PROPOSED MECHANISMS AND TREATMENT STRATEGIES FOR MOTION SICKNESS DISORDER: A CASE SERIES

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ABSTRACT

Objective: To present an overview of symptomatic motion sickness disorder and to propose allopathic and chiropractic approaches for treatment.

Clinical Features: Three representative cases are presented involving patients with motion sickness. All 3 patients had suffered from this condition throughout their lives. A discussion of the hypothesis of sensory conflict as a causative factor in cases of motion sickness is offered.

Intervention and Outcome: Following methods founded in applied kinesiology and using spinal and cranial manipulative treatment, the patients were able to travel long distances without nausea, sickness, or dizziness. The evaluation of these patients' responses to treatment was determined by the doctor's observation, the patients' subjective description of symptoms while riding in a motor vehicle, a visual analog scale for neck and associated pain, and applied kinesiology chiropractic physical assessment tools.

Conclusion: Further studies into chiropractic manipulative treatments for sensory conflict and proprioceptive dysfunctions associated with the problem of motion sickness are indicated. The hypothesis of sensory conflict as the cause of motion sickness should be explored more fully by other chiropractic physicians and researchers. (*J Chiropr Med* 2006;5:22–31)

Key Indexing Terms: Motion Sickness; Diagnostic Tests, Routine; Vestibular Function Tests; Cranial Nerve Injuries; Proprioception; Therapeutics; Chiropractic; Kinesiology, Applied

INTRODUCTION

Motion sickness is the umbrella term for the condition also known as airsickness, seasickness, carsick-

ness or space sickness.^{1–6} For about 90 million Americans, equilibrium and dizziness disorders in general cause more than a passing problem. More than 8 million people visit their doctors each year because occasional or chronic feelings of wooziness, spinning, lack of balance, and fainting seriously interfere with their ability to work or enjoy their leisure. Dizziness and other equilibrium disorders are some of the most common symptoms reported to physicians.^{7–8}

Almost everyone has experienced the disagreeable symptoms of motion sickness in a car, on a boat, sometimes on a train, or even at the movies. Motion sickness is thus connected to movement, whether visual or bodily. Astronauts are susceptible to it, and for many people the only relief is medication with occasional unpleasant side effects and sometimes no improvement.³

Non-prescription medications such as Dramamine, Bonine, and Marezine can prevent both vestibular and optokinetic motion sickness. Other drugs are available by prescription. Many people wear a scopolamine patch (*Transderm Scop*) prescribed by a physician when they go on a cruise.³ The medication is absorbed by the body through a patch on the skin that is placed behind the ear and effectively prevents motion sickness for up to 3 days. However, it may cause adverse side effects such as excessive drowsiness, dry mouth, and blurred vision. The patch has a latency of 6–8 hours, requiring patients to anticipate the need for it well in advance of beginning a trip. Occasionally wristbands and other devices are marketed as cures for motion sickness.

Although it is small comfort while one is actually suffering, motion sickness usually stops within 24 hours if the stimulation stops. But it might take a few days if the stimuli continue (such as on an ocean cruise), until the brain learns to adapt to the motion. Many of those who suffer from motion sickness will have symptoms for a few days or longer after the trip. This condition is called *mal*

débarquement syndrome, just as seasickness is sometimes referred to as *mal de mer* (American Academy of Otolaryngology www.entnet.org).⁴ Two of the patients in this report experienced this syndrome.

Some people are so vulnerable to motion sickness that they dread each extended car or bus ride and must avoid airplanes and boats. They appear to be especially sensitive to the stimulation that causes the dizziness, vertigo, nausea, vomiting, and generalized feelings of illness of motion sickness. Children over the age of 2 are more susceptible than adults, but most of them outgrow this vulnerability.

There is no firm agreement in the literature as to why some people are more susceptible to motion sickness than others.⁶⁻⁹ Some of the older theories regarding the cause of motion sickness were discarded in the early 1970s when a better explanation was advanced.^{1,4,5,6} This explanation is known as the sensory conflict or neural mismatch theory and describes motion sickness as an individual's response to discordant motion cues to which s/he is not adapted. However, researchers were unable to specifically pinpoint the dysfunctions in the tissues responsible for the sensory conflict in patients suffering from motion sickness. They were also unable to adequately treat the tissue problems producing the sensory conflict found. Motion sickness is now thought to occur when there is a discrepancy between the external stimuli gathered by the senses and the internal expectations associated with the body's actual state of movement.

Improper sensory stimulation due to muscle, joint, visual, and vestibular problems of a mechanical nature, as the factors that may produce the sensory conflict found in motion sickness disorders, has not been extensively explored in the literature.^{6,10} Abnormal afferent input into the central nervous system has been linked to balance problems as well as visual disturbances.¹¹⁻²⁵

Motion sickness may persist in mild form for months or years after unusual trauma involving the head and neck. Traumatic injuries to the head and neck have been linked as a risk factor for motion sickness in the literature.¹⁸⁻²⁵

Recent scientific experiments were conducted to replicate the setting in which some people experience motion sickness.⁹ Results suggest that those

people who develop motion sickness secrete increasingly greater amounts of the stress hormones epinephrine (adrenaline) and norepinephrine while their bodies are perceiving motion earlier than those who don't suffer from the syndrome. Vasopressin (also called antidiuretic hormone or ADH) will then rise to high levels in the blood of those vulnerable individuals. At that point, contractions in the smooth muscles of the stomach increase, causing stomach ache and nausea. In the experiments, those people without motion sickness did not have any of increased hormone levels or accelerated pulse rates within the stomach. There is some evidence that eating increased dietary salt before or during a trip, such as that found in salted crackers, may help protect against motion sickness.

Orthostatic hypotension (Ragland's sign) was found in all the cases described in this paper, possibly indicating the prevalence of abnormal adrenal hormone physiology in patients with motion sickness disorders. Ragland's sign is an abnormal drop in the systolic blood pressure when a patient rises from a supine to a standing position, and it is easily measured in the clinical setting. For normal sympathetic nervous system activity to raise blood pressure from the sitting to the standing position, it is necessary to have a proper release of norepinephrine.^{26,27}

One particular kind of motion sickness known as pure optokinetic motion sickness relies on visual input. This less appreciated type of sensory conflict or neural mismatch occurs when the eyes perceive motion that the body cannot register.⁶ The optokinetic system is a reflex that causes the eyes to move when the object moves. Thus many people suffer intense motion sickness if they are viewing a tilted room, or if the images on a panoramic screen keep moving to allow the viewer a simulated experience of being in a plane, roller coaster, space ship or fast-moving vehicle. People who visit a wide-screen theater to see a film that incorporates a helicopter flight (eg, IMAX or Cinerama) or go on a flight simulation ride at an amusement park well know the odd feelings of vertigo that occur when the camera moves at high speed over uneven terrain.⁶ Many patients, when asked, describe anxiety and discomfort while taking these rides.

Research does suggest a genetic predisposition to motion sickness, and it seems to be occasion-specific.^{9,17} However, because vestibular, ocular, and somatic problems producing sensory conflict

can be identified and corrected using manipulative therapy in patients with motion sickness, the question of whether this is primarily a genetic disorder should be investigated in patients who present with this problem. Other conditions that may produce motion sickness are circulatory problems to the head, acute allergies, and a number of neurological diseases such as multiple sclerosis, syphilis, tumors, and head injuries. Therefore it is naïve to think of motion sickness as a disorder with a single origin and thus a single cure.

CASE SERIES

The chiropractic care described below is based upon original work developed by Goodheart²⁸ and the International College of Applied Kinesiology (ICAK) from 1964–2005.

Case 1

A 66-year-old woman had low back pain and numbness in her left leg extending from her sacroiliac joint to her knee. During the examination, proprioceptive tests (described in the discussion) indicated disturbance in her equilibrium mechanisms. The patient also described motion sickness problems that had been present throughout her life, but because she did not think chiropractic care could help with this disorder, did not mention it earlier during the consultation.

When she was a young child her family would often drive through the Rocky Mountains and while in the car her father would smoke a cigar. She would always have to get out of the car to walk around in order to keep from vomiting. Airplanes did not cause her motion sickness, but being on a boat made her dramatically sick. She was able to drive her own car if she did not have to turn her head too much (as on a mountain road), but if she sat in the back seat or did not look straight ahead as a passenger, she had motion sickness causing dizziness, vomiting, and nausea. She felt quite disoriented in the car. It took her several hours to recover from an episode of motion sickness after a car ride. She mentioned that if she ate a small amount of food before she goes driving she did not feel quite as ill, but this did not resolve the problem.

Radiological evaluation revealed hyperlordosis of the cervical spine, with slight osteophytosis of the vertebral bodies. Oblique films of the cervical spine showed no encroachment of the intervertebral fo-

ramina. The 1st cervical vertebra was not orthogonal to the ground on the AP open mouth view. Hyperlordosis of the lower lumbar spine was present, with anterior slippage of the L-4 vertebra. There was no evidence of a fracture of the pars interarticularis. The iliac crests were unlevel on the AP lumbar view. Disc spaces were well maintained, though there was anterior wedging of the L5-S1 intervertebral disc.

On examination Ragland's sign was positive (orthostatic hypotension). Proprioceptive testing revealed a positive Freeman Wyke one-leg standing test and positive Hautant's test (described in discussion section). Hautant's test produced unsteadiness with right head rotation and cervical extension. Ocular lock testing was positive in the left lower quadrant of the cardinal fields of gaze. Positive challenge to the feet was present bilaterally. Challenge is an essential diagnostic procedure in applied kinesiology used to determine the body's ability to cope with external stimuli that can be physical, chemical, or mental. An example of a physical stimulus is pushing on an articulation and determining muscle strength change. After an external stimulus is applied, muscle-testing procedures are done to determine an improvement in or weakening of muscle strength as a result of the stimulus.

Upper cervical subluxations were found by challenge. The left piriformis, right pectoralis major (sternal division), deep neck flexors, anterior scalenes, and sternocleidomastoid muscles were inhibited during manual muscle testing.^{29–31} Cervical spine extension and flexion produced inhibition of the psoas muscles bilaterally. The gluteus maximus muscles were inhibited bilaterally also, and strengthened with therapy localization (TL) to the upper cervical spine. The left hamstring muscle was inhibited. The cervical extensor muscles were inhibited bilaterally. A category II pelvic fault was found by TL and challenge.

A left internal, right external frontal cranial fault correction strengthened the anterior scalenes bilaterally and removed the ocular lock finding. Rotation of the neck was limited to 60 degrees bilaterally on initial examination, and after these cranial corrections the neck rotated 90 degrees bilaterally with greater comfort to the patient. An upper cervical fixation correction strengthened the gluteus maximus muscles bilaterally and corrected the inhibition produced in the psoas muscles bilaterally with cervical spine flexion and extension. The category II

correction balanced a leg length inequality of $\frac{1}{4}$ inch and strengthened the left hamstring muscles. Trendelenburg's test was negative after this correction. Subluxations of the calcaneus and talus bones were corrected bilaterally in the feet and removed the shock absorber test to both feet. The shock absorber test is a method of placing a mechanical shock into an articulation to determine if there might be a subluxation present. Irritation of the already disturbed articulation(s) will produce muscle inhibition in a muscle that previously tested normotonic. It is primarily used as a screening technique. A left posterior sacral fixation was corrected. Freeman Wyke and Hautant's tests were then negative.

On the patient's next office visit 2 days later, her nausea and dizziness were improved. After the second visit she agreed to be the passenger in her own car while her daughter drove her around town. There was very little queasiness in her stomach or dizziness in her head. She returned to the office and was immediately tested. Cranial faults and muscle facilitation remained corrected except for the left posterior sacral fixation and the talus subluxation that recurred during her test drive. Ragland's sign was still positive, and adrenal nutritional support was given.

After 4 visits the patient's proprioceptive tests were negative and Ragland's sign was normal. For the following 16 months she was asymptomatic and enjoyed driving to see her grandchildren on the other side of the Rocky Mountains. This type of travel had been forbidding for her while she suffered from the motion sickness. She was even able to go on a trip to Europe as a passenger on an airplane, a bus, and her own car, and drove a total of 2,400 miles on this vacation. She never felt any motion sickness.

A sense of motion sickness returned sixteen months after the initial corrections. Following a cranial fault correction to the temporal bones bilaterally, a lower and upper cervical subluxation correction, correction of inhibited upper trapezius muscles bilaterally using neurolymphatic reflex treatment,¹¹ and an upper thoracic fixation correction, the patient was once again asymptomatic and has remained so.

Case 2

A 45-year-old woman was seen for cervicogenic dizziness problems and motion sickness disorder.

This patient frequently vomited in her car and was always nauseous. She carried "sick bags" in her car. Her husband was her driver throughout their married life. She would be "walking in a haze" some days after driving. She had only flown on an airplane 2 times in her life and vomiting and nausea were present on both trips. She had experienced frequent dizziness for several years. When she turned her head to the left more than 30 degrees or turned her eyes to the left more than 30 degrees she felt the dizziness immediately. Her legs 'fell asleep' below the knees and she had daily headaches. She had experienced low back pain since age 19 when she fell off of a mechanical bull. In 1996 she had to crawl for 4 months due to her low back pain.

On examination Ragland's sign was positive. Hautant's test was positive with cervical extension and with left head rotation. The Freeman Wyke one-leg standing test was imbalanced on the left foot. TL to the neck improved her balance during the test. The ocular lock test was positive in the left upper quadrant. As the patient followed the author's finger through that quadrant, she saw two fingers in front of her when in actuality there was only one. Saccadic motion of the patient's eyes was observed as the eyes moved through that quadrant. An upper cervical subluxation was found by challenge. The psoas and gluteus maximus muscles were bilaterally inhibited on manual muscle testing (a common inhibition pattern found with atlanto-occipital joint problems on examination). The left sternocleidomastoid, gracilis, teres minor, and rectus femoris muscles were also inhibited on testing. Sagittal opening of the jaw produced a slight crepitus in the left TMJ and global muscle weakness on testing. A category II sacroiliac subluxation was found by TL and challenge.

The subluxations, muscle weaknesses, and cranial faults were all corrected on her first visit using applied kinesiology chiropractic methods. During correction of her TMJ problem, she noticed that her nasal passage on the left opened for the first time in years. An anterior 5th cervical was corrected on the left side of her neck and Hautant's test was negative after this procedure. The Freeman Wyke one-leg standing test was also negative. A nutritional supplement to support the adrenal glands was given to the patient. When this material was in the patient's mouth, several muscles that were inhibited on manual muscle testing were strengthened (the gracilis and teres minor muscles).

After this patient's second visit there was an improvement in all of her pain patterns (from a 10 to a 5 on the visual analog scale). The orthostatic hypotension was now approaching normal on testing; however, she still suffered from some dizziness and the motion sickness remained. On her third examination, deviation of her eyes to the left still caused global muscle weaknesses. A frontal cranial fault and a counter-rotational cranial fault of the temporal bones were corrected. After this correction, and for the first time in years, the patient was able to look to her left without experiencing immediate dizziness. After the third visit she felt confident that her motion sickness problem was improved. The cranial corrections had to be repeated on three subsequent visits, but each time she returned to the office she had less and less dizziness, the motion sickness symptoms were no longer present, and her "brain fog" was disappearing. She was able to drive herself to the office for her fifth visit and continued to do so ever since.

Over the next 6 months her energy levels improved, the ocular lock and cranial tests remain negative, headaches and sinusitis were corrected, her low back pain (a 9 when she first started care) remained at 2 on the visual analog scale.

Case 3

This 9-year-old girl had problems with movement, clumsiness, and reading. She was forced to read slowly, assisted by following text with her finger. She tripped and fell constantly and bumped into things. She had a fear of heights and of walking along walls or curbs and avoided physical competition and running. Her problem with reading was the primary reason she was brought to the office, but during the initial consultation it was also found that she suffered from motion sickness while being driven in a car. The child had a difficult birth process due to delayed cervical dilation by her mother. She had had 18 sinus infections in her life; antibiotics were used throughout her life. She did not go through a crawling stage during her development; she went immediately from scuttling on the floor to walking.

On examination, the Freeman Wyke test was positive on the right foot. TL to the cervical spine improved balance on her right foot. She wobbled on her feet as she stood during this test. During the consultation she sat on her own foot; she touched

and leaned into things around her constantly for support. Her near pointing was inaccurate with her left hand. Hautant's test showed deviation of the arms with all movements of the neck. Ocular lock was positive in both upper quadrants. She was mixed dominant: her left ear, right eye, right hand, and left leg were dominant. There was inhibition of indicator muscles globally while she was reading. Problems in the feet, pelvis, spine, jaw, and cranium were found using applied kinesiology diagnostic methods and corrected. After treatment, Hautant's test and the Freeman Wyke test were corrected.

On follow-up examination, the mother reported that her child seemed to be moving better and more confidently. Her habit of reading with her finger along the line of text had ceased. The patient was treated 4 times before all proprioceptive tests remained negative. After her fourth visit the child could be driven around town comfortably without any symptoms of motion sickness. She progressed over a year from being a very average student to being one of the best in her class. Her clumsiness problem also improved and she passed her physical education class and her teacher commented that she was doing much better in her athletic activities and liked to run and compete like the other children in class. She was driven to California twice in a period of 8 months and had no nausea, dizziness, or discomfort.

DISCUSSION

The precise mechanisms of motion sickness are still not adequately determined in the literature.^{6-9,17}

One interesting hypothesis is called sensory conflict.^{2-6,8-10,17} I propose that this hypothesis helps to explain the positive effects of chiropractic treatment upon the motion sickness disorders in the cases described here. Diagnosis and treatment of these mechanisms using chiropractic methods to reduce the sensory conflict producing motion sickness is described. The sensory conflict hypothesis explains motion sickness as a conflict between the sensory messages supplied by the visual, vestibular, and somatosensory (proprioceptive) sensors related to movements of the head. With sensory conflict, stimulation from these receptors by the body's movements sends discordant information to the integrative vestibular and cerebellar nuclei. More generally, this symptom is produced by an incongruity

between the messages relayed by the sensors of orientation and those of movement (Fig 1).

The sense of balance is maintained by a complex interaction of the following parts of the nervous system: 1) muscle and joint sensory receptors, 2) the inner ears, 3) the eyes, 4) skin pressure receptors, and 5) the central nervous system. The muscle and joint sensory receptors tell what parts of the body are moving. The inner ear (labyrinths) monitors the directions of motion, such as turning, forward-backward, side-to-side, and up-and-down. The eyes monitor where the body is in space (ie, upside down, right side up) and also directions of motion. Skin pressure receptors (such as those in the feet or the buttocks) tell what part of the body is down and touching the ground. The central nervous system processes all of the information from the 4 other systems to coordinate these sensory experiences.

The visual righting, labyrinthine, and head-on-neck reflexes are responsible for evaluating and main-

taining the body's orientation in space.¹² In 1948, Spector¹³ described the mechanisms of multi-sensory integration as the underlying cause of some types of vertigo and nausea, but he provided no therapeutic approach for correction. If all 3 reflexes are not providing the same information about the body's orientation, this neurologic disorganization produces the misdirected effort known as dysponesia.¹⁴ This can happen when 1 reflex indicates that the head is level and another that it is tilted. It also occurs when the body detects motion that the eyes cannot detect.

If muscles, joints, and the vestibular or visual systems are signaling improperly, they send incorrect messages through the afferent sensory nerves into the spinal cord and cerebellum. When the CNS responds to this aberrant afferent information, the motor response and signals will be incorrect and produce improper motor movement, tension, timing, posture, and autonomic response.¹⁵ As a result, some muscles will be hypertonic and others will be hypotonic during orthodox manual muscle testing.²⁹⁻³¹ This causes structural misalignment of the body, sensory disturbances, uncoordinated movement, and pain.

Central to the concept of applied kinesiology chiropractic evaluation and treatment is the consideration that the senses of seeing, hearing, smelling, tasting, feeling, and balance are not simple, specific sensations; rather they are sensory systems closely interrelated among themselves and intimately linked with motor functions. Manual muscle testing is the method of testing functional neurology in applied kinesiology. Normally there is predictable facilitation and inhibition of muscle function. When there is not, applied kinesiology testing methods are employed to discover and correct the factors responsible for the disturbance in predictable muscle function. The method used in these cases of motion sickness to diagnose where the sensory conflict arose is described below.

Proprioceptors in Muscles

Proprioceptive testing, although not a routine part of a chiropractic examination, should be performed on any patient who complains of equilibrium disturbances, vertigo, dizziness, or nausea with movement.

Goodheart introduced the Freeman-Wyke one-leg standing test into applied kinesiology in 1989.^{16,32}

Figure 1. Theoretical model: The sensory input from the upper cervical spine and the sternocleidomastoid muscles are not congruent with those coming from the eyes and inner ears. The goal in therapy is to eliminate sensory conflict between the proprioceptors in the muscles and skin, the mechanoreceptors in the joints, the vestibular receptors in the inner ears, and the visual righting reflexes in the eyes. Image courtesy of David S. Walther, DC, with permission.

The Freeman-Wyke one-leg standing test has the patient stand on one foot in front of the doctor, find his balance if he can, and then close the eyes. If the patient loses balance, the test is positive. The Freeman-Wyke one-leg standing test requires the integrated function of various proprioceptors all over the body. Upon asking the patient to therapy localize to the area causing postural imbalance during the test, he may immediately improve his balance. TL to the upper cervical spine helped all 3 cases presented here to regain their balance while standing on 1 foot with the eyes closed.

TL is a diagnostic procedure in applied kinesiology that consists of placing the patient's hand over areas of suspected involvement, then using muscle testing procedures to determine any change in strength or improvement in function. Placing the patient's hand on different locations stimulates nerve endings in joints and muscle, skin, ligament or tendon. Therapy localization is strictly a diagnostic tool in applied kinesiology that is to be combined with the other diagnostic findings to arrive at a final conclusion.

Hautant's test has the patient seated during the test, so that she feels safe even if dizzy. The patient is seated and the eyes are open, with both arms stretched out in front of the patient. The patient should point the fingers at the doctor's thumbs. The patient then closes the eyes in the neutral position while the doctor watches the hands and trunk for a few seconds to see whether the patient's hands deviate to 1 side in relation to his own fingers. After examination in the neutral position, the test is repeated in different head positions, ie, rotated right, rotated left, extended back, flexed forward. While the patient changes position the doctor holds the patient's hands in the neutral position to prevent deviation of her fingers due to synkinesis of the arms. Synkinesis is an unintentional movement accompanying an intentional movement. Drifting of the arms, deviation of the hands, vertigo, and blurred vision are positive findings. Certain positions of the head will create deviation of the hands that are pointing at the doctor's thumbs, and certain positions will abolish the deviation. In this test any deviation that takes place when the patient turns her head is the result of the head position relative to the trunk. All 3 cases described here had a positive Hautant's test. Cronin¹⁸ showed that if one is able to reproduce the patient's dizziness or postural embarrassment on testing active cervical spine movements, a mechanical disorder is indicated.

Mechanoreceptors in Joints

The head-on-neck reflexes come from the equilibrium proprioceptors located in the upper cervical ligaments and can be disturbed by subluxations or fixations of these vertebrae after trauma.^{19-25,33} The methods used in applied kinesiology to discover where this type of dysfunction exists are challenge, TL, and manual muscle testing during activity that produces signaling into the CNS.^{28,31,33}

The Vestibular Mechanisms in the Inner Ears

If proprioceptive testing (Hautant's, Freeman-Wyke, ocular lock, and others³⁴) remains positive after clearing the cervical spine of subluxations and soft tissue problems, then a disturbance to the labyrinthine or visual righting reflexes should be suspected, leading to cranial evaluation and treatment of the patient. It is not only aberrant afferent stimuli from the joints and muscles in the cervical spine that produces disturbances in equilibrium; proprioceptive disturbances can also come from intracranial structures, including the labyrinth of the inner ear inside the petrous portion of the temporal bone, and the temporomandibular joint itself.³⁵⁻³⁷ The labyrinthine and visual righting reflexes may be disturbed by joint or muscle problems in the neck and by cranial faults that may result from either whiplash dynamics or a blow to the head during an automobile accident or other head and neck trauma.^{24,25,38-42}

The forces of trauma affect the membranous labyrinth within the temporal bones of the skull. Acceleration and deceleration forces following collision activate in a violent and intense way the ciliated cells of the semicircular canals and the matrices of the otolithic organs.^{13,17,21} The forces of acceleration and deceleration can deprogram the sensory cells of the membranous labyrinth within the petrous portions of the temporal bones. When the trauma is violent, some ciliated cells may be partially destroyed. Anti-gravitational muscle function, muscle tone, and posture are not properly maintained. Consequently, the vestibular nuclei do not receive proper sensory input for decoding and distribution to the body, particularly in regard to cerebellar control. The cerebellum normally integrates labyrinthine, visual, and kinesthetic information that is used to provide motor output for correct balance.¹⁷ In post-traumatic syndromes, some of the sensory inputs no longer yield proper responses.⁴³

Temporal bone cranial faults can result in imbalance in one or both sternocleidomastoid and upper trapezius muscles due to mechanical irritation of cranial nerve XI as it exits the skull through the jugular foramina, and could thereby disturb the biomechanics of the joints in the neck. This phenomenon may be found regularly in the clinical setting, and inhibition of the upper trapezius or sternocleidomastoid muscles was found in the 3 patients described here.

Cranial bone dysfunction and manipulative strategies and techniques are used in several different chiropractic techniques.⁴⁴⁻⁴⁶ The diagnostic method used to discover cranial faults in applied kinesiology is termed a "cranial challenge," and has been described in the literature.⁴² With the temporal bone cranial faults, an obliquity of the reciprocal tension membranes (RTM) may result.^{38,39,44-46,47} The RTM consists of the periosteal attachments of the meningeal dural membrane of the falx cerebri and cerebelli, tentorium cerebelli, and the diaphragma sellae. The lateral attachments of the tentorium cerebelli on the internal aspect of the skull can be modified by temporal bone cranial faults. In the clinic the temporal bones are frequently found in counter-rotation, producing discordant integration once again from the vestibular sensory inputs coming from each side of the head.^{44,48,49} The tissue and fluid tensions inside the vestibular mechanism

within the petrous portion of the temporal bone, as well as its geometrical inclination on either side of the head, can therefore be changed by cranial faults like these.

Anatomical distortions of the geometry of the vestibular mechanism within the temporal bones (producing a tilt in the angular geometry of the semicircular canals) may create discordant sensory input into the CNS compared to that coming into it from the spinal joints and muscles, thereby creating sensory conflict and producing poor stability and deficient motor activity. This lays the groundwork for motion sickness as these physical faults increase the sensory conflicts already present in the patient when he is moving in cars, trains, planes, and boats. Thus the examination and treatment of the cranial mechanism, along with other chiropractic procedures that improve proprioceptive signaling throughout the body, assume a new significance and even greater importance than was formerly appreciated (Fig 2).

Visual Righting Reflexes

In applied kinesiology chiropractic methodology, a means for testing the integration of the muscles in the body with the visual reflexes has been termed ocular lock.^{28,44} It demonstrates the failure of the

Figure 2. It is hypothesized that temporal bone cranial faults may change the angulation of the semicircular canals and the movement of fluids through the vestibular mechanism. They may also change the shape of and the tension within the jugular foramina, as well the elasticity of the tentorium cerebelli, and increase tension on the cranial membranes throughout the skull. Under these conditions, it is proposed that the cranial nerves are subject to adverse mechanical influences of various kinds, including compression, stretch, angulation, torsion, and edema.^{38,39,46,50}

eyes to work together on a binocular basis through the cardinal fields of gaze. This is usually not gross pathology of cranial nerves III, IV, and VI; rather it is poor functional organization. Mechanical irritation of cranial nerves III, IV, or VI (usually VI) may be responsible for disturbed binocular function leading to discordant sensory inputs from the visual righting reflex.^{38,39,45,46,49-51}

When the eyes are turned in a specific direction, a previously strong indicator muscle will weaken when the ocular lock test is positive, and there is probably disturbance in the visual righting, vestibulo-ocular, or opto-kinetic reflexes. This is observed in the examination by simply having the patient visually track the examiner's finger around a circle. An eye saccade, or jerky movement, indicates poor binocular activity.

The eyes must work together as a team so that each eye is directed to the same fixation point on a page, somewhat like two spotlights highlighting a dancer at the center of the stage. This is called convergence. The focus of the eyes must also be quickly adjusted from one distance or angle to another, called accommodation. The eyes must be able to track or scan along the horizon or a line of print smoothly and evenly so that the brain can receive a flow of sequentially correct visual information. This is called tracking. Eye motion and position are integrated with proprioceptors throughout the body, as well as those of the vestibular apparatus and head-on-neck reflexes.⁵²

The ocular lock phenomenon is theorized to be a consequence, most frequently, to cranial faults. There has been some substantiation for this premise, which demonstrates the possible effects of dural tension on the cranial nerves.^{45,46,49-51,53} The cranial nerves carry dural sleeves with them for some distance; therefore, any abnormal meningeal tension may be transmitted to a nerve and affect its function. Proper cranial correction will often eliminate the positive test and improve binocular activity.^{17,38,39,42,44-46,47,49,51} In the 3 cases described here, each patient had a positive ocular lock finding, indicating that the visual righting, vestibulo-ocular, and opto-kinetic reflexes may have been disturbed.

Constructing a coherent perception of the relationship between the body and its environment, or the body in a car or other vehicle moving at high velocity, depends on parallel mechanisms that combine

sensory cues and signals connected to action. So it is not surprising that deficits in certain circuits or sensory pathways induce a breakup of this coherence. Because of the importance of accurate sensory input to the overall function of the human organism, direct clinical measurement of this factor in a patient's functional ensemble is desirable. However, many of the clinical assessments used for proprioceptive diagnosis do not enable the physician to differentiate between the muscular, mechanoreceptor, vestibular, or visual contributions to postural control and motor performance. The tests described in this case series have proven to be clinically adequate for this kind of differential diagnosis.

Currently, no traditional chiropractic examination method and treatment strategy offer demonstrated guidance in treatment decisions for patients with motion sickness disorder. The information presented in this article suggests that chiropractic treatment and motion sickness disorder are associated via sensory conflict. It is the author's hope that proposing this relationship will lead to future investigation by a variety of researchers to further document the relationship between vertebral subluxations, cranial faults, abnormal muscle tone, equilibrium disturbances, sensory conflict, and motion sickness.

Specific testing of the muscular, joint, visual, vestibular, and cerebrospinal (cranial nerve) systems has shown in certain cases to adequately resolve the involved areas of sensory conflict that existed in these patients' functional ensemble. Further research into which of these separate systems is the critical factor in successful treatment, or whether all of these mechanisms require treatment, is needed.

CONCLUSIONS

Corrections specifically planned, using applied kinesiologic chiropractic differential diagnostic methods to improve sensory input and elicit proper motor output, brought about more normal function for the 3 patients with motion sickness disorder described in this study.

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