CASE STUDY

Meniere's Disease Symptomatology Resolution with Specific Upper Cervical Care

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ABSTRACT

Objective: To describe the outcomes of a patient with Meniere's disease undergoing upper cervical chiropractic care.

Clinical Features: A 52 year old male presented to the office with a previous diagnosis of Meniere's disease and associated symptoms of tinnitus, sensations of fullness in the left ear, vertigo, tension headaches, right mid-back and scapular pain along with other musculoskeletal complaints.

Interventions and Outcomes: The patient received orthogonal-based, specific upper cervical chiropractic care based on the National Upper Cervical Chiropractic Association (NUCCA) technique protocol. After the first adjustment, the patient noted a minor reduction in tinnitus. After 4 adjustments and 5 weeks of care his subjective feedback demonstrated an improvement of about 60% and a significant reduction of musculoskeletal complaints.

Conclusions: The positive response observed in this case suggests that tinnitus, vertigo, auricular pressure, migraine tension headaches and Meniere's may be linked to atlas subluxation. The need for further investigation of the connection between atlanto-occipital and atlanto-axial vertebral subluxations and cranial nerve dysfunction is indicated.

Key Indexing Terms: *Upper cervical, subluxation, orthogonal, NUCCA, Grostic, dentate, denticulate, vestibular, vestibulocochlear, cord distortion, chiropractic, Meniere's Disease.*

Meniere's disease causes bouts of vertigo or severe imbalance; usually unilateral hearing loss, or distorted hearing; buzzing or tinnitus in the ear; sometimes an associated sensation of fullness in the ear, aural pressure, possibly due to a build-up of fluid; and nausea.[1-3] A patient may have a couple or all of these symptoms with their presentation. Intermittent episodes last from minutes to hours and the intensity can be mild to debilitating.[2] Currently there is no known cure, but successful management strategies include conservative lifestyle changes, minor surgical intervention, intratympanic gentamicin therapy, endolymphatic sac surgery, dietary modification, anxiety control, diuretics, and steroids.[2-6] Detailed pathophysiology underlying the symptomatic presentation is still conjecture at best, but includes increased endolymphatic fluid impeding proper neural impulses from the saccule and utricle; dehydration of the endolymphatic fluid causing increased viscosity delaying signals from the hair cells of the inner ear for proprioception; and thickening of the surrounding temporal bone disrupting normal inner ear mechanics.[2,7,8] Meniere's

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diagnosis tends to follow the exclusion principle making the final diagnosis and research of the pathophysiology difficult.[6]

Annular prevalence of vestibular vertigo is very common and incidence is estimated to be between 0.5 and 7.5 of 1,000, often with the exact cause being left undiscovered in the medical community due to the difficulty of diagnosis.[9] Presentations of vertigo and dizziness are estimated to remain unexplained in 40-80% of the population often because the method of resolution follows the diagnostic approach for a syndrome versus the cause of individual and common symptoms.[3-6,10]

A functional connection between the vestibular and trigeminal systems has been established in experimentation where the trigeminal nerve is stimulated initiating a vestibular response observed objectively via nystagmus. Vertigo attacks are reported by patients with and without migraines and idiopathic vertigo is three times more prevalent in migraine patients, and migraines are an associated co-morbidity with Meniere's disease.[10,11]

Case Report

Patient History

The patient presented into the office for a chiropractic consultation with a previous diagnosis of Meniere's disease by his otorhinolarygologist two years previously. His symptoms include cyclical bouts of tinnitus, vertigo, aural pressure, and low frequency hearing loss for six months duration with associated tension headaches and musculoskeletal complaints. Case history revealed attempts to resolve symptoms with diuretics, low sodium diet, traditional chiropractic care and upper cervical chiropractic care.

His symptoms reduced mildly with the medication and dietary modification. He recalled losing consciousness at work due to dehydration which prompted him to seek an alternative approach through traditional chiropractic care with a full-spine adjusting approach. His vertigo symptoms were reduced more than with previous care, but he still had tinnitus, buzzing in his ears, associated sensations of fullness in his ears and much of the vertigo remained. He stated that after minimal change he decided to go to New York to receive care from a well-known doctor who utilizes the NUCCA technique.

He reported a significant reduction of all symptoms after the first adjustment with the NUCCA doctor in New York, and after a couple more adjustments says all symptoms of headaches, tinnitus, vertigo and fullness in his left ear had resolved. After returning home his symptoms stayed resolved for months until he was working under the dashboard of his truck with his neck turned in maximal rotation, lateral flexion and extension that he felt a strain in his upper cervical spine and his symptoms began to recur. After the return of these symptoms he consulted the author's office because the doctor practices NUCCA, the same technique utilized by the doctor in New York.

Physical Examination

His complete presentation consisted of the Meniere's symptoms, associated tension headaches, right sided midback pain, ankles that ache, bursitis in the right shoulder and occasional low back pain aggravated by prolonged standing.

The physical exam revealed blood pressure of 122/70 bilaterally; 2^+ deep tendon reflexes for the triceps, brachioradialis, biceps and patellar tendons; 1^+ deep tendon reflex for the Achilles tendon; static palpation revealing taut muscle fibers and vertebral malposition at C0, C1, C2 and C3 on the left, taut and tender muscle fibers with bony misalignments throughout the upper and middle back on the right, taut and tender muscle fibers with vertebral malposition and edema noted between L5, Sacrum and Ilium; postural distortions into the right frontal plane with a right head and neck lean, a left high shoulder and ilium, and a twisting of the right shoulder anteriorly into the transverse plane; a functional leg length inequality on the right due to pelvic obliquity of three eighths of an inch.

No orthopedic tests were required. The following diagnoses were made based upon the clinical exam findings and history: 780.4 Vertigo/Dizziness, 353.2 Cervical nerve root lesions, 353.3 Thoracic nerve root lesions, 739.3 Lumbar segmental dysfunction.

Diagnostic Imaging Analysis and Interpretation

X-ray analysis of the nasium, vertex, and neutral lateral cervical films demonstrated the atlas vertebra to have misaligned laterally and superiorly to the right into the right frontal plane by three and one half degrees, and to have rotated posteriorly on the right side in the transverse plane by five degrees. The odontoid of the second cervical vertebra had not shifted off the midline of atlas, however the spinous process of axis had deviated into the left transverse plane by two degrees. (Figures 1-3)

The atlas plane line drawn through the posterior inferior attachment points of the posterior arch as demonstrated on the nasium film was noted to be one quarter of an inch inferior to the true horizontal demonstrating an acute angle of 85 degrees between the plane of atlas and the mid-axial point of the neural canal from C2-C7, known as the lower angle. The whole lower angle had deviated left into the frontal plane and rotated left gradually until the top of the lower angle was rotated two degrees into the left transverse plane with the neck lean.

A head tilt into the right frontal plane was clearly demonstrated on the nasium film creating another acute angle on the right side between the midsagittal point of both the cranial vault and the foramen magnum and the atlas transverse plane. This functional malposition of the spine, the atlas and skull result in a kinking of the spinal cord classified as a type two misalignment.[12]

The contact point was the right transverse process of atlas and the correction vector is measured from that contact point to be superior one eighth of an inch and posterior 5 inches to precisely leverage the atlas back under the skull and draw the lower angle back under both the atlas and the skull orthogonally. Thus, removing all torsion and pulling forces from the spinal cord as the structures are put back into their

anatomically designed position in relation to one another.

The contact point was determined from the neutral lateral cervical and nasium films, as being located one quarter of an inch diagonally inferior and anterior of the bony apex of the mastoid process on the temporal bone. Also noted on the lateral cervical film was an inconsistency of George's line, slight retrolisthesis of C4 on C5, associated early arthrosis at the posterior junction and arthritic degenerative changes with bony proliferation at the anterior C5/C6 articulation indicating intervertebral osteochondrosis and anterior ligament laxity from a past damage or injury of the cervical spine. Also, the cervical spine was hypolordotic with anterior head translation of one and a half inches.

Intervention and Outcome

A vertebral subluxation is the misalignment of a vertebra, or vertebrae, capable of causing neurological detriment sufficient to result in harmful effects, while an adjustment is the reduction of vertebral misalignment factors to remove the neurological insult.[3,13,14] This specific misalignment is measured with the combination of three cervical radiographs to give a three dimensional understanding of the complex misaligned state and provide the opportunity to determine a vector of correction and precise patient placement to facilitate such a correction.[15] NUCCA protocol outlines highly detailed procedures for ensuring x-rays are obtained and analyzed in a consistent manner with high interexaminer reliability.[3,12,15,16]

Supine leg checks were used during the physical exam because a positive correlation between various leg length presentations measured at the heels and an upper cervical subluxation complex has been noted by comparing x-ray analysis, having the patient turn their head during the leg check altering the short leg, comparing postural distortions before and after the adjustment and comparing apparent leg length discrepancies before and after the adjustment.[12,15,17,21,22]

These checks are part of the pre-adjustment protocol to determine the presence of the subluxation and used as immediate objective outcome assessment after the adjustment. The foundational principle behind the leg check is based on the neurological insult resulting from the complex mechanical forces distorting various tracts in the spinal cord, thus altering both afferent and efferent activity.[3]

The patient was placed in a side-lying position with his right side up and the center of cranial support just below the skull center of gravity to allow the crown of the skull to tip back to the left during the correction. The doctor measured from the contact point, the right transverse process of atlas, superiorly one eighth of an inch and posteriorly five inches to place the episternal notch and body in precisely the right line of correction for the patient's particular misaligned state.

NUCCA adjustments are described as a low-force adjustment that is delivered to the patient with a depth of excursion between 1/16" to 1/8".[12,17] The first cervical vertebrae was leveraged by contacting the right transverse process with the right pisiform and utilizing the triceps pull with the exact amount of force, depth and acceleration to direct the force superiorly into the right occipital condyle joining the gravitational force of the skull being off center superiorly and ultimately redirecting the force circularly down into the contralateral lateral mass of atlas.[12,13] Atlas is being used as a class two lever in this scenario so the fulcrum point is the left axial superior articulating facet. The remnant force of the skull tipping combined with the effort applied by the left lateral mass of atlas on the fulcrum point exert an inferiorward force transmitted through the articular pillar of the cervical vertebrae.[12]

This force travelling down the articular pillar combined with the inferior torque, determined from x-ray analysis of the transverse plane rotation of C2 and the lower angle compared to that of C1, facilitates the movement of the cervical spine into the right frontal plane and the derotation of the neural canal back to an orthogonal position underneath atlas.[12] The resultant postural changes have the skull, atlas and cervical spine in an orthogonal position with no torque left in the neural canal, meninges, and spinal cord.

Post x-ray analysis revealed that subluxation reduction brought the atlas lateral slippage into the right frontal plane down from three and one half degrees to three quarters of a degree, the C2 neural canal was brought to neutral with no transverse plane deviation under atlas, the lower angle was reduced from five degrees left frontal plane deviation to 2 degrees, and the head tilt down to within one degree of neutral. (Figures 4-6)

Further reduction was made after the post-adjusment x-rays were taken and anaylzed, but more radiographs were not taken since positive reduction values were obtained after following the initial vector and analysis of the post x-rays indicated the need to follow the same vector for the remaining reduction. Incidentally, a lateral cervical x-ray taken during the one month re-examination demonstrated a reduction in the retrolisthesis of C4 on C5 by 80-90% and another x-ray taken during the one-year physical exam demonstrates complete reduction of the posterior slippage of C4 on C5. Subsequent films taken during the normal course of care show the correction of retrolisthesis to have remained.

Subjective feedback five weeks after starting care revealed 60% improvement after 4 adjustments and 5 visits. The patient noted a decrease in symptoms after the first adjustment which continued for the first couple of adjustments especially after he began to retain the adjustment he received.

After two months of care he began to hold his adjustment for extended periods and only experienced the Meniere's symptoms after specific events he noticed had caused him to lose the adjustment, such as traveling long distances, twisting his neck maximally in tight spaces and being knocked over in a sporting event.

Following such events he would require administration of another adjustment to bring him back to a level of being asymptomatic and maintaining that state. Since undergoing care he has become a powerful advocate of NUCCA chiropractic care for Meniere's patients.

Discussion

A survey of literature on the connection between chiropractic and Meniere's disease reveals a correlation between the resolution of vertigo, tinnitus, and hearing deficit with chiropractic care. Currently, the body of literature is composed of clinical case studies. Many of these patients have a previous diagnosis of Meniere's disease and some present into the office with symptoms matching the diagnosis but no formal diagnosis has been made.

The largest study on the connection between chiropractic and Meniere's disease was a case series which included 139 patients receiving Blair upper cervical care.[3] 136 of the patients reported significant improvement of their condition that resulted in ability to carry out normal activities of daily living following care. 130 of the 139 patients had a posterior-inferior atlas listing and an average of 15 years between the onset of their symptoms and a known mechanical injury to their neck. Of all the symptoms resolved, vertigo and associated nausea are the two symptoms lowered in a dramatic fashion.[3] A preliminary case study series was conducted with 10 patients and equivalent results.[6]

Another case study reports a 75 year old female with a longstanding history of vertigo, tinnitus, and hearing loss and a recent diagnosis of Meniere's Disease receiving three knee-chest upper cervical specific adjustments over 29 visits in 3 ½ months. At this point in her care, objective x-rays and audiometric studies demonstrated improved head carriage and hearing with subjective feedback demonstrating improved hearing, reduced vertigo, aural pressure, anxiety, insomnia and cessation of medication usage. Care continued and a second examination was conducted at 9 ½ months with continued improvement. The follow-up audiometric study demonstrated improved hearing by 35 db at low frequency bilaterally and a reduction of 35 db in the left ear at high frequency and 10 db in the right ear at high frequency.[18]

Positive results have also been noted with the full-spine techniques combined with muscle trigger point therapy, surface electromyography, medication, rehabilitation stretches and exercises. Results for these combinations of care were comparable to the upper cervical techniques, but required a higher frequency of care, more time per session and more intervention procedures to obtain similar outcomes.[19,20] More research has been conducted on the connection between chiropractic care and individual symptoms related to Meniere's disease such as vertigo, tinnitus or aural pressure alone.

The Dentate Ligament—Cord Distortion Hypothesis explains how the upper cervical subluxation can produce symptoms akin to direct cord involvement, mechanical cord irritation and vascular compromise of the cervical spinal cord.[22,23] The denticulate ligament is composed of tough fibrous sheets of pia mater mechanically connecting the outer dural sac with the posterolateral spinal cord.[22,24]

Denticulate attachment points are slightly posterior to the coronal plane of the spinal cord and unbalanced tension affects the tracts in a concentric manner with the greatest effects being immediately adjacent to the attachment.

Microcirculation is extremely important to the proper function of neural tissue, for example the CNS constitutes 2% of the body's mass and uses 15-40% of the body's glucose.[25] The direct mode of delivery to the spinal cord, brainstem and posterior fossa of the brain via micro and macrocirculatory systems are altered by uneven tension from the dentate ligaments and stretching of larger vascular structures with an upper cervical subluxation.[17,22] Mechanical distortion of axonal diameter is associated with altered signal transmission in addition to the microvascular changes and ischemia.[23,26]

Grostic notes cord distortion as a narrowing in the midsaggital plane and a widening of the cord from tension in the coronal plane. Cord distortion first alters the circulation of these delicate vessels causing ischemia, nitric oxide (NO) build-up, physical alterations in shape of the axons and synaptic clefts possibly altering anterograde, retrograde and synaptic activity.[22,27]

Microvascular changes and vasospasm of brainstem arteries, specifically in the vicinity of the medulla, have been implicated in ischemic activity sometimes related to a previous hemorrhage.[3,28] This longitudinal and transverse tension placed on the most caudal aspects of the brain stem disrupts the reticular formation with afferent, efferent and inhibitory functions between the cortex and soma.[14]

The major bulbospinal reflexes include the vestibulospinal reflexes and associated neural structures within the spinal cord.[29] These neural interactions are studied with galvanic stimulation demonstrating immediate effects on static and dynamic posture and more significant effects from upper motor neuron lesions, especially within stroke patients.[29-31]

Furthermore, age is a major complicating factor when determining the extent of spinal distortion before encountering detrimental effects to health and symptomatic expression, which is consistent with the typically later age of onset in Meniere's patients.[32]

Vestibular nuclei are located in the posterolateral medulla of the superficial alar plate between the sulcus limitans posteriorly and the tela choroidea anteriorly.[2,3,33] The Dentate Ligament Hypothesis would suggest the denticulate attachments from the foramen magnum just posterolateral to the coronal plane at the lateral most aspects of the fourth ventricle and the anterior alar plate are perpendicular to the cord being the most taut and strongest of the 21 dentate pairs in the spinal column.[22,24]

Thus, the vestibular nuclei are subject to axial and transverse forces because they are located in direct proximity to denticulate attachment sites and they are in the same plane in which the cord is stretched during distortion.[3] The occipito-atlanto-axial complex with its unique movement to allow coupled movements of flexion, extension, rotation and lateral flexion between three contiguous segments, together with the most taut dentate ligaments in the cervical spine, could possibly cause enough microcirculation ischemia and soft tissue distortion at the vestibular nuclei to alter their transmissions to the medial and lateral longitudinal fasciculi.

From the vestibular nuclei transmissions are dispersed to

control ocular movement in response to the body's position in space and to the sensory cortex for further processing to adapt properly.[2,3] With these signals being altered in any manner spontaneous bouts of vertigo with varying degrees of intensity and duration might be conceivable.

Specifically, the spinocerebellar tract is one of the most proximal tracts to the dentate attachment and is responsible for regulation of limb unconscious proprioception, movement and somatosensory mapping; modulating the descending motor systems in the brainstem and cortex; and uses feed-forward mechanisms to regulate movement.[2,25,33] Dorsal spinocerebellar tract involvement and lamination of the cord with lower extremity channels being most posterolateral are a fundamental premise on using supine leg check as a significant indicator for upper cervical misalignment in the NUCCA protocol.

Interactions between the vestibulospinal and spinocerebellar pathways are integral for the body to know where it is in space and for it to respond properly with postural and limb changes.[29,34] Alterations in the various afferents ultimately produce an incorrect basis for reaction and efferent stimulation of muscle tone, ocular reflexes, and many other mechanisms by which the body responds to the input received.[30,35] Spinocerebellar inhibition results in decreased dynamic stability, poor neuromuscular control, decreased dynamic posture and proprioception.[29,34]

Additionally the suboccipital muscles find their origin and insertion on these three contiguous segments and are highly innervated with a small ratio of nerves to muscle spindles. The main function of these tiny and highly innervated muscles is to provide proprioceptive information to the brain about the position of the upper neck and head.[36] With long-standing misalignments in several degrees of coupled motion, further incorrect proprioceptive information is sent to the somatosensory cortex as a complicating factor.[35,37,38]

A possible link between pia and dural mechanical stress and the initiation of inflammation via nitric oxide synthesis has been suggested and is in the preliminary stages of research. If legitimized, this connection could add further insight into the various complicating factors of dura mater, pia mater, and spinal cord mechanical stresses and the associated symptoms of upper cervical patients.[27] The occipitoatlantoaxial complex has a very unique structure and function. A misalignment into any plane creates an incongruity at both of these junctions, thus altering dura and pia mater tension, degrees of spinal cord distortion and microcirculation, and decreasing the efficiency of sensory and motor signals from the brain to the body.

The intimate relationship of the upper cervical spine and the nervous system produces direct postural changes both mechanically and neurologically, as demonstrated above.[19,39,40] Posture imbalances and supine leg length inequality are two principal factors for determining if a patient presents with neurological insult due to an upper cervical misalignment. Palmer provides a brief history of medical and chiropractic literature detailing the importance of postural changes for health and biomechanical outcomes as well assessment to ensure the proper reduction of a subluxation was accomplished during the adjustment procedure.[41]

A survey of medical literature found that postural corrections were correlated with building resistance to infection, curing visceral pathologies, decreased fatigue, decreased musculoskeletal complaints and many other health complaints. NUCCA specifically addresses postural distortions in the physical exam process, x-ray analysis, pre-adjustment, post-adjustment and at reexamination periods.[21]

Most postural corrections occur in the coronal plane with additional saggital plane corrections coming both from the adjustment and with addressing muscular imbalances and lifestyle changes later in the care plan. Prolonged abnormal mechanical and physiological postures in the cervical spine generate improper proprioceptive information for the brain due to receptors in the joint capsules, tendons, ligament, muscle bellies, and skin of the cervical spine.[37] These sources of input provide information for static and dynamic posture and balance, thus decreasing speed and accuracy of movement leading to decreased joint stability by the supporting structures and increasing the likelihood for injury. [37,40]

Conclusion

The positive response observed in this case suggests that tinnitus, vertigo, auricular pressure, migraine, tension headaches and Meniere's may be linked to occipitoatlantoaxial malposition creating subsequent neurological insult. Specifically, the outcomes in this case demonstrated a possible connection between Meniere's disease and upper cervical subluxation with a temporal relationship of both the administration of an adjustment and resolution of particular symptoms and the return of the previous condition with return of upper cervical subluxation complex.

More research and investigation of the connection between the atlanto-occipital and atlanto-axial vertebral subluxation connection with cranial nerve dysfunction, histopathology and cord distortion components of a subluxation, and the neural insult of subluxations is needed in order to be more conclusive on the exact relationship.

References

1. Thorp M, James A. Prosper Ménière. Lancet. 2005; 366: 2137–39.
2. Kandel E, Schwartz J, Jessell T. Principles of Neural Science. New York: McGraw-Hill; 2000.
3. Burcon M. Upper Cervical Specific Protocol and Results for 139 Patients with Medically Diagnosed Meniere's Disease. J Vert Sublux Res. 2010 Nov 14; 1-10.
4. Sajjadi H, Paparella M. Meniere's Disease. Lancet. 2008; 372: 406–14
5. Akagi H, Yuen K, Maeda Y, Fukushima K, Kariya S, Orita Y, et al. Meniere's disease in childhood. Int J Ped Otorhinolarygol. 2001; 61: 259–264
6. Burcon M. Upper Cervical Protocol to Reduce Vertebral Subluxation in Ten Subjects with Menieres: A Case Series. J Vert Sublux Res. 2008 June 14; 1-10
7. Kim S, Swanson T, Hussain N. Underground clinical vignettes: Neurology. Baltimore (MD): Lippincott Williams and Wilkins; 2007.

8. Horner K. Functional changes associated with experimentally induced endolymphatic hydrops. Hear Res. 1993; 68:1 -18
9. Monsell E, Balkany T, Gates G, Goldenberg R, Meyerhoff W, House J, et al. Committee on Hearing and Equilibrium guidelines for the diagnosis and evaluation of therapy in Meniere's disease. Otolaryngol Head Neck Surg. 1995 September. 113:3
10. Russell G, Abu-Arafeh I. Paroxysmal vertigo in children – an epidemiological study. Int J Ped Otorhinolarygol. 1999. 49:S105-S107
11. Marano E, Marcelli V, Stasio E, Bonuso S, Vacca G, Manganelli F, et al. Trigeminal Stimulation Elicits a Peripheral Vestibular Imbalance in Migraine Patients. Headache. 2005 April. 45:325-331.
12. ThomasM. NUCCA Protocols and Perspectives. 1st ed. Monroe (MI): National Upper Cervical Chiropractic Research Association; 2002.
13. Gregory R. Some Biomechanical Aspects of Chiropractic. The Upper Cervical Monograph. Monroe (MI): National Upper Cervical Chiropractic Association; 1987;4(4):8-9.
14. Seeman D. The Biomechanics and Neurological Aspects of the Atlas Subluxation Complex. The Upper Cervical Monograph. Monroe (MI): National Upper Cervical Chiropractic Association; 1977;2(2):1-3.
15. Goodman R, Mosby J. Cessation of a Seizure Disorder: Correction of the Atlas Subluxation Complex. Chiropr: 1990 Jul (6:2): 43-46.
16. Seeman D. A Reliability Study Using a Positive Nasium to Establish Laterality. The Upper Cervical Monograph. Monroe (MI): National Upper Cervical Chiropractic Association; 1994;5(4):7-8.
17. Bakris G, Dickholtz M Sr, Meyer P, Kravitz G, Avery E, Miller M, et al. Atlas vertebra realignment and achievement of arterial pressure goal in hypertensive patients: a pilot study. J Hum Hypertens. 2007: 1-6.
18. Kessinger R, Boneva D. Vertigo, Tinnitus, and Hearing Loss in the Geriatric Patient. JMPT. 2000 June;23(5)352-362.
19. Bracher E, Almeida C, Almeida R, Duprat A, Bracher C. A Combined Approach for the Treatment of Cervical Vertigo. JMPT. 2000 June;23(5)96-100.
20. Emary P. Chiropractic management of a 40-year-old female patient with Meniere disease. J Chiropr Med. 2010;9:22-27.
21. Seeman D. The Predominant Factor Theory Reexamined. The Upper Cervical Monograph. Monroe (MI): National Upper Cervical Chiropractic Association; 1987;4(4):1-5.
22. Rochester R. Neck pain and disability outcomes following chiropractic upper cervical care: a retrospective case series. J Can Chiropr Assoc. 2009; 53(3):173-185.
23. Grostic JD. Dentate Ligament – Cord Distortion Hypothesis. CRJ. 1988(1:1): 47-55
24. Thomas M, Wood J. Upper Cervical Adjustments May Improve Mental Function. J Manual Medicine. 1992;6:215-216.
25. Moore K, Dalley A. Clinically Oriented Anatomy. 5th ed. Philadelphia (PA). Lippincott Williams and Wilkins. 2006.
26. Guyton A, Hall J. Textbook of Medical Physiology. 11th ed. Philadelphia (PA). Elsvier Saunders. 2006.
27. Fehlings M, Tator C. The Relationships among the Severity of Spinal Cord Injury, Residual Neurological Function, Axon Counts, and Counts of Retrogradely Labeled Neurons after Experimental Spinal Cord Injury. Exp Neurol. 1995; 132:220-228.
28. Zinck T, Illum R, Jansen-Olesen I. Increased expression of endothelial and neuronal nitric oxide sythase in dura and pia mater after air stress. Cephalalgia. 2006; 26:14-25.
29. Cetas J, Lee D, Alkayed N, Wang R, Ililff J, Heinricher M. Brainstem control of cerebral blood flow and application to acute vasospasm following experimental subarachnoid hemorrhage. J Neurosci. 2009; 163:719-720.
30. Jankelowitz S, Colebatch J. Galvanic evoked vestibulosphinal and vestibulocollic reflexes in stroke. J Clin Neurophysiol. 2004; 115:1796-1801.
31. Parietti-Winkler C, Gauchard G, Simon C, Perrin P. Sensorimotor postural rearrangement after unilateral vestibular deafferentation in patients with acoustic neuroma. J Neurosci Res. 2006; 55:171-181.
32. Allum J, Bloem B, Carpenter M, Hulliger M, Hadders-Algra M. Proprioceptive control of posture: a review of new concepts. Gait Posture. 1998; 8:214-242.
33. Clarke E, Cheng S, Bilston L. The mechanical properties of neonatal rat spinal cord in vitro, and comparisons with adult. J Biomech. 2009; 42:1397-1402.
34. Fix J. Neuroanatomy. 4th ed. Baltimore (MD): Lippincott Williams and Wilkins. 2008.
35. Mohan G, Pal P, Sendhil K, Thennarasu K, Usha B. Quantitative evaluation of balance in patients with spinocerebellar ataxia type 1: A case control study. Parkinsonism Relat D. 2009; 15:435-439.
36. Masi A, Hannon J. Human resting muscle tone (HRMT): Narrative introduction and Modern concepts. J Bodywork Mov Ther. 2008; 12:320-332.
37. Malstrom E, Karlberg M, Halstrom E, Fransson P, Hansson G, Magnusson M. Influence of prolonged unilateral cervical muscle contraction on head repositioning – Decreased overshoot after a 5-min static muscle contraction task. Manual Ther. 2010; 15:229-234.
38. Strimpakos N. The assessment of the cervical spine. Part 1: Range of motion and proprioception. J Bodywork Mov Ther. 2011; 15:114-124.
39. Kelly D, Holt K. Resolution of Vertigo, Migraines and Neck Pain in a 12 Year Old Boy Receiving Chiropractic Care – A Case Study. JPMFH. 2010 September 29; 4:150-153.
40. Sholten J, Plotsky J. Our Energetic Relationship with Gravity: Success of the NUCCA intervention measured by postural balance. Canadian Chiropractor. 2007 February; 22-23.

41. Perez M, Field-Fote E. Impaired posture-dependent modulation of disynaptic reciprocal Ia inhibition in individuals with incomplete spinal cord injury. Neurosci Lett. 2003; 341:225-228.
42. Palmer J, Croswell K. A brief history of the Modus Operandi of measuring and correcting the atlas subluxation complex syndrome and the role of posture in the National Upper Cervical Chiropractic Association (NUCCA) standard of care. The Upper Cervical Monograph. 2008; 7(1):8-14.

Figures

Figure 1. Pre Nasium View

Figure 2. Pre Vertex View

Figure 4. Post Nasium View

Figure 5. Post Vertex View

Figure 3. Pre Lateral Cervical View

Figure 6. Post Lateral Cervical View

Post x-ray analysis revealed that subluxation reduction brought the atlas lateral slippage into the right frontal plane down from three and one half degrees to three quarters of a degree, the C2 neural canal was brought to neutral with no transverse plane deviation under atlas, the lower angle was reduced from five degrees left frontal plane deviation to 2 degrees, and the head tilt down to within one degree of neutral. (Figures 4-6)