critically review the existing theories and propose a more comprehensive theory as to the cause of the atlas fixation and the neurologic mechanism that produces the functional leg length inequality.

Dentate Ligament/Cord Distortion Hypothesis

As proposed by John D. Grostic, DC., FICR, translations of the atlas vertebra up the occipital condyle (atlas laterality) of more than 3/4° or 0.75 mm, causes tractioning of the spinal cord via the dentate ligament, which is firmly attached to the spinal cord and, hypothetically, through the *dura mater* to the inner ring of the atlas (6). Such tractioning of the spinal cord is hypothesized to cause venous blockage and hypoxia in the spinocerebellar tract. The spinocerebellar tract is the main conduit for sensory information on muscle tone, joint position, and the internal

state of lower motor centers to the higher centers of the brain. Hypothetically, these higher centers, receiving the altered spinocerebellar signals, increase muscle tone, which results in hypertonicity and functional short leg.

The weaknesses in this theory is that many authorities see lateral flexion at C0/C1 as normal; White and Panjabi report that 8° of lateral flexion normally occurs at C0/C1 (7). A small study (n=4) using specific upper cervical x-ray procedure and analysis found that there is an average change of 2.5° between C0/C1 from a neutral and laterally flexed position (8). A literature review reported in that same study found reports of lateral flexion at C0/C1 ranging from 8° to 3°. All these studies found normal C0/C1 lateral flexion motion to be much greater than the 3/4° proposed in the dentate ligament/cord distortion hypothesis as the maximal amount of normal movement, bringing this theory into question.

In regards to lateral movement of the atlas, a computerized tomography study of 26 healthy subjects found that lateral displacement of the atlas with respect to the occiput has a mean value of 4.4 mm to either side (9), far more than the hypothetical maximum of 0.75 mm the dentate ligament/cord distortion hypothesis postulates. The ability of the atlas to move laterally to such an extent may be why, in most anatomical texts, the *dura mater* is not described as attaching to the inner ring of the atlas. Such movement, with dural attachment, likely would cause traction of the spinal cord/medulla oblongata.

So, although dentate ligament / spinal cord traction may not be the mechanism responsible for the production of the short leg in all cases, it may be a causative factor in cases where the *dura mater* does attach to the atlas.

TABLE 1
Types of Articular Receptors

Type I	Mechanoreceptor, low threshold (fire easily), slowly adapting, found in fibrous capsule of joint and in periarticular ligaments and tendons, proprioceptive, provides information whether joint is moving or not, respond primarily to extreme range of motion.
Type II	Mechanoreceptor, low threshold, rapidly adapting, found in deeper layers of fibrous capsule, at junctions of fibrous capsule and fat, and in fat pads, stimulated at beginning and end of joint movement and are not active in non-mobile joints, respond primarily to extreme range of motion.
Type III	Mechanoreceptor, high threshold, very slowly adapting, found in ligaments, tendons and dense fibrous connective tissue of the joint capsule, stimulated at extreme ranges of motion and positions harmful to the joint.
Type IV	Nociceptor, non-adapting (does not fatigue), found in dense capsular tissues, synovial and areolar tissues, and in all periarticular and intraarticular tissues except for articular cartilage, not active under normal joint conditions.

Proprioceptive/Mechanoreceptive Insult Theory

Before beginning a review of the proprioceptive/mechanoreceptive insult theories, a quick review of spinal joint neurology is in order. Anatomical examination of cervical facet joints found mechanoreceptors (based on a classification scheme by Wyke) Types I, II, III, and IV (10) (see Table 1). Types I, II, and III are thought to be involved in proprioception, responding primarily to extremes of motion (9), whereas Types III and IV signal harmful positions (11). Type IV are pain receptors (nociceptors). Studies of peripheral joints suggest that joint mechanoreceptors monitor joint movement and capsule tension and may initiate protective muscular reflexes (10). Except for articular cartilage, all tissues of the synovial joint receive some form of innervation (10). Nociceptive nerve endings were found in dense capsular tissues, synovial and adipose tissues and ligaments (10, 12).

Proponents of the proprioceptive/mechanoreceptive insult theories as an explanation of the upper cervical subluxation have written that the distribution of joint mechanoreceptors in the vertebral column is greater in joints closer to the head, with the highest concentration of receptors being in the occipital-atlanto-axial joints (13). Because of this high receptor concentration, it is believed that vertebral subluxation – joint dysfunction, misalignment, and altered weight bearing – causes mechanoreceptor stimulation and aberrant afferent input into the spinal cord, inducing changes in neural output that result in a functional or contractured leg. In contrast to earlier reports, a recent study of mechanoreceptors in human cervical facet joints from C1/C2 to

C7/T1 found that "there was no apparent difference in the distribution of receptors within the facets of the upper cervical versus lower cervical spine" (10). Another study found that manipulation of the lower cervical spine had a greater effect on lumbar spinal muscle tone than manipulation at C2 (14), again indicating that spinal cord input from mechanoreceptor stimulation is not necessarily concentrated in the upper cervical spine at the C2 level. Finally, joint receptors are concerned with signaling joint movement, not position, over most of the joint's working range (15). Unless the atlas in subluxation is positioned at the extreme end of atlanto-occipital joint range of motion, it seems problematical that joint mechanoreceptors could chronically fire and cause abnormal afferentation and resultant LLI.

Review of Proprioceptive/Mechanoreceptive Insult Hypotheses

Cockwill provides a detailed overview of these types of proposed neurological mechanisms (16). The first involves the aberrant afferent input from mechanoreceptor stimulation spilling into the reticular formation (13). The more cephalic connections of the reticular formation facilitate spinal motor discharge, whereas the more caudal exert an inhibitory action (17). Abnormal proprioceptive/mechanoreceptive input into the caudal portion of the reticular formation is thought to suppress the normal inhibitory influences of muscle tone, thereby creating body distortion and a contractured leg (13).

The second proprioceptive/mechanoreceptive insult hypothesis involves spinal reflexes associated with the vestibular system. The vestibular system is exquisitely sensitive, capable of discriminating to even one-half degree of head tilt (18). Tilting the head also stimulates upper cervical mechanoreceptors that reflexively inhibit the extensor reflex in the vestibular apparatus (18). Abnormal mechanoreceptor stimulation in the atlanto-occipital joint resulting from misalignment and altered weight bearing is hypothesized to cause a nonfatiguing facilitation of mechanoreceptor signals to the utricle and saccule, which results in extensor muscle spasm and a functional short leg.

The Tonic Neck Reflex

The final proprioceptive insult theory, the Tonic Neck Reflex, is perhaps the most promising as an explanation of the functional short leg. Two basic reflexes allow the body to maintain an upright posture: the vestibular labyrinth, which keeps the head perpendicular to the ground using the prime mover neck muscles, and the tonic neck reflexes, which align the body to the position of the head and stabilize the trunk with respect to the vertical axis by activating extensor muscles (19). These reflexes work in opposition, allowing the head to move on a stable platform without movement of the trunk or limbs (20, 21).

Tonic neck reflexes were originally thought to be a manifestation of incomplete neurologic development (22, 23). However, Tokizane, et al., demonstrated muscle tone changes of the tonic neck reflex electromyographically among healthy adult humans (20). Since then, Hellebrandt and others have demonstrated these reflexes in normal children and adults by using postural tests (22-25). Tonic neck reflexes are classified as symmetric, in which flexion and extension of the head causes symmetrical muscle changes in all limbs (19, 20), and asymetric, in which rotation and lateral flexion of the head causes one-sided changes in extensor muscle tone (25). Less used classifications of the asymmetric tonic neck reflex are normal, in which the reflex attenuates after several seconds (26), or pathologic, where the reflex, left alone, does not attenuate (20, 25, 27).

In evoking the asymmetric tonic neck reflex, lateral flexion of the head is associated with shortening of the contralateral leg or arm, and rotation of the head is associated with more pronounced leg shortening ipsilateral to the side of rotation (19, 22, 24, 25). Hellebrandt found that head rotation would produce relative leg length differential of up to two inches and combinations of head movement – rotation, lateral bending, and/or sagittal plane movement – produced mixed changes in the relative leg length, depending on dominant influence of each component and whether the influences were harmonious or conflicting (22).

Although the effects of the tonic neck reflexes can be observed in humans, the study of the mechanism has been done primarily in cats. Hellebrandt believed the source of the tonic neck reflexes to be the proprioceptors located in the upper cervical spine (22). Originally the upper cervical joint mechanoreceptors were thought to be responsible, because cutting the nerve roots to the upper cervical joint capsule of a cat in which all the major muscles of the head/neck had been dissected away abolished the tonic neck reflexes (28). However, joint mechanoreceptors were not found to be in special abundance in the upper cervical spine, and it is now believed that muscle mechanoreceptors in the form of muscle spindles, concentrated within the small intervertebral upper cervical muscles, are the probable source for signaling head position and the tonic neck reflexes (15, 29, 30).

The spindle index or density is the number of muscle spindles per gram of muscle and ranges from 5 to more than 120 in cat skeletal muscle (31, 32). Anatomical studies on cats have shown that the small perivertebral muscles contain a profusion of muscle spindles at a density of up to 500 spindles/g, whereas the spindle concentration in the large dorsal cervical muscles, previously thought to be of high density, was 100/g (33). In humans, researchers have found a "bewildering number of spindles" in the suboccipital muscles (34).

Higher muscle spindle density suggests that the spindle system can supply fine-grain information about the muscles (32, 33, 35, 36). Higher spindle density is found in muscles responsible for delineating fine movement, including lumbricals, extraocular muscles, small muscles of the hand, and small vertebral muscles (31, 33, 35).

In the upper cervical spine of the cat, the muscle spindles are oriented in a variety of planes, suggesting that they "...create an accurate three-dimensional picture of vertebral alignment, in its many degrees of freedom, by monitoring muscle events in many slightly different planes around the vertebral column (30)." Spindles in the upper cervical muscles can provide a detailed pic-

ture of head position and movement (29). Indeed, in experiments with cats, the cervical perivertebral muscle spindles showed large changes in firing rates with the smallest of movements of vertebral joints (36).

In humans, the muscles and spindles believed to provide the signals for the tonic neck reflexes are the atlanto-occipital muscles, including the rectus capitis lateralis, obliquus capitis superior and rectus capitis anterior.

Pain-Spasm-Pain Cycle

A major problem with the proprioceptive/mechanoreceptive insult theories is that mechanoreceptors are subject to fatigue; the mechanoreceptor insult wears out. If proprioceptive/mechanoreceptive insult produces LLI by one of these neurologic reflexes, then it would be expected that LLI would itself be subject to fatigue. Clinically, this does not seem to be the case. Problems with the hypothesis of altered weight bearing, proprioceptor/mechanoreceptor output, and fatigue have been noted elsewhere (37). Nociceptors do satisfy the requirement for a non-fatiguing somatosensory receptor, but after initial tissue insult and healing, it seems unlikely that nociceptive stimulation would continue.

Roland has reviewed the establishment of a pain-spasm-pain cycle as the cause of musculoskeletal disorders, including chiropractic subluxation (38). Such a cycle may be responsible for the nociceptive input necessary to initiate the spinal reflexes producing the short leg. However, as Roland points out, if the pain-spasm-pain cycle theory is correct, eliminating pain with analgesics or muscle spasm via biofeedback, physical therapy, or muscle relaxant drugs should interrupt the putative feedback loop. Although this type of positive feedback loop may be active in some cases of musculoskeletal pain, many patients seeking chiropractic evaluation have been treated already using methods that would abolish the cycle without long term relief of their complaint.

In normal atlas movement, researchers have recorded as much as 8° (7) of lateral flexion and more than 7° in rotation (39) (although a CT scan by Penning of normal subjects found only 1° of atlanto-occipital rotation (9)). Yet, with the atlas in presumptive subluxation, such magnitudes of misalignment are rarely measured. A study in the NUCCA Monograph (40) showed the average atlas *laterality* or upper angle in 869 patients was 2.6°, much less than the maximum of 5°- 8° that might be expected with *spasm* of the suboccipital muscles. In the same study, of 623 cases, the average atlas *rotation* was found to be 2.5°, again much less than the maximal 7°. If the pain-spasm-pain cycle is responsible for atlas misalignment/fixation, the observed magnitude of misalignment should be much greater.

Finally, one small (n=36) retrospective study found that the side of atlas laterality/misalignment (as determined by upper cervical x-ray analysis) after motor vehicle trauma is ipsilateral to the side of angular acceleration of the head (41). However, soft tissue injury, and thus nociception, is on the contralateral side, where the muscles, tendons, and ligaments have been damaged in an attempt to keep the massive head fixed on the cervical spine (42, 43). For example, if the force in a motor vehicle accident is from the right and anterior, hyperflexion injury to the neck muscles would be to the left lateral muscles/ligaments. Such injury with sufficient soft-tissue damage might be expected to initiate nociceptive input and reflexively increased output to (alpha) motoneurons and result in decreased segmental mobility and joint dysfunction (44). Because, in this example, the injury initiating the nociceptive input and pain-spasm-pain cycle is on the left side, muscle spasm and joint dysfunction/misalignment would be expected to take place on that side, producing a left laterality. Yet the results of the previously noted study have shown that, in the example, the atlas misalignment more often occurs to the right side, opposite the soft tissue damage.

Collectively, the review by Roland, the average amount of atlas misalignment and the study of atlas laterality and motor vehicle trauma cast some doubt on the hypothetical pain-spasm-pain cycle as the primary cause of atlas subluxation, and the source of the aberrant mechanoreceptor input resulting in LLI.

Atlas Misalignment and Fixation

Neither of the reviewed theories of atlas subluxation offers an explanation for relative fixation. Although relative fixation of the atlas in subluxation could be tested by x-ray and was demonstrated in one case study (45), such investigation using ionizing radiation on otherwise healthy people has been deemed unethical. Since the ligaments across the occipito-atlanto-axial complex are lax, movement of the atlas must be the result of muscular action (46). For relative fixation of the atlas in its normal range of motion to take place, muscles must be involved. How the relative fixation of the atlas occurs and is sustained is where this new theoretical model begins.

Bartol described the characteristics of diarthrodial joint motion in four stages (47). The initial motion is active range of motion, then passive motion to what Bartol calls the elastic barrier. Movement past this barrier occurs in what is called the paraphysiological space. Forced joint movement beyond the elastic barrier of resistance has been shown to cause plastic deformation (strain) of restraining capsular ligament(s) (48-51) and results in altered kinesthetic sensation (49-51). Such forced motion is also hypothesized to cause entrapment of anatomical structures inside the joint (52-54).

Entrapment of intra-articular inclusions has been proposed as a mechanism for the "acute locked back," a syndrome in which an intra-articular fibro-adipose meniscoid of a lumbar zygapophysial joint gets trapped outside the articular surfaces, deforming the joint capsule and causing pain and muscle spasm (52). An anatomical study by Mercer and Bogduk found similar intra-articular anatomy in the cervical spine, and the same entrapment mechanism has been proposed as a possible cause of acute neck pain and muscle spasm, including torticollis (53) and atlanto-axial rotatory fixation (54).

In the upper cervical spine, 93% of atlanto-occipital joints studied contained intra-articular fat pads that were described as

FIGURE 1
A Superior View of the Atlas Condyle Showing the Intrarticular Fat Pad

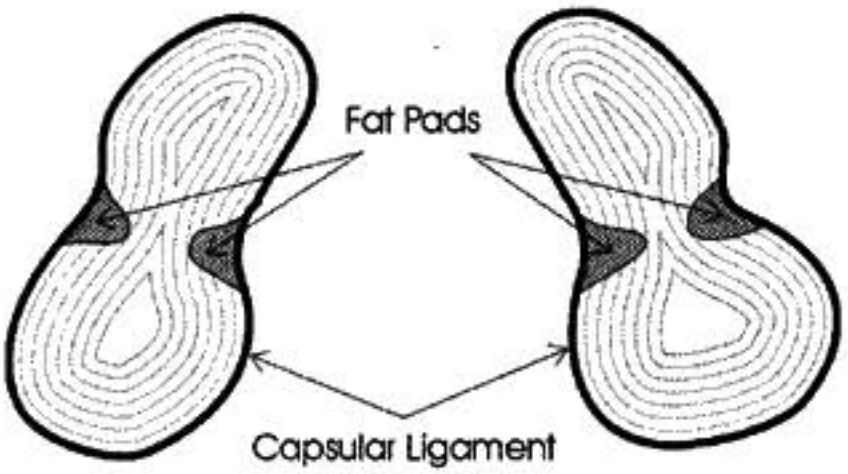
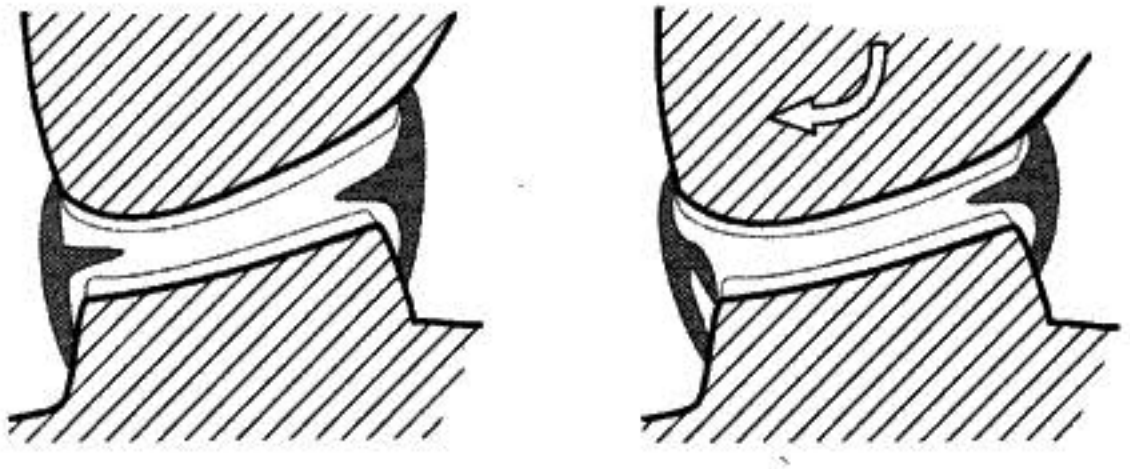


FIGURE 2
A Frontal Schematic View of the Atlanto-Occipital Joint Showing a Possible Fat Pad Entrapment



smooth and triangular in shape with wide bases attached to the joint capsule and free borders projecting toward the center of the joint space (53). The fat pads occupied the space bounded by the joint capsule but did not intervene between the articular cartilages (53). The authors believed the function of the fat pads is to fill the nonarticular portion of the joints, acting as displaceable space fillers when the joint moves, much as the fat pad of the knee joint (Figure 1).

In a case of upper cervical trauma resulting in joint movement beyond the elastic barrier of resistance, could the atlanto-occipital intra-articular fat pad become trapped, impinged between the articular cartilages? Is there a precedent for fat pad impingement in a synovial joint? What sort of symptoms would such an entrapment produce?

Hoffa first described impingement of the intra-articular fat pad in the knee (55-58), which now bears his name – Hoffa's disease. Usually caused by trauma (56, 57), the entrapment causes pain, characteristics of a sprain and slight flexion contraction of the hamstrings (55, 58). In the acute phase, the pain is caused by activation of the nociceptive fibers present in the fat pad; in the chronic phase, the main source of pain is the stretching of the joint capsule due to the impingement (55). Conservative treatment of the fat pad impingement involves avoiding weight bearing, applying ice, and occasionally immobilization (55). However, most patients require surgery to resect the fat pad (55-58).

Impingement of the intra-articular fat pad in Hoffa's disease causes pain (nociception), and guarding muscle tension and results in joint dysfunction. Hypothetical entrapment of the fat pad between the articular surfaces or between the joint surface and capsular ligament (extrapment) in the atlanto-occipital joint (Figure 2) is hypothesized to cause similar symptoms.

Wyke, as reported in a review by Slosberg (44), found that activation of nociceptors by irritating the joint capsule generated an intense, non-adapting muscle response to guard the joint. The musculoskeletal reflexes triggered by nocifensive input are termed "nocifensive reflexes" (59). The prototypical nocifensive reflex is the flexion reflex, in which stimulation of pain endings causes contraction, less than a spasm, of the flexor muscle and inhibition of the extensor muscle about the associated joint (12, 18). This reflex response has also been demonstrated in the muscles of the upper cervical spine (60). The nocifensive flexor reflex may be seen in Hoffa's disease as a slight flexion contracture of the hamstrings. Corroborating evidence for the reflexive muscular response to joint injury is found in a study that demonstrated the temporary restoration of joint mobility by intra-articular anesthetic injection (44).

Nociceptive discharges can also be augmented by joint inflammation, which causes normally silent nociceptors to display rest-

TABLE 2
Atlanto-Occipital Muscles from Gray's Anatomy (62)

Muscle	Origin/Insertion	Action
Rectus capitis lateralis	Arising from the upper surface of the transverse process of the atlas and inserts in to the inferior surface of the jugular process of the occipital bone.	Contraction would pull the atlas laterally, up the condyle in lateral flexsion.
Obliquus capitis superior	Arises from the upper surface of the transverse process, passing upward and backward, attaching to the occipital bone between the superior and inferior nuchal lines.	Contraction would pull the atlas posterior on that side.
Rectus capitis anterior	Arises from the anterior surface of the lateral mass of the atlas and the root of the transverse process, ascends to the inferior surface of the occipital bone anterior to the occipital condyle.	Contraction would rotate the atlas anterior on that side.

ing discharges of higher than normal frequency and decreases the threshold for activation of joint nociception by movement (60). Such inflammation elicits nociceptive responses and pain under what would normally be innocuous conditions (60). In addition to inflammation, the muscles guarding the pain-producing joint, subjected to continued contraction, begin to tire, generating products of fatigue which further activate nociceptors (44).

External application of analgesics, muscle relaxers, and physical therapeutics would not be able to break this feedback loop – unlike the pain-spasm-pain cycle – as long as the initiating nociceptive event, the entrapped atlanto-occipital intra-articular fat pad, remains.

If an atlanto-occipital intra-articular mechanism is responsible for the muscle splinting and relative fixation of the atlas, it might be expected that intra-articular injection of analgesics and/or anti-inflammatory drugs would break the reflex cycle. Such an atlanto-occipital intra-articular injection technique and three case reports were described in a paper by Dreyfuss (61). All the cases presented were chronic; one had right suboccipital, neck, and shoulder pain for over 12 years. All had undergone previous treatment with drugs, two had physical therapy and cervical manipulation, and one had chiropractic "manipulation." The C0/C1 intra-articular joint injections of anesthetic and steroid anti-inflammatory provided complete pain relief for six months to more than a year. In addition to the C0/C1 joint injection, all three patients were given stretching/mobilization exercises, and in one case, upper cervical joint manipulations were utilized. Dr. Dreyfuss found that after injection, attention needed to be placed on reestablishing articular range of motion and length and strength relationships in the soft tissue.

The results obtained by Dr. Dreyfuss provide evidence of an intra-articular C0/C1 problem causing joint fixation, nociception, and head/neck and shoulder pain. It is interesting to note that while the analgesics injected in the C0/C1 joint silenced the nociceptive afferentation, the patients also required joint manipulation/ROM treatment. This author speculates that it was necessary to manipulate the C0/C1 joint because the hypothetical causative event, intra-articular fat pad impingement/entrapment, was still present, something analgesic injection alone could not correct.

The muscles likely to be involved in reflexive nocifensive contraction and protection of an injured atlanto-occipital joint are the small intervertebral muscles that surround the joint; the rectus capitis lateralis, obliquus capitis superior, and rectus capi-

tis anterior (see Table 2). The actions of these atlanto-occipital muscles are presumed to be lateral flexion and rotation of the skull; yet, according to *Gray's Anatomy*, "The actions of the above deep-seated muscles can only be deduced, and their precise roles in everyday activities await adequate technical appraisal (62)." If the rectus capitis lateralis contracts to stabilize an atlanto-occipital joint in which the fat pad is entrapped creating nociceptive afferentation, shortening of the muscle would tend to move the atlas laterally, up the condyle. This, in chiropractic terminology, would appear to be atlas "laterality." In upper cervical specific chiropractic parlance, this muscular action would create an "upper angle." Contraction of the rectus capitis anterior, again to stabilize a nociceptive atlanto-occipital joint, would cause anterior rotation of the atlas; the obliquus capitis superior, posterior rotation of the atlas.

The position best minimizing nociception in the case of a nocifensive muscular reflex is not at the maximally shortened position for a muscle around its joint (13, 47); the guarding muscles are hypertonic but not spasmed. This fact dovetails nicely with the finding that the average magnitude of atlas misalignment is far less than expected with muscle spasm (40).

It is my hypothesis that contraction of the suboccipital muscles, singularly or in combination, causes the relative fixation and aberrant movement noted on palpation and the direction and degree of misalignment measurable on specific upper cervical x-ray (63-67).

Cause of Fat Pad Extrapment

Finally, although this hypothesis postulates trauma, movement of the atlanto-occipital joint beyond passive resistance, as an initiating event for atlas subluxation complex, subsequent subluxation often occurs without a traumatic event. Such a clinical observation and the general repeatability of the direction of atlas misalignment (barring new trauma) as noted on x-ray, may be explained by this intra-articular entrapment hypothesis.

Studies of the proprioceptive warning systems in the foot (49), shoulder (50), and knee (51) have shown that after capsuloligamentous injury to these joints, sensory feedback at the limits of joint motion is reduced. Such reduced proprioceptive warning is thought to disturb the complex feedback between muscles, joints, and the central nervous system (49) and "...may lead to biomechanically unsound positions being adopted (51)."

Transposing these findings to a C0/C1 joint where prior trauma has altered the joint's kinesthetic sensitivity, the patient may be able to force his or her head/neck into positions that would otherwise have been warned against and braced for. Thus, moving the C0/C1 joint into the paraphysiological space which results in atlas subluxation complex is more likely after injury than before it. This is what is often seen in specific upper cervical practice. If the neck is forced nontraumatically to the point of entrapment, resubluxation (relative fixation with neurologic manifestation) of the atlas often results. Because the initial injury occurred in a specific vector, the listing, or position of the atlas, when compared with the initial x-ray, is very much the same (68). Such a clinical finding allows the specific upper cervical doctor of chiropractic to adjust the atlas off an x-ray listing from weeks, months, or even years before.

The Fat Pad Impingement/Tonic Neck Reflex Hypothesis of Atlas Subluxation

Movement of the atlanto-occipital joint past the elastic barrier impinges the intra-articular fat pad. Nonfatiguing nociceptive impulses generated from the entrapment cause guarding contraction of the associated atlanto-occipital muscles. Atlas misalignment and relative fixation from the muscular contraction, consisting of laterality and rotation, is visible on x-ray. Contraction of the suboccipital muscles activates the tonic neck reflexes causing chronic tension in the extensor muscles and a pathologic asymmetric tonic neck reflex, or functional short leg The hypothesis is shown as a schematic algorithm in Figure 3.

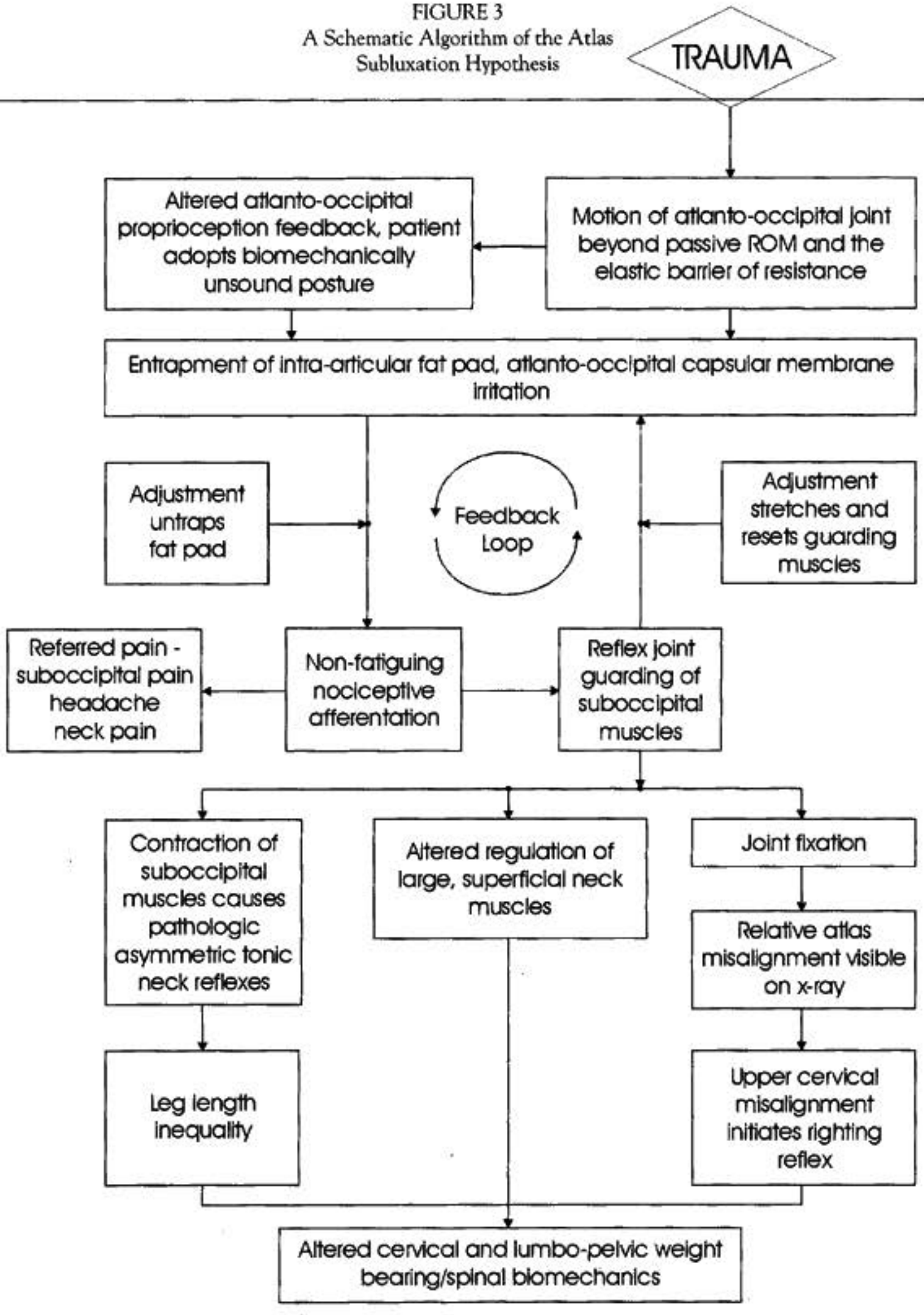
Effect of Adjustment

Application of high-velocity, low-amplitude chiropractic adjustment to a vertebral segment activates the muscle spindles (69), bypasses slower nociceptive reflexes that may block the vertebral movement (44) and forces the contracted muscle to lengthen, inhibiting the alpha motoneurons and resulting in a sudden relaxation of the muscle (70). The hypothesis that a rapid thrust is necessary for a successful manipulative treatment is given some credence in a small (n=20), matched, nonrandomized, controlled trial by Rogers (71). A group of patients with chronic neck pain, treated with high velocity, low amplitude manipulation, was shown to have decreased pain and an enhanced ability to position the head in comparison to a similar test group which was given a series of stretching exercises for the cervicothoracic muscles. The author speculated that the stretching exercises have the greatest effect on the large, superficial muscles, whereas, "a manipulation thrust in the plane of normal movement of a joint would presumably be in such a plane as to affect deep interarticular muscles."

In the theoretical entrapment mechanism outlined so far, such a high-speed chiropractic adjustment, in a vector opposing the reflexive muscular contraction, would preferentially stretch the small atlanto-occipital muscles that hypothetically create the relative joint fixation, causing them to relax. Relaxation of the suboccipital muscles eliminates the spindle stimulation responsible for the asymmetric pathologic tonic neck reflexes and the contractured or functional short leg. The rapid adjustment would also cause distraction of the atlanto-occipital joint, releasing the entrapped intra-articular fat pad and thus ending the nociceptive stimulation and reflexive muscular contraction.

Manipulation to break up an upper cervical fixation may or may not move the atlas to accomplish what the theoretical mechanism hypothesizes is necessary – distraction of the correct C0/C1 articulation, elimination of fat pad extrapment, and stretching of the specific contracted atlanto-occipital muscles. This is important to note because several other methods used to determine the degree and direction of atlas misalignment have not been shown to correlate with upper cervical x-ray findings (72). In addition, many techniques for atlas manipulation, particular-

FIGURE 3
A Schematic Algorithm of the Atlas Subluxation Hypothesis



ly rotary types, use the posterior rotated aspect of the atlas for contact and thrust. Because anterior rotation on the side of laterality is found in roughly 70% of patients (68), manipulation of this type may not be desirable.

Such concerns about the need for specificity in adjustment vector should be tested because manipulation of the highly mobile atlas in a direction other than the correct vector may not only fail to eliminate the joint fixation and LLI, but may also cause an iatrogenic reaction (73). If the proposed theory is correct, adjustment of the atlas requires specificity beyond joint mobilization, and given the potential for iatrogenesis, caution in nonspecific manipulation is recommended.

Clinical Correlations

A theoretical framework is useful if it can explain various clinical observations. With that in mind, we will review some common clinical observations juxtaposed against the entrapment, tonic neck reflex theory.

Side and Magnitude of LLI — Compare the effect of the tonic neck reflexes with clinical findings in chiropractic practice; LLI is generally contralateral to the side of atlas laterality, except when atlas rotation is large compared with laterality (74) and provocation tests, primarily rotation of the head, have been clinically found to augment the degree or change the side of LLI (1). Recent development and reliability testing of a "friction reduction" table by Cooperstein (3) has enabled the detection of changes in absolute and relative leg positions with rotation of the head (75), as predicted by the asymmetric tonic neck reflex.

Unlike most types of mechanoreceptors, a pathological asymmetric tonic neck reflex, defined as a reflex that does not attenuate, can persist as long as the stimulus, head rotation/lateral bending, is maintained (23, 25). This could explain the chiropractic clinical finding of longstanding LLI. As long as the perception of the body, via the small atlanto-occipital muscle spindles, is that the head is rotated/laterally flexed, the asymmetric tonic neck reflex is maintained, and a LLI could be demonstrated.

Furthermore, multiple attempts to elicit a postural check for asymmetric tonic neck reflex arouses resistance reactions in the subject, which break down the reflex changes in the muscle tone and makes them difficult to identify (25). This may account for the difficulty in inter-examiner testing of the LLI phenomenon. Multiple hands-on testing of a subject with asymmetric tonic neck reflex/LLI may cause changes in the muscle tone and temporary diminution or elimination of the LLI.

Tonic neck reflex inputs for both lateral bending and rotation of the head vary in their effects on LLI. Theoretically, an atlas misalignment which involves equal asymmetric tonic neck reflex inputs for lateral flexion and rotation could result in the clinician's not being able to detect LLI. Such detracting asymmetric tonic neck reflex inputs may also account for the clinical observation that there is rarely a correlation between the severity of atlas misalignment and the magnitude of LLI– an observation heretofore unexplained by other upper cervical subluxation mechanism hypotheses.

Torticollis and Shoulder Pain — Experiments in cats indicate that another function of the small suboccipital/intervertebral muscles is to act as receptors to modify the actions of larger, longer muscles which span multiple joints and act as prime movers and stabilizers of the head/neck (76). It could be speculated that contraction of the suboccipital muscles to guard an irritated atlanto-occipital joint might induce abnormal contraction of the sternocleidomastoid, causing torticollis, or the trapezius, causing neck and shoulder pain, and explain the efficacy of upper cervical adjustment as a treatment for these conditions (77-79).

Balance Disturbances — Two pilot studies using MRI have shown atrophy of the upper cervical extensor muscles, the rectus capitis posterior major and minor, in patients with chronic neck pain (81, 82). This replacement of muscle tissue with fat, called "neurogenic atrophy" (81), was correlated with balance problems and palpable somatic symptoms (putative vertebral subluxation) in the atlanto-occipital region (82). C0/C1 subluxation may therefore cause the somatic symptoms demonstrated by palpation, the atrophy of suboccipital muscles and the balance problems as a result of alterations in the tonic neck reflex.

Frequency of Anterior Atlas Rotation — Hypothetically, fat pad extrapment causes a nociceptive reflex, and the prototypical nocifensive reflex is a flexor reflex (12, 34, 61). Hence, the suboccipital muscle most likely involved would be the rectus capitis anterior, which, ostensibly, flexes the skull (62) or rotates the atlas anteriorly. As such, anterior atlas rotation would be expected to predominate as an x-ray finding over posterior rotation. This is indeed the case for 68% of atlas rotations seen on specific upper cervical x-ray (n=1,300) are anterior (on the side of laterality) (68). This reflex may also help explain why the study of atlas misalignment and the vector of angular acceleration of the head and neck in motor vehicle accident found significant numbers of anterior atlas rotation despite a posterior vector of acceleration (41).

Scoliosis — A final clinical correlation with the asymmetric tonic neck reflex, a reflex that increases extensor muscle tone, is the role it may play as a potential indicator in the early detection of scoliosis (24). A study found a relationship between asymmetric tonic neck reflex, scoliosis, and the side of the major spinal curve (59). Such a causative factor in idiopathic scoliosis may be behind dramatic reversals of scoliosis with specific upper cervical care noted in the literature (80).

Testing the Hypothesis

The studies of the tonic neck reflex and its potential sensory mechanism have been done primarily on the cat. The cat has suboccipital/atlas skeletal and muscular anatomy analogous to humans (60). As such, the cat would seem to be an ideal experimental animal to test the outlined entrapment/capsular irritation, pathologic asymmetric tonic neck reflex theory. Irritation of the cat C0/C1 joint capsule should result in an asymmetric tonic neck reflex detectable by electromyography as an excitatory or inhibitory reaction of the suboccipital muscles. An x-ray procedure similar to that done in specific upper cervical technique protocol might be used to determine if C0/C1 capsular irritation causes relative misalignment/fixation of the atlas vertebra.

In humans, there may be some ways to test the hypothesis that pathologic asymmetric tonic neck reflexes are responsible for the clinically noted phenomenon of functional leg length inequality. As pointed out earlier, the ephemeral nature of these muscle reflexes may have made it difficult to confirm the reliability of leg checks and provocation tests (25). However, the "friction reduction" table (3) may represent a gold standard (75) for such tests, allowing for clinical trials of these hypotheses.

Nevertheless, if such a method comes to pass, the following provocation tests, which would not be explainable by the other upper cervical theories proposed for leg length inequality, are suggested.

1) It has been hypothesized that lateral eye rotation can trigger the tonic neck reflex (83), and a mechanism for such a phenomenon is provided by Abrahams (32). It would be instructive to have patients showing LLI rotate their eyes while holding the head still and see if such an action causes changes in the leg length. A positive finding with such a provocation would tend to rule out other causes of LLI; rotating eyes cannot affect the dentate ligament nor aggravate upper cervical joint mechanoreceptors.

2) Stimulation of posterior cervical muscles by vibration has been shown to affect the complex body/head/eye orientation systems (84, 85), possibly including the tonic neck reflex. An experiment could subject patients to isolated suboccipital vibration and check LLI for changes. A positive finding on such provocation would tend to rule out the dentate ligament hypothesis and joint mechanoreceptor stimulation.

CONCLUSION

This is a speculative theory, not based on direct research, but instead, on a conglomeration of related scientific studies that seem to explain what is noted clinically. The danger of theoretical musing on the basis of selected studies that fit what are believed to be the "facts" is acknowledged. Yet there is an aesthetic in science that values parsimony, in which a theory explains more varied observations. I believe the intra-articular fat pad entrapment, pathologic asymmetric tonic neck reflex theory, as outlined in this study, does just that.

I fully expect and encourage thoughtful criticism of this atlas subluxation complex theory. Such criticism will strengthen this theoretical model or find weaknesses, leading to alternative explanations. Either way, our understanding of the phenomenon of the upper cervical subluxation complex and the neurologic manifestation of leg length inequality will likely increase. ◆

"Interference with muscles and receptors in the neck can cause profound postural disturbances."

V.J. Wilson in Control of Head Movement, Peterson & Richmond ed.

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