CASE STUDY

Resolution of Vasovagal Syncope (VVS) Following Upper Cervical Chiropractic Care: A Case Study & Review of the Literature

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ABSTRACT

Objective: The purpose of this study is to describe the resolution of symptomatology in a patient with Vasovagal Syncope (VVS) following chiropractic care to correct upper cervical subluxation.

Clinical Features: A patient formerly diagnosed with VVS, chronic diffuse neck pain and episodes of spontaneous fainting and blackouts presented for chiropractic care. Pharmacotherapy and the surgical implantation of a pacemaker had been recommended. Chiropractic examination revealed a patient with global postural misalignments and vertebral subluxations. X-ray analysis revealed severe rotation between C1 and C2; with C1 rotated posterior 7 3/4 degree in the right transverse plane and C2 rotated an additional 5 degrees left, on C1.

Intervention and Outcomes: Quantum Spinal Mechanics 3 Dimensional protocol (QSM3) was utilized. Following adjustment to reduce the upper cervical subluxation the patient experienced resolution of her syncopal episodes.

Conclusions: This case study revealed complete resolution of VVS disorder following the administering of QSM3 care. Additional research on the benefits of tonal based chiropractic is further needed and encouraged.

Key Words: *Vasovagal syncope, neurally mediated, neurocardiogenic, reflex mediated syncope, Quantum Spinal Mechanics 3, chiropractic, adjustment, subluxation, atlas, upper cervical*

Introduction

With origin and derivative from ancient Greek, the term syncope, means to "cut short" or "interrupt." According to the World Journal of Cardiology (WJC), syncope is defined as "a transient and self-terminating loss of consciousness (LOC) with rapid onset, short duration combined with spontaneous, prompt and complete recovery."[1] Syncope is said to be a result of a large number of different disorders, all of which cause a transitory reduction in cerebral blood flow adequate enough to disturb the normal functions of the brain and characterized as global cerebral hypo-perfusion. The Journal of the American College of Cardiology denotes syncope to be a particular type of disturbance and has categorized it by the following: 1) loss of consciousness is temporary; 2) recovery is spontaneous, prompt, and complete; and 3) the cause is insufficiency of cerebral nutrient supply. A transient fall of systemic arterial pressure to levels below cerebrovascular auto-regulation

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requirements is most often to blame.[2] In clinical terms, patients who present with having experienced a transient loss of consciousness (TLOC), report their phenomenon to be an episode of loss of consciousness, collapse, fall, and/or a period of black out.[3]

According to the British Medical Journal (BMJ) based on a Cohort and population related study around 40% of the adult population has experienced a syncopal episode (usually described as a "faint" or "blackout"), with women more likely than men to report such an episode. The incidence is higher with advancing age, and this trend coincides with the increase in prescription of vasoactive drugs and increasing incidence of cardiac arrhythmia in the elderly population.[4] The prevalence rate for a syncopal episode is said to account for 3% of all emergency room visits and between 1 to 6% of all hospital admissions. Furthermore, syncopal episodes recorded in the general population were between 1.9 to 4.3 times higher, than that recorded in general practice population. They are 13 times higher than the number of cases that have presented to the emergency or intensive care departments; suggesting that not all individuals who experience a syncopal episode present to a health care provider. The incidence of syncope occurring has shown to increase with age where 30% of patients with syncopal episodes experience recurrent attacks. Morbidity from syncope, although less frequent, is said to occur from injuries such as: lacerations, contusions, subdural hematoma's, limb fractures, and possible related organ damage made by automobile accidents and/or falls. Depending on the mechanism of the disorder, the annual mortality due to cardiac causes is said to be between 20 to 30%, whereas mortality due to non-cardiac origin is said to be between 5 to 10%.[5,6]

Over the past few decades focus has been made on types of syncope that occur as a result of a centrally mediated, or "reflex" fall in systemic blood pressure occurring. Research into the nature of this disorder revealed it to be a single aspect of a large and wide-ranging group of disturbances affecting the autonomic nervous system (ANS), and each of which possibly resulting in hypotension, orthostatic intolerance, and ultimately syncope.[7] Continued investigations into the nature of these similar yet different disorders has led to the development of a system of classification that attempts to more accurately reflect our understanding of these conditions and their interrelationships. The present system of classification includes a group of disorders that most investigators have thought to be principally autonomic in nature and categorized with the term 'reflex syncopes'.[3,7]

Vasovagal syncope (VVS) is also referred to as neurocardiogenic, neurally mediated, vasodepressor and/or reflex mediated syncope. According to the 2005 edition on Heart Disease Diagnosis and Therapy: A Practical Approach, Second Edition, author M. Gabriel Khan, MD, concisely assembles a format on the clinical presentation of VVS.

Vasodepressor: a profound fall in peripheral vascular resistance and marked reduction in BP occurs, but the heart rate usually remains above 60 beats per minute (BPM); Vasovagal: predominantly cardio-inhibitory; a fall in BP occurs, but there is marked vagal induced bradycardia of less than 60 BPM; A combination of vasodepressor and vasovagal features. The vasodepressor component with marked reduction in BP appears to play an important role in loss of consciousness. Bradycardia plays a secondary role. These features explain the poor response to atropine. Thomas Lewis, in 1932, in his classic paper, stated that: While raising the pulse rate up to, and beyond, normal levels during the attack, leaves the blood pressure below normal and the patient still pale and not fully conscious. Abboud makes the relevant comment that 60 years later, Sra et al. can make the same statement with respect to the implantation of a pacemaker. Thus, the marked vasodilatation causes temporary, but profound, hypotension, with SBP of less than 65 mmHg which produces syncope even when the heart rate is 60–80 BPM. The marked vasodilatation is caused by the inhibition of sympathetic vasoconstrictor activity at the very moment when arteriolar vasoconstriction is necessary to combat the marked fall in BP. In most patients, the onset of bradycardia is consistently preceded by hypotension. Sra et al. have shown that an increase in myocardial contractility and a decrease in left ventricular (LV) systolic dimensions occur 2–4 minutes before the onset of syncope.[8]

Although a benign condition, VVS is the most frequent source and cause of fainting. NMS is said to be more common in children and young adults, but can occur at any age. According to the American Heart Association (AHA), NMS occurs as a result of blood pressure dropping, causing a reduction in circulation to the brain and resulting in loss of consciousness. Typically, VVS occurs while standing and is often followed by nausea, sensation of warmth, lightheadedness or blackout occurring; where prolonged episodes have shown to even trigger a seizure.[9] The occurrence of seizure-like activity yielding a tonic-clonic response, is said to have association with both cardiac and neurological origin, resulting in syncope.[10]

According to the American College of Cardiology (ACC), a modern form of examination that has become a widely accepted tool in the clinical evaluation of patients presenting with syncopal symptoms, is the use of a head-up tilt table (HUTT) test. Presently, there is substantial consensus that tilt table testing is an effective tool and technique in providing adequate and direct diagnostic evidence indicating susceptibility to NMS syncope. HUTT testing is used in an effort to generate a response to an upright head tilt in patients with suspected NMS, as well as, allows differentiation from other patients experiencing syncope, without neurally mediated origin.[11] The procedure and protocol for the HUTT test ranges from centre to centre and as such, the consideration reported in the literature widely differs between the ranges of 30 to 85%. Preceding the guidelines on administration of the HUTT test, regulated by the ACC, sensitivity of the study is said to be around 65%, while specificity is reported to be between the ranges of 80 to 90%.[12]

According to an article published by the BMJ journal entitled "Midodrine in the management of syncope," VVS is characterized by "a baroreceptor mediated hypotensive/ bradycardic response to orthostatic stress. Head up tilt induces venous pooling with an associated decrease in cardiac output, which stimulates a baroreceptor mediated sympathetic response resulting in an increase in vascular tone and heart rate. This increased heart rate and inotropy coupled with reduced venous return, however, stimulates mechanoreceptors

that may be situated in the wall of the left ventricle, which in susceptible individuals can trigger a centrally mediated withdrawal of sympathetic tone to the peripheral vasculature and an increased parasympathetic response, resulting in vasodilatation and bradycardia."[13]

The HUTT test is utilized in an effort to examine the neuro-cardiovascular orthostatic response, in an environment that provides maximal control. The effect of passive orthostatic stress on cardiovascular control maximizes the stress placed on the sympathetic nervous system by means of obstructing the influence of the lower limb's musculoskeletal contractions, that have potential in rendering an increase in venous return. Protocol for HUTT testing, including the degree of table tilt and angle, duration of tilting, as well as the addition of pharmacologic stimulation, are all within the examiner's control and discretion. The HUTT test is considered to be a dedicated test in which the orthostatic challenge is much longer duration than that encouraged in an office setting, the controlled variables of the test allow its value to be maximized, as well as, enabling the physician to pay more attention to the patient's symptoms being exacerbated and observed during the partly automated setup.[14]

Conventional management of NMS is said to consist of education and skills in an effort to avoid syncope occurring. Pharmaceutical treatments as well as surgical implantation of a pacemaker are also used. The preferred drug management for initial treatment consists primarily in recommendation of beta-blockers. Beta-blockers are prescribed to reduce the degree of mechanoreceptor activity and their ability in blocking the effects of circulating catecholamines. However, according to numerous research studies where randomized controlled trials were conducted, the efficacy of these drugs was questioned due to showing no difference in comparison from placebo. Furthermore, beta-blockers have been suggested to possibly worsen syncope due to negative chronotropic effects changing the rate and rhythm of the electrical conduction of the heart and their effect in blocking conduction of the atrioventricular node. Other drug treatments used in treating NMS include alpha-agonists, selective serotonin re-uptake inhibitors, fludrocortisone, disopyramide, scopolamine, and anticholinergic agents.[6] A further discussion in regards to the management of this condition is beyond the scope of this case study.

This case study will review and discuss the chiropractic QSM3 mechanism utilized in the management and remission of a female patient initially presenting with an upper cervical subluxation and VVS.

Case Report

Patient History

A 57-year-old female patient presented with a chief complaint of fainting which was diagnosed by her cardiologist in 1998 as vasovagal syncope (VVS). The patient also has a secondary complaint of chronic diffuse neck pain between C0-C7, primarily on the right and associated with decreased right rotation. The patient claims her episodes of fainting first began in 1996, where she first noticed that turning her head to the left (rotation) exacerbated a syncope attack. The patient reported that her symptoms began two years prior to her seeking medical treatment and was diagnosed by her cardiologist with VVS in July of 1998. An EKG, cardiac Echo, and carotid artery doppler were performed and revealed as negative studies. The tilt table test was also administered, which rendered as positive when performed to the patient's left. The patient was prescribed fludrocortisone acetate, a drug normally used to treat low blood pressure and associated with preventing vasovagal syncope. She was also deemed eligible for surgical consultation for implantation of an electrical pacemaker. However she claims to have denied both pharmaceutical and surgical interventions, choosing to seek holistic health care prior to committing to either means initially recommended by her cardiologist.

Chiropractic Analysis

Quantum Spinal Mechanics 3 Dimensional (QSM3) is a method in which postural analysis and examination is applied in a pre and post system of assessment for the evaluation of a patient's subluxations. The initial means of measurement will be constructed through a pre X-ray taken prior to the first adjustment and a post X-ray acquired after the initial adjustment is given.

X-rays are done in the cervical region of the patient's spine and include a lateral, vertex and nasium views. A supine leg check, use of the posture-measuring device (PMD), and a postural grid, are all used to aid in the formation of a postural algorithm that will take into effect the correction of the patient's misalignment and its correlation to head piece positioning.

The use of the supine leg check will be measured for the observation of leg length inequality and discrepancy, ultimately revealing the short leg. Next in routine is the recording taken on what is called the posture-measuring device (PMD). The PMD measures two planes of movement: 1) the frontal plane, around the z-axis, which is seen in lateral flexion or bending and 2) the transverse plane, around the y-axis, which is seen in rotation. Its use in the evaluation is for recognition of a low shoulder or what is called the "bow", as well as for the measurement in rotation of the shoulders and pelvis. At the base of the PMD and what the patient places their feet on, are two weighted scales. The scales are used in a manner of measurement for mass and recorded in pounds (lbs.). The heavy side, or side greater in weight, is the side recorded. Lastly, is the use of the "grid". The grid is used to evaluate the neck and head relationship and/or distortion. With the patient standing in front of the grid and facing the doctor for assessment, the doctor is able to note which way the neck deviates, as well as the head.

Pre-Assessment

Pre-assessment X-Ray analysis taken of the cervical spine first requires a lateral view as a means of determining what is called the "s-line". The s-line, as standard procedure, was used as a preliminary reference to line up the central ray of the tube, used for the angle in the nasium view.

The patient presented with an s-line categorized as S4, which requires the central ray in the nasium to be centered through

the inferior aspect of the orbit of the eye. The lateral view was also used to show the rotational component of atlas, as well as the integrity of the cervical spine. Figure 1, exhibits the rotation of the patient's Atlas, C1 in the cervical spine; with reference and use of lead markers that were positioned on both inferior lobes of the ear. (Figure 1) These markers are used and placed in this location as a means to line up with the atlas. The line drawn upon analysis of the lateral view is titled the atlas plane line. The atlas plane line is drawn through the thinnest part of the posterior arch of atlas in conjunction with the anterior tubercle of atlas. The lateral film, once analyzed using the atlas plane line signified the patient to yield an S4 line of structural correlation. The S4 line was then implicated in the nasium view and the angling of the x-ray tube.

Figure 2, represents the nasium view, which is used for head angle and tilt. Figure 3, demonstrates the vertex view, displaying rotation of atlas (C1) to axis (C2) and its indirect correspondence to the cervical spine. The lateral cervical (sagittal), nasium (frontal), and vertex (transverse), detailed the patient to present as a type three out of pattern misalignment. The most significant finding was the rotation of C1 and C2 to the center of the foramen magnum. The vertex and nasium views demonstrated C1 to be rotated posterior 7 3/4 degrees in the right transverse plane and C2 to be rotated an additional 5 degrees on C1.

The patient presented with a short leg in the supine position of right by 1/2". Following the leg check, the patient was positioned on the digital device (PMD), measuring the side of greater weight distribution. The PMD demonstrated on digital scales a right frontal imbalance of 10 pounds, associated with a pelvic laser reading of rotation posterior by 6 mm in the transverse plane, as well as, the shoulders to be rotated anterior by 12 mm in the transverse plane. The fixed point of C7 was also recorded, measuring left. Following the PMD reading, the patient was further placed in front of the postural figure, presenting with a right low shoulder, a left neck and a left head tilt. The postural listing of the patient written in an algorithmic form, is as follows: RL R10 A12 P6 RLL. The patient's postural listing was then articulated to express a headpiece listing composed as LPA 2-13, LL.

Method

The methodology, in detail is done by the evaluation and correction of abnormal posture in correlation of the short leg of the pelvis, to the neck and skull of the head. The foundation and philosophy of this technique is an association and congruency of vitalism, the unique model of tensegrity, a myofascial envelope (MFE), and the neuromuscular connection between fascia and the brain. QSM3's protocol follows a flow equation used as a means to develop a misalignment guideline where a model of "Cause to Correction" is exemplified.[15] The flow equation incorporates three components which include: 1) Cause, implication of the trauma induced in to the human body, 2) Compensation, the sensory-motor relationship and effect following the trauma, and 3) Collapse, the gravitational aspect that leads to tension and compression within the body, over time. Thus, creating a triune of understanding the body and its relationship between physical body, energetic matter, and gravitational forces over time. This technique provides an understanding of basic to complex postural biomechanics and takes into consideration the effect of the body to rebalance from above in respect to the "righting reflex." This understanding integrates a model of tensegrity and its relation to anatomy and biology of being. Donald E. Ingber, creator and writer of "Tensegrity and Mechanotransduction," states in his article, "Thus, opposing muscles and bones establish a mechanical force balance and place our entire musculoskeletal system in a state of isometric tension, so that they experience this type of stabilizing pre-stress. Hence, the shape stability of our arm or leg (whether it is stiff or floppy) depends on the level of tension or "tone" in our muscles. Architects call this type of pre-stressed structural network, composed of opposing tension and compression elements that self-stabilizes its shape through establishment of a mechanical force balance, a tensegrity (tensional-integrity) structure."[16]

Another article, printed in the "International Journal of Osteopathic Medicine," made a much simpler definition or explanation to describe tensegrity, stating, "A tensegrity structure as a set of struts under compression, and an arrangement of cables under isometric tension, that always balances in the most energetically efficient configuration." [17] This method combines tensegrity and the tension caused from subluxation as it relates to the short leg, weight, low shoulder or "Bow of the Box", rotation in the shoulders and pelvis, neck and skull tilt. With use of a tensegral model, the patient presented with faulty biomechanics, with respect to the PMD recording, along with x-ray findings; presenting as significant stress on the brainstem and spinal cord as a result of the dentate ligaments, the anterior juxtaposition of the left vagus nerve on the anterior aspect of the left transverse process, as well as, with the right translation of C1 vertebral body. Due to the defective biomechanics seen in analysis, syncope episodes occurring could be a result of the demonstrated display of improper sensory input by pressure received where the vagus nerve and autonomic nervous system lie. Significance regarding this study is the relationship between the vagus nerve and the compression by C1 vertebra, being the main cause of VVS, discussed previously, and the location of the tension generated from improper biomechanics.

Case Management

The patient was assessed using the QSM3 method of approach. This method was applied in an effort to correct the vertebral subluxation by accessing pathways of interference within the body, or distortion of proper function. Mode in application was for the restoration and release of the nervous system to its proper place where optimal function may be obtained. Resultant and following in this paradigm, is the return of joint symmetry, from asymmetry, and muscle stability, from instability. QSM3 is a systematic approach, which takes origin from its upper cervical roots in NUCCA. This chiropractic procedure examines and analyzes a model of 3 dimensional posture, using the PMD; that creates a description and illustration on how trauma is conveyed within the body, and by which a disturbance in tensegrity and orthogonality is produced. QSM3 application is the utilization of the upper cervical area for its location and insertion of these myofascial "tracks". These tracks are designed to most optimally modulate posture due to received trauma, and ultimately result in the production of tensional torsion within

the body. The release of the MFE through the activation of the Golgi Tendon Organ (GTO) response, within the muscle belly of contact, causes a reaction of relaxation. Therefore restoring anatomical balance, thus being the ultimate purpose of the QSM3 doctor and method.[15,18]

Although an upper cervical technique, QSM3 looks beyond just the cervical spine for involvement in a subluxation. Identifying the entire body as one system, that cohesively moves together, makes this a closed kinetic chain relationship. When a disruption of tensegrity, or loss of orthogonality, occurs in a system that has a closed kinetic chain relationship, blood and oxygen is not properly supplied to the area in compression. Deprivation and insufficiency of vital nutrients, to an involved area or areas, leads to the inevitable process understood as "Dis-ease".[15,18,19]

Post-Assessment

After the preliminary analysis is obtained, an integrated algorithm is compiled to formulate a pattern of headpiece placement. QSM3 uses a two positional headpiece algorithm based on the postural pattern, obtained in the pre-assessment. From origin at the feet, lines that modulate posture and generate a tension in compression model, are said to all attach in the upper cervical region, base of the skull and mastoid process. Myers model of myofascial meridians is revolutionizing the way many of us look at the body and is used in application for location of connection in contacting these lines of tension.[20] The principle of the "Anatomy Trains" model is that it shows how individual muscles link together to form functional, myofascial continuities organized along longitudinal lines of pull throughout the body and attach in the upper cervical area.[18] The purpose of the headpiece placement is to isolate, increase, and release tension within specific pathways of the body. With precise patient placement, from skull to short leg, a creation of tension known as "isolated release," can be created and influenced through energy, to most optimize this release. Releasing Myers lines of fascia is made in the adjustment by contacting the Golgi tendon organ (GTO). GTO's are sensory receptors of the peripheral nervous system, and are located at the origins and insertions of tendons within skeletal muscle. The primary purpose of the GTO is to sense tension placed on a muscle, as a protective "negative-feedback system" and to diminish the stimulation of the force generated within the muscle. In other words, the GTO causes a relaxation mechanism to occur, which indirectly releases the compensatory tension and stabilizing lines generated in a misalignment.[15,18]

Outcomes

A chiropractic adjustment was administered to the obtainable subluxation listed between C0-C7 vertebral segments. The Quantum Spinal Mechanics 3 dimensional method and technique was chosen as a means in removing the patient's subluxation. The adjustment was used with the sole purpose in correcting the patient's subluxation, as well as, all and any nerve interference by adjacent anatomic compression. The technique entails and requires the chiropractor to follow a specific protocol that designs a headpiece placement constructed by x-ray analysis and the postural reading. Upon the patient's first adjustment, post assessment was conducted by means of the PMD recording, as well as post x-ray analysis. The patient originally presented with 10 lb.'s on the right side, with an anterior rotation of her right shoulder by 12mm and posterior rotation of her right ilium by 6mm.

Following the initial adjustment, she was replaced on the PMD where an even and symmetrical presentation was concluded as 0.6 lb.'s of weight differential exhibited between right and left anatomical sides. The patient's post rotation of her right shoulder and right ilium were recorded and listed to be both at 0^{o} of translation, in relation to the contralateral side. The patient was further evaluated for a post x-ray positioning where a series of two films, including a post nasium view and a post vertex view, were obtained. (Figure 4) The post nasium view revealed head tilt to the left by zero degrees, with relation to the cervical spine below; and a lower angle displaying less than 1/2 a degree of disassociation between the neck and skull. (Figure 5) The post vertex view was recorded and revealed 3/4 degrees of anterior rotation between atlas (C1) and zero degrees of rotation between axis (C2).

After the initial adjustment, the patient was further consulted and recommended on a care plan consisting of two visits per week, designed for treatment over a 12-month period. Since the patients initial visit to present day, not a single syncope episode or fainting attack has been noted to occur. The patient also claims that following her fourth QSM3 adjustment and office visit, all diffuse and related neck pain completely diminished. The patient is currently seen for QSM3 treatment, as maintenance once a month, and states to be appreciative for the ability in gaining the quality and function back in her life, via chiropractic.

Discussion

Quantum Mechanics

In current times, modern medicine is facing a great burden and upheaval in the effective treatment of present day disease. Older paradigms, once useful in solving the problem of infectious diseases in the past, do not provide adequate solutions for the infinite chronic and degenerative diseases prevalent today. It appears that a new way of thinking, and an alternative way of looking at health and disease is thus, a required action. In modern medicine, Newtonian mechanics are still being applied, using a classic and linear model of cause-and-effect, where a "one disease-one-cure" concept still dominates the model in treatment of disease. Nevertheless, in most branches of science, quantum theory has been accepted, while many in the medical model continue to cling to the cause-effect mentality defined by Newtonian mechanics. Current medicine continues to base its main premise on a concept of lock and key, in which the lock is expressed as disease and the key being the medicine or medical technique (chemotherapy, surgery, etc.) utilized. This linear model in thinking and in application makes implication that each part of the body, as well as, cell, is an independent entity that maybe effectively treated for its independent disturbances and/or disease. However, the human body is complex in nature, not linear in any aspect, and considered to survive as a multicellular being, where cells cannot and do not exist individually. This scientific dogma where symptoms are quantified and used as indicators of specific syndromes has led

to a model aimed toward treating local ailments, rather than observing global structural deficit. In opposition, and for point and purpose of this study, basic premise in the treatment of this patient was with utilization of quantum theory, with expression of the "superposition principle," which suggests that everything is related to and connected with everything else.[21]

From Form to Function

The nervous system is divided into two divisions of central and peripheral systems. Within the peripheral nervous system (PNS), there are two subdivisions including the somatic nervous system (SNS) and the autonomic nervous system (ANS). Of these subdivisions, the ANS is considered to monitor and control most visceral functions of the human body, regulation and maintenance of arterial pressure, heart rate, gastrointestinal motility and secretion, sweating, as well as, control of maintaining body temperature. With respect to the majority of effector organs having full control by the ANS, others effector organs are only partially and moderately within control by the ANS. Nonetheless, the ANS consists not only of efferent motor system involvement, but a mixed relationship with afferent sensory system association, as well.

The autonomic nervous system has two further subdivisions: the sympathetic nervous system and the parasympathetic nervous system. The sympathetic nervous system is considered the "fight or flight" system, where the parasympathetic nervous system is often referred to as the "rest and digest" system. In most situations, both of these systems have opposing actions where a single system activates one physiological response and the other system further inhibits it. Within this understanding, sensory fibers arising from visceral sensory neurons, gather the information from receptors located within the end organs and relay detail to the central nervous system; functioning as a communication and feedback system. This information is further integrated and processed in multi-neuronal pathways consisting within the brain and spinal cord, which in turn can regulate and adjust the autonomic outflow that controls the end-organ.[22] The overall complexity and function of the ANS is to maintain a state of homeostasis within an organism and in ability to adapt when faced with changes in the external and internal environment.[22,23]

The cardiovascular system is subject to precise reflex regulation as means of appropriately supplying oxygenated blood being delivered, and for reliability in providing blood supply to different body tissues under a wide variety of factors and circumstances. The sensory detector for this critical homeostatic process involves two mechanisms: mechanical-barosensory information is primarily responsible in monitoring pressure in the arterial system; and chemical-chemosensory information is accountable in regulating the level of oxygen and carbon dioxide present in the blood. The parasympathetic and sympathetic activity relevant to cardiovascular control is determined by the information supplied by these sensors.[24]

Mechanoreceptors, also known as baroreceptors, are located in the areas of the heart and main blood vessels. The nerve endings located within baroreceptors are stimulated and activated by deformation, monitored by the elastic elements of the vessel walls expanding and contracting. Afferent information from baroreceptors is innervated by large arteries including; carotid sinuses, the aortic arch, and the right subclavian artery. Baroreceptor activity provides the main excitatory influence on cardio-vagal efferent neurons, while under normal resting conditions. A change or increase in blood pressure evokes a further reflex, rendering an increase in cardio-vagal activity, as well as, a corresponding decrease in heart rate. Conversely, cardio-vagal activity is then inhibited and heart rate further increases when blood pressure is diminished or reduced.[25] Chemoreceptors, which are small-distinctly specialized organs, are found within the area of the bifurcation of the common carotid arteries, primarily within the carotid body. Chemoreceptors in the carotid body relay and respond directly to the unbalanced pressure of oxygen and carbon dioxide in the blood. Both afferent-sensory systems convey their information via the vagus nerve to the nucleus of the solitary tract, which further transmits data to the hypothalamus and the relevant brainstem tegmental nuclei.[26]

The vagus nerve (CN X) is the longest of all twelve cranial nerves. The name vagus is derived from Latin denoted and meaning "wandering". In accordance to its name, the vagus nerve wanders, descending from the brain stem and migrating and innervating organs within the neck, thorax and abdomen. The vagus nerve departs from the brain stem, exiting through rootlets in the medulla, that are located caudally to the rootlets for the ninth cranial nerve, glossopharyngeal; and leaving the cranium via the jugular foramen. The vagus nerve consists of five functional components with distinct purpose: 1) special visceral afferent (SVA), 2) general visceral afferent (GVA), 3) general sensory afferent (GSA), 4) special visceral efferent (SVE), and 5) general visceral efferent (GVE). A group of fine rootlets veneer in the medulla within the dorsolateral sulcus, inferior to the glossopharyngeal nerve (CN 9) and just superior to the spinal accessory nerve (CN 11). The rootlets combine, where formation is made of two distinct bundles; a smaller inferior and a larger superior that collectively configure and form the vagus nerve.[27]

Similar to that of glossopharyngeal nerve, two sensory ganglia are also associated with the vagus nerve; known as the superior (jugular) and inferior (nodose) vagal ganglia. The superior ganglion is residence to the cell bodies of pseudo-unipolar first order sensory neurons supplying GSA information from the pinna portion of the inner ear, external auditory meatus, as well as, the dura of the posterior cranial fossa. The inferior ganglion include the pseudo-unipolar first order nerve cell bodies communicating GVA sensory innervation from the mucosa of the soft palate, pharynx, and larynx. SVA pseudo-unipolar neuron cell bodies are also located within the inferior ganglion, where transmission of taste sensation from the epiglottis is made. Both the SVA and GVA pseudo-unipolar neurons enter the brainstem along their course in the solitary tract where both terminate in the solitary nucleus. GSA pseudo-unipolar neuron cells central processes enter the brainstem, where they join the spinal tract of the fifth cranial nerve and further terminate in the spinal nucleus of the trigeminal nerve (CN 5). The SVE, also known as, branchial efferent or brachio-motor neurons, originate from a group of large motor neurons, situated deep within the medullary reticular formation in the nucleus ambiguus. The fibers of

these neurons innervate all the muscles of the larynx and pharynx; with the exception of the stylopharyngeus and the tensor veli palatini muscles. The visceromotor component, GVE, of the vagus nerve originates from the dorsal motor nucleus of the vagus within the dorsal medulla; housing the nerve cell bodies of preganglionic parasympathetic neurons. These fibers join and accompany the other vagal fibers upon their decent from the brainstem, where they run in the main trunk of the vagus into the thorax. From there these fibers then leave the main trunk and join the autonomic plexuses distributing throughout the thoracic and abdominal cavities. The preganglionic fibers terminate and synapse in the terminal parasympathetic ganglia or ganglia surrounding or within the viscera.[27]

Parasympathetic innervation causes a decrease in heart rate, a reduction in adrenal gland secretion, stimulates peristalsis, as well as, activates glandular activity of various organs. Parasympathetic mediated changes in heart rate are initiated mainly within the central nervous system or from stimulation and/or inhibition of afferent nerves. Afferent stimulus of arterial baroreceptors, trigeminal receptors, peripheral chemoreceptors,, and branches of cardiopulmonary receptors with vagal sensory feed, reflexively cause an increase in cardiovagal activity and decrease in heart rate.

Alternatively, sensory stimulus to pulmonary stretch receptors with vagal afferent influence, as well as, somatic and visceral receptors with spinal sensory stimuli reflexively cause a decrease in cardiovagal activity and an increase in resting heart rate. Sensory or afferent information from changes in arterial pressure and alterations in blood gas levels, reflexively modulate the action of the related visceral efferent pathways and, fundamentally, of targeted smooth and cardiac muscle, as well as, other specialized structures. By way of illustration and for example, an increase in blood pressure stimulates and activates baroreceptors, indirectly causing inhibition of the tonic activity of sympathetic preganglionic neurons located within the spinal cord.

In correspondence, a rise in pressure stimulates the activity of parasympathetic preganglionic neurons in the dorsal motor nucleus of the vagus nerve and the nucleus ambiguus, influencing heart rate. This transition and shift in balance of sympathetic and parasympathetic activity, results in the stimulatory noradrenergic effects of postganglionic sympathetic innervation on the cardiac pacemaker and cardiac musculature being reduced; an effect contributed to the diminished output of catecholamines from the adrenal medulla and decreased vasoconstrictive effects of sympathetic supply to peripheral blood vessels. In concurrence, stimulation on cholinergic parasympathetic innervation of the heart causes a decrease in the discharge rate of the cardiac pacemaker (sinoatrial node), resulting in inactivity of the ventricular conducting system. An extensive series of parasympathetic ganglia within and around the heart mediate these parasympathetic responses, influencing the release of acetylcholine to receptor cells of the cardiac pacemaker and cardiac muscle fibers. As a result of integration of sympathetic and parasympathetic influence, the effectiveness of the atrial and ventricular myocardial contraction is reduced, as well as, a reduction in heart rate and dilation of the peripheral arterioles; therefore lowering the blood pressure.[25,26]

Tension out of Integrity

The human body has a remarkable capacity to maintain a stable blood pressure in the presence of constant changing forces that continuously shift and redistribute the circulation and volume of blood. In effort to achieve this stable control, reflex mechanisms continuously monitor and adjust the cardiac output, as well as, vascular tone. Changes as simple as modulating posture, such as going from sit to stand, have potential to result in a relatively unfilled ventricle, due to the shifting in blood from the thorax to abdomen and lower extremities. This shift in blood volume can markedly decrease the cardiac output, which is typically sensed by arterial baroreceptors located within the carotid sinus and aortic arch. The receptors in these locations further propagate signals to the nervous system, where they are integrated and modulated via reflex, increasing sympathetic output. Furthermore, the vascular system reciprocates by restricting local blood flow to organs deemed non-vital such as the skin, adipose tissue, and musculoskeletal system; in effort of increasing peripheral resistance. Diagnostically, this reaction exhibits as an increase in heart rate, between a range of 10 to 15 beats per min, which is considered to be mediated by escalated sympathetic output; as well as, probable mediated local vasoconstriction, yielding a gradual increases in diastolic-pressure, within a range of about 10 mmHg.[28]

Vasovagal syncope is caused by "hypersensitivity" of the autonomic nervous system (ANS), which over responds to a multitude of divergent stimuli. Orthostatic stress is one of the most frequently encountered precipitating factors seen in clinical practice. According to the 'Texas Heart Institute Journal, when associated to orthostatic stress, syncope is considered to involve the subsequent steps: "1) The heart is partially emptied as a result of a fluid shift. 2) Activation of the above-described normal sympathetic reflex response results in hypercontractility of the ventricle in an attempt to increase the cardiac output. 3) Cardiac mechanoreceptors, which are usually activated by distension of the heart (for example, in conditions involving severe hypertension), undergo abnormal stimulation. This paradoxical stimulation is believed to result from the combined hypercontractility and emptiness of the ventricle. 4) Abnormal mechanoreceptor stimulation transmits neural signals, as the afferent limb of the abnormal reflex, to the tractus nucleus solitarius in the brainstem, via the vagus nerve. 5) The tractus nucleus solitarius synapses with other centers in the brainstem, which are not clearly understood but may be located in the rostral ventro-lateral medulla. 6) Through the efferent limb of this reflex, the para- sympathetic output is increased, and sympathetic output is inhibited, resulting in bradycardia, hypotension, and syncope."[28] The action most pertinent to this case study in presenting to render a syncope episode, is step number four.

Philosophy

The conception of "subluxation" has been the basis of theory and practice of chiropractic since its founding by D.D. Palmer in 1895. D.D Palmer defined a subluxation to be as follows: "A (sub)luxation of a joint, to a Chiropractor, means pressure on nerves, abnormal functions creating a lesion in some portion of the body, either in its action, or makeup."[29]

Currently in health care today, the concept of subluxation remains to be extremely controversial, with supporters and critics present within and outside the chiropractic profession. Originally, the concept of subluxation was said to be "that of a slightly misaligned vertebra, not sufficient to be qualified as a true luxation or dislocation but substantial enough to impinge on the segmental nerves associated with it."[30] While this primary concept requires minor modification in regards to current research findings, there has also been an abundance, within the past two decades, in the accumulation and validity supporting the concept of vertebral subluxation as a justifiable entity. Furthermore, it must be mentioned that from the contemporary and scientific chiropractic understanding, the subluxation is a dynamic occurrence and process involving various tissue levels and integrative components.[29,30]

Palmer founded what further became the chiropractic profession on the premise and importance of neurospinal influence upon physiology; citing the nervous system as automatic functions, which is now referenced to as the ANS. Published in 1954 and entitled, "Autonomics in Chiropractic" reference was again mentioned on the importance of the ANS to the chiropractic profession. In Müller's text a statement was made claiming, "The essential role of the autonomic nervous system as an integrator and controller of body functions is a fact all are agreed upon. That structure or function is disturbed, sometimes seriously, however this correlation is deranged from any cause is becoming more widely recognized by all schools of healing. It is the very bedrock upon which the premise of chiropractic is based."[31] More recently, trials conducted with intensive supplementary studies and advanced neurophysiology research, have been concluded on such topics concerning the essential interaction between the musculoskeletal system and the autonomic nervous system.[31]

A variety of hypotheses exist on the relationship between the structural spine and interconnected neural dysfunction; ranging from involvement of immense irritation due to bombardment of noxious mechanoreceptors brought on by localized pathomechanics, to aggravation and inflammation responding at a radicular level. In regards to segmental neurospinal dysfunction, appreciation and for the purpose of this paper, a vertebral subluxation is said to not be limited as just a literal mechanical displacement of a vertebra. But more presently, as well as, accurately termed a 'vertebral subluxation complex' (VSC), involving elements including those of structural segments influencing functional components.[31]

According to an article published by the Chiropractic Journal of Australia (CJA), a significant component of this complex is contributed to dysfunction within the mechanics of articulations intersegmentally, stating "This may comprise aberrant movement, fixation (hypomobility) or hypermobility between adjacent facets, as well as articular muscular and ligamentous changes triggering neural firing of mechanoreceptors, proprioceptors, effectively nociceptive noxious input. The VSC would then include disturbances of these structures and their function, especially their effect upon articular physiology (function) and the integral neurophysiology. Inflammatory and circulatory disturbances of the articular environment could also be associated. It is this total pathophysiological complex that would provide the opportunity through which manual intervention by way of a vertebral adjustment may be directed in order to influence internal body physiology. It is submitted again that segmental dysfunction more than osseous displacement, may be the primary physical-mechanical feature involving any associated neural aberration in this situation but, that is only one part of the complex. Only a dry skeleton could have osseous disruption without more complex involvement."[31]

By incorporating Kent's models of vertebral subluxations, the dysafferentation model can be used to propose a theory on how presence of an upper cervical vertebral subluxation regarding this presenting case, resulted in the relief of symptoms relating to VVS. Kent explains, "as a consequence, biomechanical dysfunction may result in an alteration in normal nociception and/or mechanoreception." These alterations may result in postural abnormalities affecting tone. Furthermore, it is believed that "correcting the specific vertebral subluxation cause is paramount to restoring normal afferent input to the CNS, and allowing the body to correctly perceive itself and its environment."[29]

Biomechanical Consideration

"Life is the expression of tone. In that sentence is the basic principle of Chiropractic. Tone is the normal degree of nerve tension. Tone is expressed in functions by normal elasticity, activity, strength and excitability of the various organs, as observed in a state of health. Consequently, the cause of disease is any variation of tone—nerves too tense or too slack."[32]

This study encourages the need for further research to substantiate this theory. Quantum Spinal Mechanics[3] (QSM[3]) is a tonal-based upper cervical technique evaluating the importance of correcting postural imbalances in the body, causing loss of tensegrity and orthogonality. Pulling forces produced by the fascia that surround and infiltrate all structures of the body from the head down to the feet, instigate these postural imbalances. Kuma and Bonar define fascia as an "uninterrupted viscoelastic tissue, which forms a functional 3-dimensional collagen matrix."[33] Structurally, "tensegrity depends on the integration of every part, as it has been proposed that this includes the whole body, from molecules, cells, extra-cellular and fascial matrix to the entire musculo-skeletal system.[17] Functionally, the myofascial envelope (MFE) plays a critical role in regulation of proprioception.[33]

A study performed by Kumka and Bonar, classified fascia according to their function and morphological characteristics. The classification system is divided into four categories: 1) linking, 2) fascicular, 3) compression, and 4) separating fasciae.[33] Generally, the adjustment delivered by QSM[3] addresses all categories, but most specifically the fascicular component. The fascicular fascia "forms adaptable tunnels which bundle vessels as well as fascicles within muscle, tendon, bone and nerves." This type of fascia is critical for "organization, transport, strength and locomotion," covering extramuscular, intramuscular and neurovascular sheaths. Fascicular fascia is mostly composed of collagen Types I and III and are organized as a mixture of both loose and dense regular multidirectional connective tissues.[33]

Kumka and Bonar explain, "The fascicular fascia of the muscle converges into a dense regular connective tissue link at the myotendinous junction to become fascicular fascia of the tendon, comprising of endotendon, peritendon and epitendon." This area is heavily enriched by Golgi tendon organs (GTO), which are activated by muscle contraction. Muscle contraction results in tension placed in the GTO's, causing a "reflex decrease in tonus in contiguous striated muscle fibers." [33] Activation of this reflex is the framework utilized by QSM[3] technique. By stimulating the GTO's located around the mastoid, ear, mandibular angle and the upper cervical region, muscle relaxation can occur by fatiguing the side of contraction or compression. Ultimately, this places the body in an upright position restoring tensegrity and orthogonality.[15,34]

Trauma to the human structure inevitably causes postural ramifications, that within this closed kinetic change, are expressed as force generated and altered between the skeletal framework of fascia, connecting pelvis to upper cervical spine and skull. Change in the centre of gravity will indefinitely produce undesirable stresses on structures within the tissues of the cervical spine. This situation of position has vital implications for the spinal cord, which leaves via the foramen magnum, which passes through the foramen (holes) of the atlas vertebra (C1) and other skeletal vertebrae to the base of the vertebral spine. This also has important ramifications pertaining to the vertebral arteries, which travel through the cervical spine from C6 to C1 vertebra; for the carotid arteries, which pass beneath the sternocleidomastoid muscles (SCM); as well as, for the cranial nerves exiting at the level of the foramen, located at the base of the skull. All of these structures listed above, share equal potential in being trapped, compressed, stretched or otherwise interfered with by the taught muscles, as well as, ligaments involved in maintaining the skull and its constituents on top of the cervical spine. Consequently and as a result, stresses on arteries yield potential reduction in production and distribution of oxygenated blood flow to the brain; as well as, stress on cranial nerves could potentially result in attenuation of nervous system signals to and from the brain. The cranial nerves most vulnerable to these stressors and which are potentially involved consist of the vagus (CN X), spinal accessory (CN XI), glossopharyngeal (CN IX) and hypoglossal nerves (CN XII).[35-37]

Vulnerability of these arteries and nerves, listed above is of concern due to their responsibility and major importance in providing proper function and maintenance of the human body. With respect to the cardiovascular system, which is regulated by the autonomic nervous system (vagus nerve), as well as, baroceptors located within the tissues; stressors, such as compression, on vessels, nerves or baroreceptors by means of muscular contracture, and/or an Atlas misalignment, directly can influence and yield a variety of symptoms, such as: irregular blood pressure, hypertension or hypotension, irregularities of the circulatory system or alterations of the cardiac rhythm, increase in the cardiac frequency and decrease in the capacity of the cardiovascular system. Chiropractic literature and research based upper cervical care, have shown to benefit other neurological conditions and deficits produced by spinal misalignments found in the C1 or C2 regions. Although definitive treatments for VVS does not currently exist, the dentate ligament-cord distortion hypothesis can suggest a mechanism to explain the benefits in receiving upper cervical adjustments to relieve episodes of syncope.[15,37]

John Grostic DC, one of the founding fathers of upper cervical based chiropractic offered an explanation as to how alterations of the upper cervical spine can lead to neuropathic disorders produced by mechanical irritation of the spinal cord. Studies performed on the role of the dentate ligament have demonstrated that its connective tissue properties are strong enough to even cause slight spinal cord deformation, especially in cervical flexion. Grostic's approach to this hypothesis had led him to discover that alteration in the biomechanics of the upper cervical spine can ultimately lead to deformation of the spinal cord; anatomically considering the dentate ligament offers an attachment of the upper cervical spine to the spinal cord. He suggested, "If misalignments of the cervical spine are to affect the spinal cord a mechanical linkage must exist between the osseous structures and the spinal cord."[38]

Continuing this further, a measurable difference of 30 mm in the length of the spinal canal occurs while performing cervical flexion and extension, placing longitudinal stretching and longitudinal compression on the cord, respectively. By observing this mechanical alteration, it is believed that dentate ligaments found in upper cervical region provide a critical role in the restriction of "downward pulling axial forces created by the lengthening of the canal when the neck is flexed from being transmitted unattenuated to the brainstem." Although this serves as a form of protection during normal movements, alterations of the cervical vertebrae can cause pathological forces to the spinal cord and brainstem.[38]

According to the CJA, a growing volume of elucidating scientific research on spine related neuro-autonomic conditions have recently surfaced and further outlined in origin, stating, "observations of the striking influence of postural mechanics on function and symptomatology have led to our hypothesis that posture affects and moderates every physiologic function from breathing to hormonal production. Spinal pain, headache, mood, blood pressure, pulse, and lung capacity are among the functions most easily influenced by posture. The most significant influences of posture are upon respiration, oxygenation, and sympathetic function. Ultimately, it appears that homeostasis and autonomic regulation are intimately connected with posture."[31] The article further elaborates, making claim, "that repetitive stimulation of small myelinated and unmyelinated somatic afferents can dramatically increase reflex pre- and post-ganglionic sympathetic discharge. Studies suggest that the alteration of afferent articular input due to joint dysfunction and nociception excitation, in conditions of noxious mechanical deformation or chemical irritation, may provoke significant changes in efferent and autonomic responses."[31]

The hypothesis proposed by Grostic suggests an explanation as to why patients exhibiting signs of neurological compromise may be as a result of certain structural abnormalities within the cervical region.[31,38] With this appreciation and understanding of how structure dictates function, chiropractic corrective treatment was shown to be effective in relieving episodes of syncope in a patient suffering from VVS.

Conclusion

The chiropractic treatment and complete resolution of a 57-year-old female patient suffering from Vasovagal syncope (VVS) is presented in this case study. Comprehensive improvement and complete alleviation of symptomatology were achieved and recorded upon the administering of QSM3 chiropractic treatment, all presented in this study. This case supports the evidence of chiropractic care in relieving neurological disorders based on anatomical association and interference. There are limitations present in every case study, incorporating and including this one. This case advocates the underlying importance in the conducting additional research on the benefits of tonal based upper cervical chiropractic techniques, treating disorders of neural and cardiac origin with underlying structural relation.

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Figures

Preliminary Cervical X-Rays

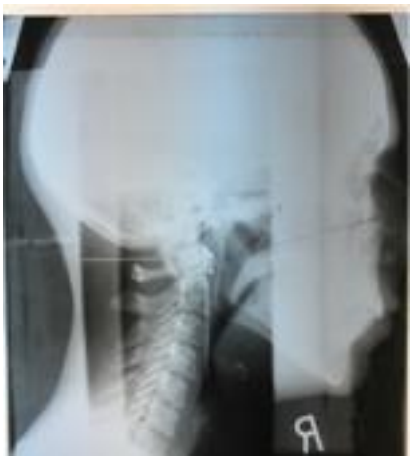

Figure 1 Lateral

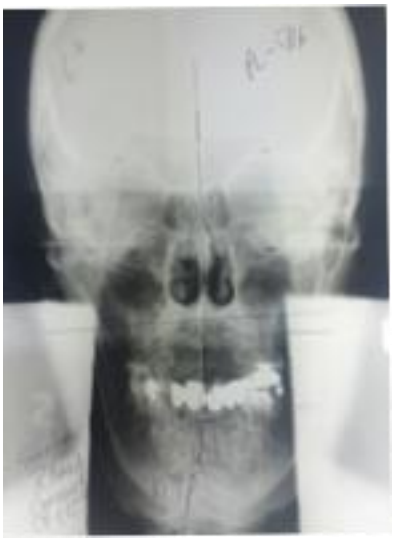

Figure 2 Nasium

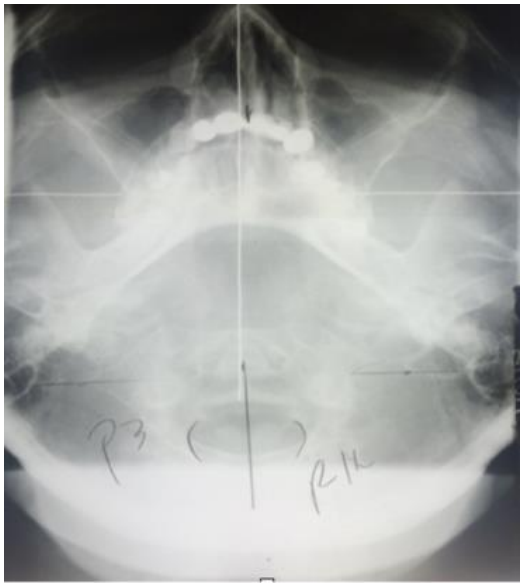

Figure 3 Vertex

Post Cervical X-Rays

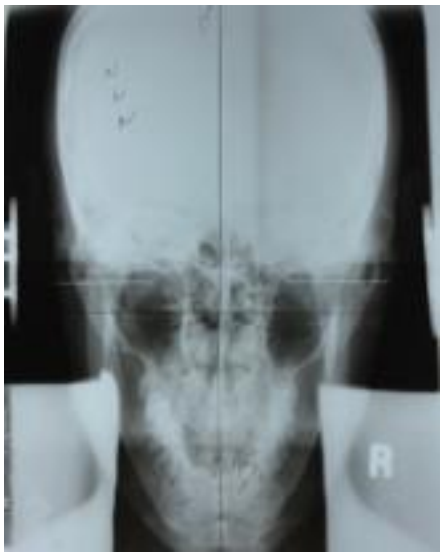

Figure 4 Nasium

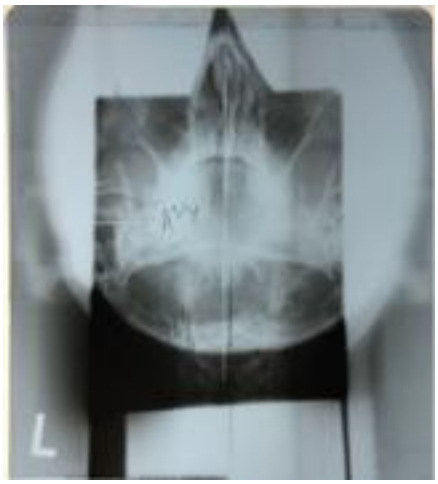

Figure 5 Vertex