CASE STUDY

Resolution of Psychogenic Non-epileptic Seizures Following Chiropractic Care: A Case Study & Review of the Literature

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ABSTRACT

Objective: The purpose of this study is to describe the outcomes following chiropractic care by a woman suffering from chronic psychogenic nonepileptic seizures (PNES) and to review the related literature.

Clinical Features: A patient who was previously diagnosed with PNES experienced tremors, loss of motor control while staying conscious, dizziness, vertigo, fatigue and body convulsions resulting in migraines. The patient was examined and found to have upper cervical vertebral subluxation and related postural and structural distortions.

Intervention and Outcomes: QSM[3] protocol was used to obtain measurements from the low hip to the skull by analyzing x-rays, digital posture (posture IQ), and low shoulder, neck and head tilt. After care, the post x-ray revealed the release of compression and twist of the spine by placing the atlas in a balanced orthogonal position. The posture IQ revealed an even distribution of weight with a decrease in shoulder rotation. At the completion of five months of management, the patient was been symptom-free.

Conclusions: This case study demonstrated a significant reduction in symptoms of a female patient with PNES disorder after receiving QSM[3] care. This case suggests the importance of conducting more research to study the effects of tonal-based chiropractic techniques on seizure disorders.

Key Words: *Psychogenic nonepileptic seizures, conversion disorder, chiropractic, adjustment, vertebral subluxation, cervical, tonal-based, Quantum Spinal Mechanics[3], biotensegrity*

Introduction

Psychogenic conditions are among the least favorable areas to treat in health care. It is often seen that people who present with a "malingering" case are turned down by those they seek out for treatment. In a clinical setting as much as, 10% of cases are treated for psychological conditions. Frustration exists due to the difficulty in properly diagnosing and treating disorders with "nonorganic" origin.[1]

Psychogenic nonepileptic seizures (PNES) are among the "somatoform disorders" that tend to be neglected and misdiagnosed. For this reason, reliable information pertaining to the incidence or prevalence of PNES in the general population has not been reported. However, patients referred to neurological centers for diagnosis have reported an "incidence of 1.5 per 100,000 per year (equivalent to about

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4% that of epilepsy), or 3 per 100,000 per year" with an estimated prevalence of 2-33 per 100,000."[1, 2] PNES is found to be almost as common as multiple sclerosis or trigeminal neuralgia and are seen commonly in certain clinical settings.[1] For instance, "about 20% of patients referred for epilepsy surgery evaluation, and up to 50% of patients with refractory 'status' have PNES rather than refractory epilepsy. Prolonged PNES treated as status epilepticus (pseudostatus, PNES status) occur in about one-third of PNES patients, and more than one-quarter of patients receive intensive care treatment for presumed status epilepticus at least once."[2] These "pseudoseizures" typically arise between 20 and 30 years of age, and rarely seen in children as young as 4 or in patients over the age of 70. Primarily, three-quarters of all patients are females; especially those previously found that were associated with an emotionally disturbing event.[2,3,4]

PNES is a disorder that is defined as having uncontrollable alterations in behavior, motor control, or sensations. This condition is not derived from a neurological origin or show signs of abnormal cortical firing.[5] Instead, the seizures are seen commonly linked to a psychological event that causes emotional distress, altering the body's ability to function properly.[6] Before the 1800s, PNES was acknowledged as a form of hysteria and then later classified by Charcot as "hysteroepilepsy" and "epileptiform hysteria." As further understanding and development evolved, the diagnosis of PNES became the acceptable norm, since insanity was not the only consideration that was a factor.[5]

Given that mental processes leading to seizures remain unknown, there is a lack of consensus in the diagnostic categorization of PNES. The DSM-IV categorizes PNES under the heading of functional neurological disorder/conversion disorder, whereas the ICD-10 places them under dissociative disorders.[2,7] Therefore, a rationalized scheme for the diagnostic categorization of PNES has been suggested based on "predisposing, precipitating, and perpetuating factors commonly interact."[2] A thorough diagnostic assessment of patients with PNES should include all of these factors; "otherwise treatment may be less likely to succeed."[2]

1. Many patients with PNES have other psychiatric disorders placed on axis I of the DSM-IV system. The commonest diagnoses are (other) somatoform or dissociative disorders, affective, anxiety, and post-traumatic stress disorder.[2]
2. Many patients show abnormalities of personality development, especially borderline personality disorder.[2]
3. Many patients have 'organic' brain disorders, most commonly epilepsy (in 10-50% of patients) and learning disability. PNES are occasionally first observed after epilepsy surgery or other neurosurgical procedures, especially if there were postoperative complications causing prolonged hospitalization.[2]
4. PNES commonly occur in the context of social or family conflict and trauma. Although no studies have examined this issue, it is also the authors' impression that the risk of developing PNES is greater in marginalized and immigrant populations.[2]
5. PNES patients often have a history of sexual or physical abuse in childhood and adolescence.[2]

PNES syndrome originates from psychological sickness or trauma that is delivered neurologically throughout the body.[5] PNES presents with a decrease or complete change in somatic function without the involvement of the central nervous system. Although there is no cortical involvement, this classification often shares similar qualities seen in epileptic seizures (ES). The presence of a visual aura is seen in both PNES and ES, along with impaired jerky movements, loss of coordination, temporary blindness or tunnel vision, loss of smell or touch and tingling sensation on the skin.[6] People with PNES will usually present with one of these symptoms, but if another episode suddenly reappears shortly after, then the symptom will relocate with a different intensity.[6,8] When symptoms occur from periods of extreme emotional stress, some doctors believe that this is an attempt to reduce anxiety because the person cannot cope or face their emotional conflict.[9] Another theory proposes the idea that postural changes and orthostatic symptoms can induce episodes of convulsive seizures.[6]

PNES is a complicated disorder and somewhat vague in its clinical diagnosis and management due to its similarities with ES, and is often mistaken for generalized tonic-clonic seizure, absence seizure and simple or complex epileptic seizures.[9,10] Moreover, "the failure to recognize the psychological cause of the disorder detracts physicians from addressing associated psychopathology (including suicidal ideation), and enhances secondary somatization processes."[2] The importance of distinguishing the cause through proper diagnosis of epilepsy is to prevent "significant iatrogenic harm" and unnecessary intake of anti-epileptic drugs, which have been proven to be potentially fatal and costly.[2,11,12]

Furthermore, differentiation exists between the conditions in an absence of epileptic activities recorded on the video electroencephalographic (v-EEG), showing no signs of abnormal cortical firing from the central nervous system.[2,9,13] Although clinical introduction of v-EEG 30 years ago allowed rapid growth in understanding and diagnosis of PNES, "the mean latency between manifestation and diagnosis remains unacceptably long at 7.2 years, and that three-quarters of patients with PNES (and no additional epilepsy) are still treated with anticonvulsants initially."[14] One explanation for the delay in diagnosis and inappropriate treatment may be that "patients often do not present to experts in the assessment and treatment of seizures but to generalists, physicians in emergency care departments, anesthetists, and even obstetricians or dentists."[2]

Some authors have suggested the initial treatment of PNES begins with a firm diagnosis, accompanied by a thorough neurological assessment, and complete understanding from the patient about their condition. One study comparing the utilization of v-EEG before and after the diagnosis of PNES demonstrated that "firm diagnosis of PNES, explanation of the problem, and arrangement of psychological treatment can lead to dramatic reductions in emergency room visits (97%),

outpatient clinic visits (80%), and diagnostic tests (76%) in the short term."[2]

Unfortunately, insufficiency in current literature has thus, limited further management of patients with PNES, and do not clearly identify types of psychological treatment offered. Therefore, upon the completion of a neurological assessment, a psychiatric examination is recommended to determine the proper use of psychological, pharmaceutical, or behavioral modification treatments, such as cognitive (behavioral) therapy.[2,8,15]

Case Report

Patient's History

A 30-year-old female patient presented with a chief complaint of full body convulsions that began in December of 2013. Before chiropractic care, she was diagnosed with a nonepileptic seizure disorder by an internist and two neurologists. The patient suffered from serious autonomic dysfunction, which appeared to cause symptoms of light-headedness, fainting without loss of consciousness, heavy chest compressions manifested from anxiety and loss of vision lasting 30-45 seconds. Other life threatening symptoms included complete loss of motor function in her legs, headaches, dizziness, nausea, fatigue and tremors. Progressively, these symptoms began to occur 3 times per week when she exercised or sat up too quickly, especially when getting out of bed in the morning, to lasting up to an hour each day. The patient assumed these symptoms were a result of low blood sugar, as she had a decreased appetite due to work stress.

The initial blood test, chemistry panel, kidney and liver function tests reported normal. Computed tomography (CT) of the brain, v-EEG and electrocardiogram (ECG) demonstrated to be within normal limits. Performance of the tilt table test was utilized to rule out potential syncopal disorders, such as postural orthostatic tachycardia syndrome (POTS).[16] The patient stated, once the procedure began, she started to acquire the same symptoms that were previously described. Therefore, Ativan, a psychotropic chemical substance, which has therapeutic effects to treat convulsive activities, was prescribed to treat her seizure-like symptoms. A study performed on the effects of Benzodiazepine demonstrated to be the initial choice in treating epileptic disorders or people who suffer from seizure-like convulsions.[12] She was instructed to take the medication as soon as she felt the onset of symptoms.

One month later, the patient noticed that her symptoms were not improving and the medication prescribed was not effective. She was still experiencing dizziness, vertigo, fatigue and weakness. Along with these symptoms, she was experiencing episodes of convulsions 4-5 times per day, lasting 5 minutes to an hour. The case was presented to another neurologist. After the patient was evaluated and re-examined, he concluded that the cause of her seizure-like symptoms were not due to POTS. The doctor identified that her symptoms were brought upon mental stress, causing her body to respond in a convulsive manner. He arrived to this conclusion based on the history of the patient and the lack of v-EEG epileptic changes with no correlation to the central nervous system. The neurologist was also informed that the patient was having convulsions in her sleep, which did not identify with POTS. In February 2013, the patient was diagnosed with a nonepileptic seizure disorder, also known as, conversion disorder.

Cognitive Behavioral Therapy (CBT) was strongly recommended, since psychotherapy is believed to be highly beneficial for PNES. Retrospective reports revealed that psychiatric therapy has shown a reduction in seizure incidences up to 70% of those diagnosed with PNES.[17] Along with therapy, as a "gold standard of care", Effexor; an anti-depressant of the serotonin-norepinephrine reuptake inhibitor (SNRI) class was prescribed to the patient.[9] Even though she did not believe that her symptoms resulted from a mental illness, she was hoping this would be the solution. The patient continued care for two months and felt that therapy and the medication were not resolving the problem. With all treatment options exhausted, the internist contacted an upper cervical chiropractor; experienced with providing services to those who did not have successful treatment with traditional medicine.

Based on her outcome assessment, the patient's symptoms have become significantly worse by 80%, since the onset in December. Personal stress levels were 3-4/10 and occupational stress levels scored 6/10. The convulsions with the other symptoms that followed interfered with her ability to work, making it difficult to produce income. Lifestyle habits were scaled as diet being very good, good exercise routine, poor sleeping patterns, and overall good health. She was drinking four 8-ounce glasses of water per day and was not consuming any beverages with caffeine. The patient denied any food allergies and the use of alcohol, tobacco, or recreational drugs.

Common triggers that lead to her episodes of convulsions included sleep deprivation that lasted for 3-4 days. Night terrors occurred 3-5 times per week, contributing to her insomnia. The patient slept an average of 5 hours, on the nights that she could sleep. Certain light patterns, velocities, noises, and periods of decreased appetite would elicit a convulsive response. She stated that these factors were equally provocative, making it difficult to identify a specific trigger.

At this point, the convulsions were happening 6-10 times per day, lasting 20 minutes to an hour. After each episode the patient experienced migraines, which became more intense and painful with each convulsion. Before each migraine, she experienced a visual phenomenon, resembling static on a television screen due to poor connection. The migraine was localized to the right side with pain sensation that was throbbing and sharp, lasting 20 minutes after each episode. The patient's condition disabled her from performing normal tasks, which included getting dressed or brushing her teeth without feeling dizzy. She was also unable to operate a vehicle and continue her exercise routine.

The patient denied any previous accidents, traumas, or hospitalizations that occurred prior to December. The patient revealed no health complications with family history. She denied any problems with gastrointestinal, cardiovascular,

genitourinary, reproductive and immunologic systems.

Chiropractic Evaluation

The QSM^3 method evaluates structural misalignment based on a pre/post measurement of the patient's posture. These measurements provide a guideline used for correcting the patient's misalignment. Initially, x-ray analysis was taken before and after the first treatment. Radiographic images included lateral cervical, nasium and vertex views. The posture IQ was utilized to obtain postural measurements, which then integrated into a postural listing used for correction. During the evaluation, palpation identified the low pelvis, and weight differentials, pelvic and shoulder rotations, and fixed point (VP; the relationship between C7 and L5) was collected with empirical data, using the posture IQ. Lastly, the patient stands in front of a grid to expose the low shoulder, neck and head tilt.[18]

Pre-assessment x-ray analysis of the lateral cervical view revealed the s-line at S4. This line was used as preliminary reference to line up the central ray used for the nasium view. The lateral cervical view showed a reversed cervical curve and break in George's line at C4. There was no evidence of fractures, bone or soft tissue pathology. (Figure 1) The nasium view revealed atlas laterality of 5^o on the left with $1½^o$ of head tilt towards the left. The C2 spinous rotation measured 10^o to the left with a left high plane line at $+7/16^{th}$ inches. The angular rotation or lower angle was found to be 3^o to the right. (Figure 2) Figure 3, the vertex view, demonstrated 2^o anterior rotation of the atlas. (Figure 3) The benefits of x-ray analysis for QSM^3 protocol was to show compression and twist of the spine and to present an image for patients to see how their misalignment affected their spine.[18]

The patient pertaining to this case presented with a left low hip. Next, the patient was placed on the posture IQ, measuring the side of greater weight distribution and spinal deviation. The device showed the patient bearing more weight on the left side by 3.2lbs (Normal=0-3lbs), compared to the right side with the VP deviated to the left. Through this procedure, the rotational measurement is then applied to the heavy side (left side) measured by infrared digital lasers. The first rotational difference is measured at the shoulders (N=0-3mm). The patient presented with a twist posterior (backward), by 21 mm. The pelvis, the second rotational difference measured (N=0-3mm), indicated an anterior rotation or twist (forward) by 6mm. Placed in front of a postural graph, the patient presented with a right low shoulder and a right neck and head tilt. The patient stated that her postural measurements correlated to the position she found herself when she was experiencing her convulsions. Respectively, the postural listing of the patient written in an algorithmic form: LL, $L^{3.2}{}_{P21A6,}$ RRR.[18]

The QSM^3 Method

QSM^3 is a forward thinking, tonal based chiropractic method designed to measure and correct global vertebral subluxation breakdown of the entire body. It is a systematic method, evolving from upper cervical roots, evaluating three-dimensional global misalignments from the pelvis to the skull. This chiropractic method analyzes the impact of stress on the body, leading to disruption in orthogonality and biotensegrity.[18] As a result, trauma entering a bio-tensegral system causes global breakdown, ultimately leading to an unstable system fighting against gravity.[19] The goal of QSM^3 is to restore three-dimensional structural relationships to a sustainable tensegral state (optimal healthy posture), in order to improve nervous system function, joint symmetry and expanded muscular motion.[18,19,20]

QSM^3 protocol follows postural misalignment patterns to create a model that details "Cause to Correction." The method provides an understanding that with every postural misalignment comes structural injury with a neurological response to that trauma. Since postural misalignments occur three-dimensionally, it is found that the human body expresses both structural and functional consequences. Structural and neurological breakdowns are obtained by measuring from the pelvis to the shoulders (structural component: low pelvis, spinal deviation from C7 to L5, weight of the heavier side, rotation of the shoulder and pelvis) using the posture IQ; and by observing the neurological response to breakdown (neurological component: low shoulder, neck and head tilt).[18,19,20]

Since trauma creates a devolving path of breakdown through the whole body, correction must follow the reverse pathway of breakdown. Pathways are released via a dynamic correction through the myofascial envelope (MFE) contained within the entire cervical region (C0-C7). The MFE coordinates balance through a matrix of pathways that originate in the feet, and anchor superiorly until they all insert in the skull. Modulation of the superior structure (skull) via the righting reflex, vestibular apparatus and proprioception (with most dense beds at C0/C1/C2 and pelvis) creates tension lines throughout the MFE to maintain balance and agility. It is the recursive coordination and orchestration of input from sensory proprioceptors, coupled with central integration, and ongoing 3-dimensional motor response by the MFE that maintains healthy position/configuration in the whole body. This keeps necessary energy input and biomechanical stress to a minimum.[18,19,20]

Using postural data, the QSM^3 method implements a multiple headpiece protocol on the side lying patient. The purpose of the head placement is to increase and isolate tension, 3-dimensionally, in order to release compressive breakdowns. This is accomplished by contacting Golgi tendon organs (GTO's), sensory receptors located at the origins and insertions of muscle fibers, which insert into the tendons of skeletal muscle.[18,19,21] The primary function of GTO's is to sense tension placed on a muscle, acting as a protective "negative feedback system" to reduce the activation of the force generated in the muscle. When activated through the adjustment, the GTO's cause muscle relaxation, which releases the causative, stabilizing and compensatory tension lines created by trauma misalignment.[18,21]

QSM^3 approaches the adjusting process by observing the body's loss of orthogonality, leading to global subluxation breakdown. The human body is observed to express postural language that explains the progressive breakdown and neurological response in a gravitational field throughout a person's lifetime. The postural listing is a real-time representation of the body's current structural position and is a

visit-by-visit relationship of the postural configuration and the energetic ability to hold it upright.[18] Although, the entire cervical region is the point of contact, QSM[3] methodically identifies the entire body as one system within a closed kinetic chain. Therefore, an orthogonal imbalance within this system will lead to a decrease in transportation of blood, oxygen and nutrients to organs and tissues. Furthermore, since toxic wastes cannot properly be removed from the body, accumulation of these byproducts will cause "Dis-ease".[18,19]

Outcomes

The patient's care plan consisted of three visits in the first week, and then twice a week for the next five months. The patient has been responding well to care and after five intense months of treatment, she was asked to come in for care once every two weeks. At the initial visit, the patient had a 21mm posterior rotation in her left shoulder. This was believed to be one of the major components contributing to her PNES disorder. After the first treatment and the release of the tension lines, opening up the pathways to restore tensegrity, the patient's digital posture revealed a shift in weight distribution from 3.2lbs on the left to 1.7lbs on the right. The rotation in her left shoulder decreased from 21mm to 8mm and the pelvis rotation decreased from 6mm to 0mm, on the same side. The right low shoulder, neck and head tilt was released from tension and brought back close to neutral. The post x-rays revealed a reduction in atlas laterality to 1^{o} on the left. The head tilt moved into the right frontal plane by 1^{o} and the high plane line reduced to $2/16^{th}$ inches. The lower angle reduced to $3/4^{o}$ on the right.(Figure 4) The vertex showed a decrease in atlas rotation on the right from 2^{o} to 0^{o}. This view also revealed a decrease in C2 spinous rotation from 10^{o} to 0^{o}. (Figure 5)

The patient responded well after the initial adjustment. She explained that she has never been able to stand up right without feeling compression on her spine and internal organs. The patient's convulsion with preceding symptoms ceased for two days after the initial treatment. On the second visit, the postural analysis showed that she was slightly holding her adjustment, but her body kept trying to move into her subluxation due to prolonged trauma misalignment. After the second visit, her signs and symptoms had not been apparent for 10 days. On the tenth day after the second treatment, the patient stated that her episode did not last longer than 5 minutes. She explained that she believed this episode was self-inflicted, as a result of exercising lightly, placing her body into the same positional pattern found on the digital device. She returned for care and after treatment, 3-4 weeks later in September, she experienced an episode of convulsions, also claiming to be self-induced. While getting her hair washed at a salon, the stylist left her head in extension over the sink to dry. After this incident, she became aware of avoiding certain body positions that would induce her symptoms and convulsive behavior.

After 7 months of treatment, there has been a significant decrease in the frequency in convulsions with the related symptoms, as stated previously in the patient's history. The patient has not experienced any episodes or symptoms related to her condition of PNES. She currently comes in for QSM[3] treatment twice a month.

Discussion

Mind Over Matter

A connection exists between an individual's emotional experience and associated physiological properties. The expression of feeling is subjective and conveyed through visceral and somatic motor responses. Biological processes are altered every day from emotional responses causing a change in genetic makeup promoting psychogenic sickness. Everyone experiences an emotion differently, making it difficult to describe the effect it has on one's body. Although true, the physiological response associated with the individual's experience is common in all human cultures. Numerous regions in the brain are critical for integrating and processing these emotions; these include the amygdala, several cortical areas in the orbital and medial aspects of the frontal lobe, and related subcortical circuitry. Regions in the forebrain (cerebral cortex) and diencephalon (thalamus, hypothalamus) control lower motor neuronal pools concerned with the body's response to emotional behavior. This diverse area of the brain integrates motor response, which is triggered by emotion.[21,22,23] In comparison, researchers have studied the effects of sensory input delivered from muscles and internal organs. These signals form the sensory limb of the reflex circuitry that allows the body to change its physiological makeup in order to adapt.[21,24]

Physiological responses arise from an intricate pathway of neural activity coursing its way from the forebrain to visceral and motor nuclei through the hypothalamus and brainstem reticular formation. The hypothalamus and reticular formation are crucial structures in the brain, which cause transformation of physiological processes produced by an emotional response. The primary targets of the hypothalamus exist in the reticular formation, scattered throughout the brainstem, containing a network of nerve cells and fibers that are intertwined to make connections that have somatic and visceral motor control. This area receives input from the hypothalamus and directs the stimulus to the effector systems in the brainstem and the spinal cord, providing motor control of muscles, tendons, and organs. These reticular neurons have extensive control delivered to somatic and visceral motor systems causing dominance over reflex systems involving almost every tissue in the human body.[21,24]

Besides the hypothalamus, the limbic system offers another descending pathway which projects from the forebrain to the brainstem reticular formation. The limbic system is composed of the cingulate gyrus, located above the corpus collosum and the parahippocampal gyrus, centrally located in the medial temporal lobe. The projections of emotional expression can be traced through a parallel system that descends from the cortex, giving off signals of somatic and visceral motor events, integrating a response of emotional behavior.[21,23]

The systems that descend parallel to one another are composed of projections coming from voluntary motor control and emotional expression. Voluntary motor control arises from the posterior frontal lobe and interconnects with the basal ganglia and cerebellum. The basal ganglia strongly communicate with the cerebral cortex, thalamus and brainstem to perform variations of functions, such as control of voluntary

movements and emotion. The cerebellum is responsible for motor control of fine movements, posture and motor learning.[21,23]

Motor projections originate from motor cortex descending through pyramidal (corticospinal) and extrapyramidal tracts (corticobulbar). The corticospinal and corticobulbar tracts terminate in the spinal cord or brainstem, respectively. These projections are arranged into medial and lateral components that make up voluntary muscle control. The medial portion controls postural tone and motor control to proximal extremities. The medial projects signals to the brainstem reticular formation and the motor neuron pool, which are composed of motor and autonomic cell bodies. The lateral portion controls fine movements of distal extremities and send their signals directly to the motor neuron pool.[21,25]

Emotional expressions descend pathways through extrapyramidal projections from the limbic centers of the ventral-medial forebrain and the hypothalamus. As seen with motor projections, these pathways are composed of medial and lateral divisions. The medial component sends rhythmical reflexes to the brainstem reticular formation and to the motor neuron pool. The lateral component sends specific emotional behaviors directly to the motor neuron pool.[21,25]

The voluntary motor and emotional expression pathways share a common route that begins in the cerebral cortex, sending signals to the brainstem and/or a collection of motor neurons that innervate muscles for contraction and movement. Located in this neuronal pool, arises autonomic ganglionic neurons that activate the contraction of smooth muscle and glands.[21,25]

Mechanotransduction and the Tensegral Model

Mechanotransduction is defined as mechanisms used by cells to transform mechanical stimulus into biochemical responses. This involves converting the stimulus into electrical or chemical signals. Mechanical forces that are received by the body produce an alteration in cellular activity and "genetic expression". This provides a mechanistic correlation between mechanical forces and their influence on genetic control at the molecular and cellular level. The mechanical stress delivered into a system will cause the nature of a cell to change its shape, which consequently alters its structure and cellular function. Therefore, the composition of the human body relies on a "tension-dependent building system" to maintain stabilization. Mechanotransduction are responsible for various types of sensations and physiological processes that occur within the body. The primary stimuli received by these receptors detect proprioception, touch, balance and hearing.[19,20,26]

Tensegrity is defined as a mechanism used in the body to balance confined "compressions" within a closed system that is continuously under "tensional forces". The "architectural" design of the human body is built from forces that are complied in effort to maintain function and stability, providing structural support against gravitational pull.[26] The musculoskeletal system offers a "supporting framework" that allow adaptation to occur from forces transmitted from the environment. Assistance from "tension-generated muscles and tension-resisting tendons, ligaments and fascia, bones and cartilage", the human body can withstand compressional forces, restoring orthogonality. A representation of the human body as a fluid-filled entity that is up and open and is made to move freely within its environment; using its musculoskeletal system as a means of communication and integration.[20]

Mechanotransduction and the tensegral model support the causative factors that play a role in the predisposition to PNES. The human body must adapt to forces delivered from the external and internal environment. Aberrant signals create a vicious cycle that is produced in the body as a coping mechanism. Psychological dysfunction sent throughout the nervous system will cause postural misalignment, placing the body off center.[19,20,26]

Proposed Mechanism

The pathophysiology of Psychogenic Nonepileptic Seizures is not understood and is usually misdiagnosed.[1] PNES are presently diagnosed under the DSM-V as conversion disorder and "defined as neurological symptoms arising without organic damage to the nervous system, presumably in relation to various emotional stress factor".[7,27] The proposed mechanism and neurological factors that cause symptoms of this disorder pose a challenge in diagnosis, management and understanding control of physiological parameters.[1] This section of the paper is intended to hypothesize a mechanism correlating the effects of emotional behavior on the neuromusculoskeletal system causing symptoms of PNES and the benefits of chiropractic treatment using QSM[3] technique.

Ivan Pavlov, a physiologist known for his work in classical conditioning, suggested that emotional stimulation derived from subcortical centers in the brain required "inhibitory control" from the motor cortex. He proposed the idea that excessive inhibition can occur as a result of extreme emotional behavior. This uncontrollable response can become overwhelming to the nervous system causing overspill to adjacent pathways, impeding on proper motor output. The functional anatomy that contributes to "hysterical conversion" includes the frontal cortex, thalamus, basal ganglia, or limbic system. Thus, alterations in excitatory sensory or motor pathways cause various pathophysiological consequences to these regions of the brain. Loss of muscle function, sensation, or vision can result from excessive signals coursing through their nontraditional pathways. Effects of inhibitory signals will obscure integration of motor, sensory, or cognitive functions into "conscious awareness."[22,27]

The primary motor cortex is mainly controlled by nerve impulses delivered from the somatosensory system. Subcortical fibers, somatosensory containing mechanoreceptor stimulus from skin and proprioception from joints and muscles and tracts from thalamic nuclei are significant incoming pathways delivered to the motor cortex. After signals are received, the basal ganglia and the cerebellum work closely together to activate a specific motor response.[25]

The premotor area, located anterior to the primary motor cortex, produces nerve impulses designed for intricate "patterns" of movement. The premotor cortex sends signals to the basal ganglia and the thalamus, which in turn sends the information back to the primary motor cortex. When

emotional stimulus overrides the signals delivered to the basal ganglia, suppression of these impulses inhibit motor control of complex patterns of muscle coordination. Motor impulses travel from the cortex to the spinal cord through the corticospinal tract sending signals indirectly to the basal ganglia, cerebellum, and various nuclei of the brainstem. The corticospinal tract, also known as the extrapyramidal tract, receives 30% from the primary motor cortex, 30% from the premotor cortex and 40% from the somatosensory cortex. Overall, the strongest motor stimulus is sent from the motor cortex and received by the basal ganglia, brainstem and cerebellum.[24,25]

Regulation of proper muscle function is achieved by constant sensory feedback from muscle spindles and GTO's. Muscle spindles are scattered throughout the belly of a muscle transmitting signals about muscle length to the central nervous system (CNS). GTO's, located in muscle tendons, send impulses about the tension placed on the muscle tendon. This is controlled subconsciously, sending information to the spinal cord, cortex and cerebellum to regulate muscle contraction. The "bulboreticular facilitatory" area in the brainstem primarily stimulates gamma efferent excitatory signals, located in the anterior horn of the spinal cord. This region receives impulses from the cortex, cerebellum and basal ganglia. The primary concern of the gamma efferent system is to stimulate muscle spindle antigravity contractions.[25]

The muscle spindle is designed to stabilize posture during tensional forces placed on the body during motor activity. Excessive emotional behavior causing active inhibition of the motor cortex, results in an overflow of inhibitory signals. These signals begin to spillover to adjacent pathways causing inhibition to sensory and motor stimulus. The bulboreticular facilitatory area and its associated brainstem regions transmit impulses to the muscle spindle. Active inhibition will suppress inhibitory signals delivered to antagonistic muscles. Therefore, muscle spindles are activated on both sides of each joint producing sustained contraction inhibiting voluntary movement.[25,28]

"Hysteria" is thought to be generated by emotional distress causing symptoms of conversion disorder that are associated with a primeval protective response, "such as motor arrest, freezing, playing dead and agitation." "Topographically" structured tracks in the basal ganglia and periaqueductal gray can alter motor function regulated by centers located in frontal cortex and subcortical limbic regions. Emotional stress can cause modification to these centers suppressing sensory signals traveling through the central nervous system or transmission of motor signals sent out to the periphery.[27]

An experimental study was conducted on a female patient with paralysis in her left leg for 2 years and was diagnosed with conversion disorder. Positron emission tomography (PET) demonstrated no activation of her right motor cortex while attempting to move her left leg, whereas signals in her left hemisphere were activated upon movement of her unaffected right leg. At the attempt to move her paralyzed leg, the PET demonstrated intensification of activity in her anterior and medial prefrontal cortical areas. These regions that were active corresponded to the "anterior cingulate cortex, as well as the ventromedial and orbitofrontal cortex", part of the limbic prefrontal cortical region. The results revealed emotional projections creating an "activated inhibition" of the motor cortex producing paralysis of her left leg. The interpretation supported the proposed mechanism of "active suppression of sensorimotor pathways under the influence of other brain systems" created by Pavlov.[27]

The development of a "theoretical model" is proposed to help alleviate the challenge of understanding the cause of PNES. The theory conceptualizes the process of "active inhibition of motor areas by the limbic system". As previously stated, the limbic system (subcortical) composed of the orbitofrontal and the amygdala, primarily sends unconscious inhibitory signals to "striato-thalamocortical premotor loops"[22,25] The orbitofrontal region is part of the frontal cortex responsible for "cognitive processing of decision-making". This region communicates with the thalamus and periaqueductal gray areas providing inhibitory and excitatory control over the autonomic system, causing rhythmical contractions seen in PNES. This system integrates stimulus into behavioral expression. The amygdala is primarily responsible in forming and storing memories that have strong correlation with emotional events.[29] Improper stimulation of the amygdala can cause numerous types of involuntary muscular contractions, which include tonic, circular, or rhythmical movements.[25]

The etiology and pathophysiology of PNES correlate to various functions, which include emotion, attention, representation, motor control and neurological networks. These proposed functions are interconnected with one another, sharing similar pathways to reach their target. An excess amount of stimulus can cause pathways to exceed its limit of neuronal activity causing leakage of signals. These signals will target different regions of the brain altering a change in biomechanical activity. As a result, change in motor output can lead to signs and symptoms of PNES.[29] A proposed mechanism to support this idea related to one of the models of vertebral subluxation discussed in Kent's article, the Dysafferentation Model.[30]

The Dysafferentation Model of subluxation can explain the effect of chiropractic management using QSM^3 technique on a patient suffering from PNES. The psychogenic disorder causes a change in afferent input delivery to the CNS by limbic overload. The overwhelming emotional stimulus causes a change in postural tone, altering biomechanical property. Postural imbalance can lead to proprioceptive dysfunction delivered by neurological stimulus that are received from mechanoreceptors.[30] QSM^3 contacts GTO's attached to the base of the skull and throughout the cervical spine to release tension lines created by myofascial networks.[18,19]

Posterior cervical muscles play an important role in sensorimotor regulation, neck pain and headaches and stabilization of the spinal cord. "Myodural bridges" link the suboccipital muscle fascia and dura, which is used to anchor the spinal cord. Dural tension may be stimulated by sensory impulse to muscle fibers; this reflex is proposed to cause dural tension. These posterior muscles are believed to anchor the other musculature that attaches to the skull.[31] QSM^3 delivers a low force adjustment through the use of the doctors' bony landmark in the hand known, as the pisiform. Through this long lever, the pisiform makes a precise and direct contact

with the GTO's, located deep within the muscle belly of the fascia fibers. This connection between pisiform and muscle tissue, and by means of wrist radial deviation, a relaxation mechanism is elicited to the GTO's, resolving the tension within the fascia itself. The energy delivered through the QSM[3] correction may trigger stimulus to hinder the motor cortex response to aberrant firing of the limbic system. Through postural analysis, QSM[3] has made means of measuring the body's ability to integrate trauma, externally to rebalance, internally. Apparatus of this model allows a series of headpiece positions to be augmented and used in an effort to release the tension within the body, assimilated due to external trauma. End result of this procedure and underlying philosophy is restoration of the body's innate ability to be within orthogonality and in its upright tensegral being; and by releasing traumatic tension exemplified within the myofascial tissue and researched cause of neuronal interference. Correction is seen by the removal of compressional forces, induced by traumatic experiences and described as a phase known as decompression. Removal of this compression allows the body to pop up and open, as if it were a spring and is represented best thru the tensegral model. All in which leads to an outcome reinstating true orthogonality, where the body is restored to its natural state of center, ultimately improving the patient's symptomotalogy.[18,19,20,26] Due to the limited amount of study provided by chiropractic literature, further research is warranted pertaining to the benefits of chiropractic treatment, specifically QSM[3], in reducing symptoms of PNES.

Conclusion

Chiropractic management of a 30-year-old female patient suffering from PNES is presented in this case study. The management that was performed utilized the QSM[3] method to correct global subluxations caused by postural breakdown of the human body, which led to a reduction in symptoms pertaining to PNES. As a result, compressional release of myofascial tension throughout the patient's body, restoring tensegrity, demonstrated both objectively and subjectively successful.

This case study supports the ability of using tonal-based chiropractic care, as an alternative form of treatment, in reducing symptoms of a disorder that has no neurological origin. Since chiropractic research is limited on conditions, such as psychogenic disorders, further research is needed to support the role of chiropractic treatment and its effects on patients with global subluxations, potentially leading to symptoms, resulting from psychogenic disorders.

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Preliminary Cervical X-Rays

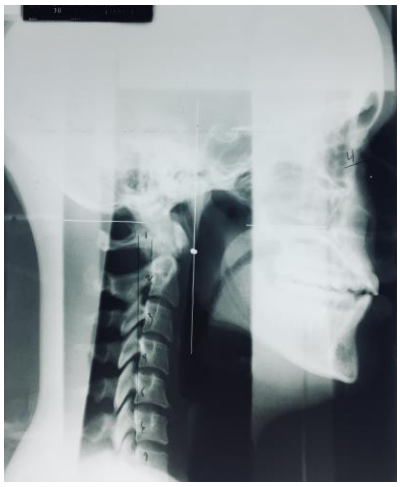

Figure 1: Lateral

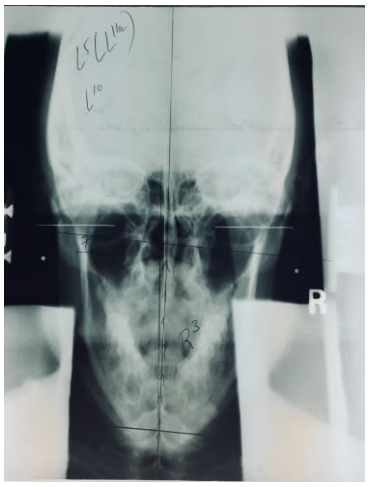

Figure 2: Nasium

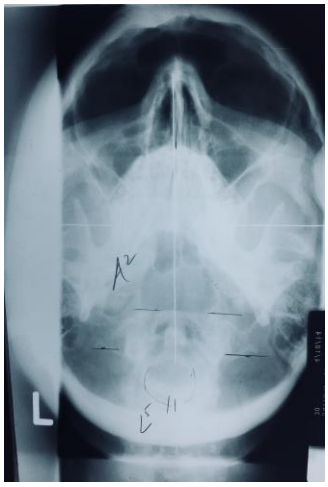

Figure 3: Vertex

Post Cervical X-Ray

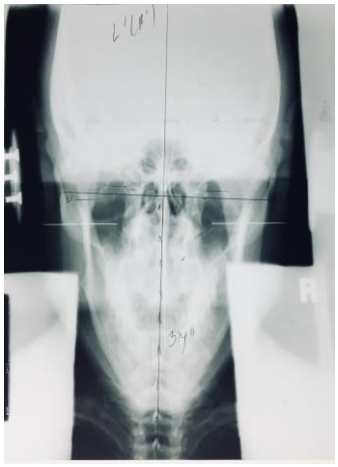

Figure 4: Nasium

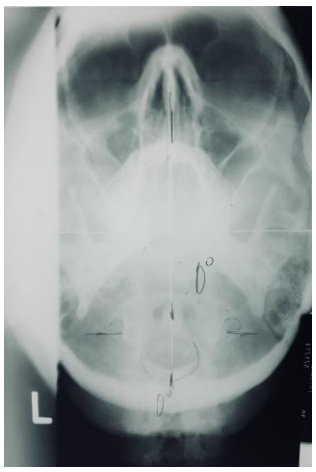

Figure 5: Vertex