Case Study

Resolution of Hypothyroidism & Irritable Bowel Syndrome in a 34-Year-Old Female Following Chiropractic Care to Reduce Vertebral Subluxation: A Case Study & Review of the Literature

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Abstract

Objective: To report on the case of a 34 year old female with secondary hypothyroidism and irritable bowel syndrome (IBS) experiencing improvement following chiropractic care.

Clinical Features: A 34-year-old female presented to a chiropractic office complaining of low back, hip, and upper back pain. She also reported that she was medically diagnosed with irritable bowel syndrome and secondary hypothyroidism.

Intervention and Outcome: Rolling paraspinal thermography was used to localize and quantify Dysautonomia secondary to subluxation. Torque Release Technique (TRT) was utilized to address areas of vertebral subluxation. After two months of care, the patient reported improvement in thyroid function determined by a thyroid function test and she was able to stop her medication. Additionally, the patient reported improvement in her irritable bowel syndrome, low back, hip and upper back pain.

Conclusion: This case study provides supporting evidence that chiropractic care may contribute to improvement in overall thyroid and gastrointestinal function. Additional research is advised to explore the benefits of chiropractic management in patients with hypothyroidism, IBS and dysautonomia.

Key Words: *Chiropractic, subluxation, vertebral subluxation, Torque Release Technique, TRT, hypothyroidism, secondary hypothyroidism, adjustment, irritable bowel syndrome, dysautonomia*

Introduction

The purpose of this paper is to report on the case of a 34 year old female with secondary hypothyroidism and irritable bowel syndrome experiencing improvement following chiropractic care. Hypothyroidism is a common endocrine disorder that is characterized by a deficiency in thyroid hormone.[1-3] In 2011, it was estimated that hypothyroidism affects approximately 10% of women and 6% of men under the age of 65 in the United States.[1] This is in comparison to the National Health and Nutrition Examination Survey (NHANES III) from 1999-2002, about 1 in 300 people, approximately 3.7% of the general population, are affected by hypothyroidism.[3-5] The prevalence of hypothyroidism is steadily increasing and remains a burden on the general population. It is found to be 2-8 times more common in women than men and has a higher incidence in Caucasian and Mexican-Americans compared to African Americans.[3]

Etiology

Hypothyroidism is classified as congenital or acquired and then categorized by primary or secondary types.[3] Primary hypothyroidism is defined as thyroid gland dysfunction.[1-3,6-8] This is characterized by increased levels of thyroid-stimulating hormone (TSH) and decreased levels of thyroid hormones T3 and T4.[1-3,6-8] Common causes of primary hypothyroidism are iodine deficiency, autoimmune disease also known as Hashimoto's Thyroiditis, surgical or radiation induced, or infection.[1,6] Secondary or central hypothyroidism is defined by dysfunction of the hypothalamus or anterior pituitary. This is characterized by low, normal, or mildly elevated TSH secretion and a decrease in thyroid hormones triiodothyronine (T3) and thyroxine (T4) production and secretion.[1-3,6-8] Common causes of secondary hypothyroidism includes pituitary or hypothalamus adenoma, as well as surgery and radiation used to treat the adenoma, gene mutation, and Sheehan Syndrome.[7,9]

Anatomy and Pathophysiology

The thyroid is a bilobulated gland that is found in the anterior portion of the neck and is situated anteriorly and around the trachea.[2,8] The thyroid gland consists of follicle cells that are filled with a secretory substance called colloid. Colloid contains thyroglobulin, a glycoprotein.[8] Thyroglobulin is involved in the synthesis and storage of thyroid hormones.[8] Thyroid hormones include tetraiodothyronine, also known as thyroxine or T4, and triiodothyronine, also known as T3. T3 and T4 play a major role in the metabolic rate of the body.[2,8] Additionally, the thyroid gland contains parafollicular cells, which secrete calcitonin. Calcitonin plays an important role in regulating plasma calcium levels.[2]

Synthesis of thyroid hormones is a complex mechanism of chemical reactions.[2,8] Production is largely dependent on the ingestion of iodine. It is suggested that 50 milligrams of iodine should be ingested per year.[2] To prevent iodine deficiency, the United States has fortified common table salt with sodium iodide.[2,3,8] The first step in production of thyroid hormones is called "iodide trapping."[2] This is the process of taking iodide from the blood into the follicular cells via an active pump mechanism. Through a series of chemical reactions, hormones T3 and T4 are formed.[2] The thyroid gland has the ability to synthesize and store molecules of T3 and T4.[2,8] It is estimated that 93% of active thyroid hormone secreted by the thyroid gland is in the form of T4.[2] The majority of T4 is converted into T3 once it reaches the target tissue such as the liver, kidneys, pituitary gland, various centers of the brain, and the thyroid gland itself.[8] T3 is four times more potent than T4, therefore it is found in lower quantities in the plasma in comparison to its target tissue.[2,8] Thyroid hormones are transported through the blood while bound to plasma proteins. Approximately 99% of thyroid hormone is bound to thyroxine-binding globulin (TBG), thyroxine-binding albumin (TBA), and thyroxine-binding prealbumin (TBPA). 1% of thyroid hormones are free, unbound hormone that is biologically active.[8]

Neuroendocrine regulation of the thyroid gland is controlled via the hypothalamic-pituitary-thyroid (HPT) axis.[2,8] Three structures regulate the production and secretion of thyroid hormones—the hypothalamus, the anterior pituitary, and the thyroid gland.[2,8] The hypothalamus secretes a hormone called thyrotrophin-releasing hormone (TRH). TRH is secreted by nerve endings from the hypothalamus and transported to the anterior pituitary via the hypothalamic-hypophysial portal system.[2] TRH then stimulates the anterior pituitary to secrete thyroid-stimulating hormone (TSH).[2,8] TSH therefore stimulates the thyroid gland to produce and secrete thyroid hormones in the form of T3 and T4. Regulation of thyroid hormone production is by a negative feedback mechanism where secretion of T3 and T4 inhibit the production of TSH and TRH (see figure 1).[2,8]

Thyroid hormones have an array of effects on the body. The main effect is to increase basal metabolic rate. This includes an increase in carbohydrate metabolism and synthesis, protein synthesis, and degradation of lipids.[2,8] In addition, thyroid hormones are necessary for myelination of nerve fibers and central nervous system (CNS) development, increased number and activity of mitochondria, assist in skeletal growth and normal muscle function. As a result of thyroid hormones control on metabolism, cardiac output, heart rate, blood flow, and respiration are also affected. Gastrointestinal mobility, sleep, sexual function, and control of other endocrine organs are all dependent on thyroid hormone production.[2,8]

Due to the extensive effects thyroid hormones have on basal metabolic rate, a hypoactive thyroid gland can produce a wide variety of symptoms. The thyroid gland can store T3 and T4 for up to four months creating a slow onset of signs and symptoms.[8] There is a decrease in heart rate, respiration rate, blood volume, and body temperature. Cold intolerance and decreased perspiration is often noted, as well as weight gain.[2,3,8] There is decreased neuronal function causing symptoms such as fatigue and loss of energy, inability to concentrate, forgetfulness, hyporeflexia, muscle fatigue, weakness, and joint pain.[3,8] Many people sleep on average for twelve to fourteen hours a day and begin to develop signs of depression.[2] There may be puffiness in the face, dry skin and hair loss. A hallmark trait of hypothyroidism is myxedema. Myxedema is a form of edema due to an accumulation of proteins, polysaccharides, and hyaluronic acid from decreased metabolism.[3,8] Hashimoto's Thyroiditis, a form of primary hypothyroidism, often has symptoms of fullness in the throat and deepening of the voice due to hyperplasia of the thyroid gland.[2,3] Hypoactivity of the thyroid gland also affects the functioning of other endocrine organs. Hypothyroidism can cause male and female infertility, menorrhagia or irregular menstrual cycles.[2,3,8]

Primary hypothyroidism is more common than secondary hypothyroidism.[2,3,7] There are two main causes of primary hypothyroidism. Worldwide, the most common cause of primary hypothyroidism is iodine deficiency.[2] Inadequate intake of iodine prevents the production of T3 and T4 by the thyroid gland. Consequently, the anterior pituitary continues to produce high levels of TSH. TSH stimulates the production of thyroglobulin in the follicular cells of the thyroid but without iodine, T3 and T4 cannot be synthesized.[2,8] In addition, there is no negative feedback cycle elicited from T3 and T4 on TSH production. The follicular cells continue to grow bigger due to over production of thyroglobulin, which in turn causes the thyroid gland to enlarge.[2,8] Enlargement of the thyroid gland is called a goiter and can grow ten to twenty times its normal size.[2]

An article by Vanderpump states that in areas of severe iodine deficiency such as South-East Asia, Latin America, and Central Africa, the prevalence of a goiter is 80%.[10] According to the Merck Manual, in areas with adequate iodine intake, the most common cause of primary hypothyroidism is autoimmune thyroiditis, also known as Hashimoto's Thyroiditis.[1] Hashimoto's thyroiditis is caused by a cell-mediated immune response that destroys the follicular cells of the thyroid.[2,8] Another possible mechanism results from the production of antithyroid antibodies that block the TSH receptor on the thyroid.[8] Vanderpump states that post-mortem studies of people with hypothyroidism showed 27% of women and 7% of men with signs of Hashimoto's Thyroiditis.[10]

Secondary hypothyroidism is a characterized by a defect in thyroid hormone production due to insufficient stimulation by TSH.[11-13,9] A pituitary ademona is the most common cause of secondary hypothyroidism.[9,13] The adenoma mechanically compresses the portal vessels of the pituitary stalk of the pituitary gland. This can result in ischemic necrosis of the anterior pituitary.[9,13] In this case, the anterior pituitary does not secrete a sufficient amount of TSH and therefore does not stimulate the thyroid gland to secrete T3 and T4. The adenoma can also create increased intrasellar pressure leading to compression of the portal veins of the pituitary, impairing the delivery of TRH from the hypothalamus.[9] Another proposed mechanism depicts a defect in the "secreted TSH isoforms that conserve immunoreactivity but display a severe impairment in intrinsic bioactivity and ability to stimulate the TSH receptors."[11] Glycosylation plays an important role in altering the biologic activity of molecules. An error in glycosylation of TSH results in varied forms of bioactivity of TSH.[11] The secretion of bioinactive TSH affects the hypothalamic function, which leads to normal or slightly elevated TSH levels upon testing.[11]

Diagnosis

Diagnosis of hypothyroidism is based on a thorough history, clinical findings and laboratory analysis.[14] If hypothyroidism is suspected, initial plasma screening is used to perform a third-generation TSH assay and thyroid function test.[3,15] The accepted reference range for normal TSH levels is 0.4-4.2 mIU/L.[3] The accepted reference range for free T4 is 10.0-21.0 pmol/L, and the normal range for free T3 is 2.2-5.3 pmol/L.[16] The American Thyroid Association states that free T4 analysis is important in determining overall thyroid function while free T3 is important in determining hyperthyroidism.[17] Plasma T4 is found in larger quantities and is eventually converted to T3 in the target tissue.[8] In the instance of primary hypothyroidism, TSH levels are elevated while there are low levels of T4. TSH levels are often found to be above 10 mIU/L in primary hypothyroidism.[5,15,17,18] In the case of secondary hypothyroidism, low TSH, free T3, and free T4 levels are observed. [5,15,17,18] This represents dysfunction of the pituitary gland, preventing adequate TSH secretion. Inadequate TSH secretion then creates inadequate T3 and T4 secretion from an otherwise normally functioning thyroid gland.[18]

Medical Treatment

The treatment for hypothyroidism follows a biomedical approach in which the gold standard treatment is supplementation of once daily synthetic thyroxine (T4).[5,15,18] The most common supplementation is with Levothyroxine for an indefinite period of time.[18] The goal is to improve the function of the patient and to restore appropriate TSH and T4 levels. However, there is evidence to suggest that this is an inadequate form of treatment. Saravanan, Chau, Roberts, Vedhara, Greenwood, and Dayan state that there are many people suffering from hypothyroidism that continue to experience symptoms despite synthetic thyroxine treatment.[19]

Case Report

Patient History

A 34-year-old female presented to a chiropractic office with chief complaints of low back and hip pain and additional an complaint of upper back pain. At the time of presentation, the patient reported that she was twenty-two weeks pregnant. The low back and hip pain started three months prior to seeking chiropractic care. When asked to locate her pain, the patient pointed to the midline of L2-S1 and described the pain shooting into her left leg. The patient self-managed the pain with aspirin and did not seek previous care for her complaints.

The patient's history included self-diagnosed chronic sinusitis, irritable bowel syndrome medically diagnosed in 2013, and secondary hypothyroidism medically diagnosed in 2014. The patient was prescribed Nature-Throid by her medical doctor in 2014. The patient stated that she exhibited symptoms such as fatigue, weight gain, dry skin, sleepiness, and constipation, which lead her to seek medical treatment. Her medical doctor performed a thyroid function test and diagnosed her with secondary hypothyroidism.

Areas of stress were also identified. The patient's job was a major area of stress due to excessive sitting and a busy schedule. At the time of the initial visit the patient was twenty-two weeks pregnant, which added additional physical, mental, and emotional stress.

Examination

Observations in the patient's posture revealed anterior head translation, superior elevation of the right shoulder and superior elevation of the left iliac crest. Cervical active range of motion revealed a decrease in right rotation without pain. All other cervical ranges of motion, both passive and active, were painless and within normal limits. Lumbar range of motion revealed a decrease in flexion and the patient verbally rated pain as 3/10 on a pain scale of 0-10. All other active and passive lumbar ranges of motion were painless and within normal limits.

Static palpatory findings revealed taut muscle fibers at C1, along the thoracic and lumbar paraspinal musculature, sacrum, and the left sacroiliac joint. Upon palpation, tenderness was noted at C1, T1-T2, T8, and L5 vertebral segments. Using motion palpation technique, a decreased end range of motion was felt at C1, T1, T2, T8, L5, and left sacroiliac joint.

At the time of initial visit, the patient was pregnant and no radiographs were taken. Four months after care and delivery of the baby, a cervical radiograph series was taken and revealed a kyphotic cervical curvature and a posterior ponticle. A lumbar radiograph series was also taken and revealed slight hyperlordosis in the lumbar curvature.

Rolling paraspinal thermography was utilized in the evaluation of the patient's nervous system. Thermography readings compare skin temperature variations relating to localization of blood flow.[20] Abnormal thermography readings directly correlate with dysfunction in the neuromusculoskeletal system in the form of dysautonomia.[20] In a healthy patient, the autonomic nervous system effectively controls temperature regulation through dilation and constriction of blood vessels. These changes of temperature within the body occur as the autonomic nervous system works with the circulatory system to dilate and constrict the blood vessels to keep the surface temperature within a manageable range.[20] When vertebral subluxations are present, the normally symmetrical temperature gradients develop an asymmetrical pattern.[20] rolling thermography is used to determine the amount of dysautonomia, the ability of the patient to adapt to the environment, and the response of the patient to the adjustment.[20,21] Rolling thermography is a reliable tool to assess the function of the autonomic nervous system and is shown to have excellent intra-examinar and inter-examiner reproducibility.[22-24]

At the initial visit, the patient's thermography scan was determined invalid due to the patient having just had coffee and taken medication. A new scan was taken seven days later and demonstrated a severe heat abnormality at C2 and mild heat abnormalities at T11 and T12. See figure 2A.

The Quadruple Visual Analog Scale (QVAS) was used as a subjective indicator to record and measure the progress of pain. The score is averaged to determine the percentage of pain the patient is in. The score is categorized into low intensity (pain <50) and high intensity (pain >50).[25] The patient's QVAS score on the initial visit was 43%.

According to physical exam findings and assessment of the rolling thermography scan, subluxations were found in the cervical, thoracic, lumbar spine, and pelvis. Reassessments were given after one, two, four, and eight months of care. A health questionnaire, QVAS, and thermography were used to monitor progress.

Interventions and Outcomes

Over the course of eight months, the patient was adjusted forty-eight times utilizing Torque Release Technique (TRT) with adjustments administered with the Integrator instrument. On the first visit, C1, T1, and sacrum were adjusted. Each visit two-three segments were adjusted. In particular, C1 was the most commonly adjusted segment; other commonly adjusted segments included C5, T1, L5, and sacrum. The segments that were adjusted varied based upon indicators of subluxation discussed below.

TRT utilizes a neurological non-linear tonal model to identify indicators of primary vertebral subluxations and classifies subluxations into testing priorities.[26] It was developed by Jay Holder, D.C from a randomized clinical trial conducted in Miami, FL. The clinical trial was designed to measure the outcomes of subluxation-based chiropractic on the quality of life.[27]

TRT uses the Integrator instrument to deliver a specific chiropractic adjustment. The Integrator was designed for Holder's clinical trial to "ensure consistency and reproducibility in the application of delivering the chiropractic adjustment and to measure its outcome" in the study.[28] The Integrator is designed to simulate a Toggle Recoil adjustment and "fires at the speed of 1/10,000 of a second while providing a three-dimensional impulse using straight axial, right or left torque."[26] There is a cocking mechanism that allows the practitioner to pre-load the instrument with a pre-determined force. Holder states he designed a "pressure sensitive pisiform tip with an automatic release mechanism for the purpose of delivering thrusts with true intraprofessional reproducibility at a constant Hertz frequency."[28] Additionally, the Integrator "is the first chiropractic adjusting instrument to receive an FDA 510K Class II medical device designation for the adjustment of vertebral subluxation."[26-28] It is the only instrument that is capable of delivering a three dimensional adjustment that fires independent of the practitioner.[26]

TRT is a non-linear tonal model that is organized according to the cranio-sacral meningeal functional unit (C-SMFU). The C-SMFU consists of "the brain, spinal cord, multilayered meningeal sheath, bones of the cranium, vertebral column, and pelvis" as they all function as one unit.[26] TRT uses fifteen indicators of subluxation in order to detect a primary subluxation. The indicators include: static, tissue, intersegmental and motion palpation, functional leg length reflex (FLLR), abductor tendency/adductor resistance, foot flare, foot supination/pronation, heel tension, abnormal breathing patterns, inappropriate sustained patterns of paraspinal contractions, congestive tissue tone, postural faults, Cervical Syndrome Test, Wrong-Un test, Bilateral Cervical Syndrome, Derifield test, and abnormal heat and energy radiation from the body.[26] Table 1 lists each indicator and what that indicator means.

TRT uses a functional leg length reflex (FLLR) and pressure tests in order to find the primary subluxation. While the patient is in the prone position, an abrupt bilateral foot dorsiflexion is used to generate a bilateral deep tendon Achilles reflex. This is used to analyze functional leg length inequality.[26,27,29,30] When testing leg length inequality with an FLLR, Nadler states that "an improvement in leg length is not sufficient, and actual leg length balancing (evening) is necessary for confirmation. In other words, improvement in leg length is meaningless; only complete leg length evening is acceptable."[29] Prone leg length analysis has shown good inter-examiner reliability upon testing and is used to analyze the condition of the nervous system.[31-33] In addition to the FLLR, pressure testing was utilized. Skin contact is made with the distal tuft of a phalange to the spinal segment of interest. Each pressure test applies a specific vector and torque on the spinal segment of interest in order to determine the line of correction.[26] The pressure test is used to "temporarily reflex the dynamic dyskinesia or dysponesis of the subluxation's line of correction."[27] The adjustment is then administered with the Integrator.

Primary subluxations are grouped into eight non-linear testing priorities in order for efficient analysis and is reference in table 2.[26] Holder recognizes there are two categories of vertebral subluxation: spinal cord pressure and spinal cord tension.[26] As described in R.W. Stephenson's Chiropractic

Textbook, spinal cord tension is a major component of vertebral subluxation.[34] Holder argues that spinal cord tension is the most severe category of vertebral subluxation. Tensile forces on the spinal cord from the dura mater affect the neuroskeleton as a whole and therefore have the greatest insult on the system.[30] Areas of direct dural attachment to bone are the sphenoid, occiput located at the foramen magnum, C2, C5, S2, S3, S4, and coccyx.[26] Holder states that these areas of direct dural attachment are the most commonly subluxated.[26] The protocol states that there can only be one appropriate primary vertebral subluxation present at any given moment. In addition, there may be up to nine secondary or tertiary vertebral subluxations.[26] Adjusting one primary vertebral subluxation can therefore clear up to ten potential subluxations. Due to this principle, "one, two, but never more than three segments" are adjusted per visit and there are never more than three adjustments given in the same order per segment.[26]

The patient remained compliant with the recommended care plan and received regular reassessments. After two months of care, the patient reported much improvement in all of her complaints including irritable bowel syndrome. Her QVAS score reduced to 38% and her thermography scans demonstrated that she was responding to the adjustments. It was at this time that the patient reported she had just seen her medical doctor for a yearly physical and a follow up thyroid function test was performed. Her TSH and T4 levels were in the normal range and her T3 levels were found to be elevated. Her medical doctor stated that her "thyroid is over-replaced" and took her off Nature-Throid. Table 3 depicts the patient's thyroid function tests before and after chiropractic care. After eight months of care the patient's paraspinal thermography demonstrated moderate heat abnormality at T1 and a mild heat abnormality at T2 with the rest of the scan being clear. (See figure 2B).

Discussion

Chiropractic Literature

Hypothyroidism is a frequently encountered clinical condition and is historically medically treated. A thorough review of chiropractic literature regarding hypothyroidism revealed sparse results. These searches resulted in eight peer-reviewed articles and are discussed below. However, of the eight articles, only three discussed chiropractic management of hypothyroidism.

Bablis and Pollard have published two articles depicting two case series discussing the chiropractic management of hypothyroidism using Neuroemotional technique (NET). In 2004, Bablis and Pollard depicted two females diagnosed with hypothyroidism.[16] According to Bablis and Pollard, the normal range for free T4 is 10.0-21.0 pmol/L, normal range for free T3 is 2.2-5.3 pmol/L, and normal TSH is 0.30-4.00 mIU/L.[16] A 45-year-old Caucasian female reported low free T4 of 11.6 pmol/L, free T3 of 2.2 pmol/L, and low TSH level of 0.07 mIU/L shortly after starting chiropractic care. After six months of chiropractic care utilizing NET, the patient reported an increase in her free T4 level to 14.8 pmol/L and TSH level to 0.33 mIU/L.[16] The second case, a 30-year-old Caucasian female, started under chiropractic care for nervousness and depression. She was later diagnosed with primary hypothyroidism and demonstrated an elevated TSH level of 8.1 mU/L. The patient received one treatment using NET and a follow up thyroid function test, which showed a drastic decrease in TSH to 3.7 mU/L. Another follow up test was performed seven months later and showed the patient's TSH level lowered again to 3.0 mU/L.[16]

In 2009, Bablis and Pollard depicts different two females diagnosed with hypothyroidism.[35] A 41-year-old Caucasian female reported an elevated TSH level of 13.99mU/L and low free T4 levels of 8pmol/L and was diagnosed with primary hypothyroidism. The patient received chiropractic care utilizing NET for eight weeks. A follow up thyroid function test demonstrated a decrease in TSH to 5.81 mIU/L and an increase in free T4 to 12 pmol/L. With this improvement, the patient discontinued the use of her thyroid medication.[35]

The last case report from Bablis and Pollard depicts a 27-year-old Caucasian female who presented with a previously diagnosed hypothyroid condition. Her initial thyroid function test showed an elevated TSH level of 14.8 mIU/L which is consistent with hypothyroidism. After chiropractic care utilizing NET for two months, the patient received a follow up thyroid function test and it was determined her TSH level was 3.58 mIU/L and was in the normal range. A long term follow up with the patient reported discontinued use of thyroid medication and maintained improvement of thyroid function.[35]

In 2010, Brown, Graham, Bonello, and Pollard discussed a protocol for a pilot study to assess the chiropractic management of primary hypothyroidism utilizing NET.[36] They proposed a placebo-controlled, single blinded, randomized clinical pilot trial of 102 participants. These participants had to be over the age of 18, previously diagnosed with primary hypothyroidism, and on a stable dose of thyroid medication for six months prior to entering the trial. The treatment group would receive ten NET treatments over a six-week period. The placebo group would receive the NET protocol but would not receive the therapeutic components of the NET procedure. Both groups would be assessed at seven weeks and six months utilizing a depression questionnaire, thyroid function blood testing, SF-36 questionnaire, resting heart rate and temperature measurements.[36] In 2015, Brown, Graham, Bonello, and Pollard published the results of this trial.[37] Overall there were forty-four participants receiving NET and forty-six participants receiving the placebo. The conclusion of the study was found to show no clinical benefit in the management of primary hypothyroidism utilizing NET.[37]

Bak and Engelhardt discuss a case on improvement of hypothyroidism under chiropractic care.[38] The patient was medically diagnosed with Hashimoto's Thyroiditis after a thyroid function test indicated elevated TSH four years prior to seeking chiropractic care. After one month of chiropractic care utilizing Chiropractic Biophysics (CPB), the patient received a follow up thyroid function tests and it demonstrated her TSH level dropped into the hyperthyroid range. The patient was ordered by her medical doctor to decrease her thyroid medication as she demonstrated an improvement in thyroid function.[38]

Jacobs, Franks, and Gilman provided evidence on the use of

Applied Kinesiology (AK) in the diagnosis of hypothyroidism.[39] The article explained the mechanism behind AK testing and how it was used in conjunction with laboratory ratings to determine thyroid dysfunction. Jacobs, Franks, and Gilman clearly state that AK can be used as a screening protocol and not for the treatment or diagnosis of thyroid dysfunction.[39]

Echeveste reported a case on chiropractic care of a 9-year-old female diagnosed with Diabetes Mellitus Type 1 and hypothyroidism utilizing Diversified Technique.[40] Echeveste reported there was stabilization in the patient's glucose levels and there was an improvement in hypothyroidism symptomatology.[40] However, Echeveste failed to objectively report the overall thyroid function or if thyroid medication was discontinued.

Edwards and Alcantara performed a case study of a 28-year-old pregnant female who presented with migraine headaches, hypothyroidism and tachycardia.[41] The case focused visual field disturbances and chiropractic care during pregnancy. Edwards and Alcantara failed to discuss overall thyroid function of the patient and chiropractic management of hypothyroidism.[41]

Subluxation Model

Chiropractic literature describes multiple theories that demonstrate how nervous system dysfunction caused by malposition of vertebrae can affect somatic and visceral structures.[42] This phenomenon is termed *vertebral subluxation* and is the foundation of chiropractic theory and practice. In order to understand the mechanisms of this case, an examination of how vertebral subluxation affects proper visceral function, specifically the HPT axis will be discussed. According to the dysafferentation model of subluxation, proper input to the central nervous system (CNS) is needed proper nervous system function.[42] Kent states, "aberrant afferent input into the CNS may lead to dysponesis" resulting in inadequate efferent response to the body and consequently to visceral function.[42] The intervertebral motion segment is richly surrounded by nociceptors and mechanoreceptors. There is extensive nerve supply to the entire vertebral column. Specifically in the cervical spine, the sinuvertebral nerve supplies the intervertebral disc at the segment of entry and the segment above.[42,43] An article by McLain states, "the presence of mechanoreceptive and nociceptive nerve endings in cervical facet capsules proves that these tissues are monitored by the central nervous system and implies that neural input from the facets is important to proprioception and pain sensation in the cervical spine."[42,44] Therefore, biomechanical dysfunction such as hypomobility of the affected segments can alter mechanoreception and nociception. It is understood that decreased mechanoreception can cause an increase in nociception.[42,45] In addition, it is also proposed that altered mechanorecption elicits a physiological reflex activation which causes altered autonomic nervous system function and general peripheral nervous system dysfunction.[40,46,47]

Stress and increased nociception is shown to elicit a neuroendocrine response.[45-49] Bablis presents a proposed mechanism of how prolonged stress affects the neuroendocrine system including thyroid function.[35] Increased levels of epinephrine have been shown to elicit autonomic and neuroendocrine responses to stress. This includes the activation of the hypothalamic-pituitary-adrenal (HPA) axis.[35] The hypothalamus releases corticotrophin-releasing hormone (CRH), a neurotransmitter found in stress response.[50] CRH then stimulates the release of adrenocorticotropin (ACTH) from the pituitary and in turn, ACTH stimulates the release of cortisol from the adrenal cortex.[50] Through Bablis' research, ACTH, cortisol, and epinephrine are common indicators of prolonged stress.[35] An article by O'Connor, O'Halloran, and Shanahan states that prolonged stress causes a decrease in TRH release from the hypothalamus resulting in a decrease anterior pituitary release of TSH, and therefore inhibition the release of T3 and T4 from the thyroid gland.[51] Bablis also noted that inhibition of the HPT axis during prolonged stress can be mediated by the chronic release of CRH, somatostatin, and cytokines.[35] In conjunction of a chronic C1 subluxation and prolonged stress exhibited by the patient, it is proposed that autonomic nervous system dysfunction triggered dysfunction in the neuroendocrine system. The chiropractic adjustment is understood to restore proper motion and reestablish the normal balance between nociception and mechanoreception.[40,42,45] In addition, a cervical adjustment elicits a parasympathetic response from the autonomic nervous system and "in turn helps rectify HPA axis dysfunction" and thereby HPT axis dysfunction.[52]

Limitations

The limitations of this study include the outside effects, mainly the biomedical intervention, on the reduction of symptomatology. It is difficult to differentiate which treatment, chiropractic care or medical intervention, was responsible for improving the function of the pituitary and thyroid gland. Another area of limitation was the medical diagnosis of secondary hypothyroidism. The cause of pituitary dysfunction was not explored. It was stated only by the evaluation of the patient's thyroid function tests that she had secondary hypothyroidism.

Conclusion

Chiropractic is an art, science, and philosophy that is created on the vitalistic premise that the human body is a self-healing and self-regulating organism and observes that the cause of disease is from inside the body.[53] D.D. Palmer, the discoverer of chiropractic stated that living organisms are born with an innate intelligence that runs through the nervous system and allows the body to heal itself when given the right conditions. Interference with the nervous system would affect one's ability to heal and adapt to the environment, resulting in disease. This interference is termed "vertebral subluxation".[42,53,54]

This case presented a 34-year-old female diagnosed with secondary hypothyroidism, irritable bowel syndrome and neuromusculoskeletal complaints. Upon examination, the patient showed signs of nervous system dysfunction and vertebral subluxations. After two months of TRT care, the patient reported improvement in thyroid function determined by a thyroid function test and improvements in IBS and her back pain. This improvement caused her medical doctor to take her off of prescription thyroid medication. The evidence gathered from this case study suggests that chiropractic care may contribute to improvement in overall thyroid and gastrointestinal function through improvement in dysautonomia.

Additional research is needed to explore the benefits of chiropractic management in these types of cases. This is the first study to use TRT in the management of a patient with hypothyroidism and IBS. Future research should implement additional objective data in order to investigate the effects of chiropractic care on nervous system function. At a minimum, each study should provide specific, objective, and measurable data about the patient's nervous system and subluxations. Examples of this would include, but not limited to, rolling thermography, sEMG, and x-ray analysis. It is also crucial that plasma analysis of TSH and thyroid hormones is used to demonstrate thyroid function before and after chiropractic management.

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Tables

Indicator	Explanation
Palpation	Performed by hand using scanning palpation, tissue palpation, inter-segmental palpation, and motion palpation
Functional Leg Length Inequality	An Achilles deep tendon reflex is performed in order to view functional leg length and determine a differential diagnosis for the primary subluxation.
Abduction tendency / adductor resistance	If abductor tendency or adductor resistance is noted in the patient's legs while lying prone on the table this indicates a C2 subluxation on the side of resistance.
Foot flare	If inversion or eversion of the feet is noted while the patient lies prone on the table, this indicates a subluxation at a level of direct dural attachment.
Foot supination/pronation	If pronation or supination of the feet is noted while the patient lies prone on the table this indicates a subluxation at a level of direct dural attachment.
Heel tension	If unequal tension of the Achilles tendon is noted, this indicates a primary subluxation at a level of direct dural attachment.
Abnormal breathing patterns	This indicates compartmentalized breathing.
Inappropriate sustained patterns of paraspinal contractions	Performed by scanning by hand or an EMG and indicates the cause of the primary subluxation is likely mental or emotional.
Congestive tissue tone	Performed by tissue palpation of the anterior neck muscles and posterior kidney areas. This indicates the likely cause of the subluxation is chemical.
Postural faults	Abnormal postural positions are noted while standing, sitting, and laying prone.
Cervical Syndrome Test	The patient turns his/her head to the long leg side. If the legs balance, this indicates a posterior rotation of C1 or C5 with or without laterality subluxation.
Wrong-Un Test	The patient turns his/her head to the short leg side. If the legs balance, this indicates a lateral C1 subluxation.
Bilateral Cervical Syndrome Test	The patient turns his/her head to both sides and the leg length switches back and forth. This indicates a possible PI listing of the one following segments: Occiput, coccyx, C5, C1, and T6, or a lateral C1.
Derifield Test	A short leg in the prone extended position becomes the long leg in the 90 degree prone flexed position. This indicates one of following possible subluxations: AI sacral base, AS trochanter, PI ilium, or superior pubic rami.
Abnormal heat/energy radiation from the body	Performed by rolling thermography.

Table 1. 15 Indicators of Dis-ease and Subluxation Utilized in Torque Release Technique[26]

Priority	Possible Subluxation
1	Lateral occiput or lateral sacrum +/- *torque* Coccyx with sphenoid (compass vectors) *no torque*
2	**Cervical Syndrome Test:** C1 posterior rotated +/- *torque* **Wrong-Un Test:** C1 lateral +/- *torque* **Bilateral Cervical Syndrome Test:** Occiput posterior inferior +/- *torque* Coccyx posterior inferior with sphenoid *no torque* C5 posterior inferior +/- *torque* C1 posterior inferior +/- *torque* T6 posterior inferior +/- *torque* C1 lateral +/- *torque*
3	**Derifield Test:** AI sacral base *no torque* AS trochanter *no torque* PI ilium *no torque* Superior pubic rami *no torque*
4	C2 (C3) +/- *torque*
5	C7 +/- *torque*
6	L3 +/- *torque*
7	L5 +/- *torque*
8	Any other segment +/- *torque*

Table 2. Non-Linear Testing Priorities Utilized in Torque Release Technique[26]

	TSH	Total T4	Total T3
Normal Range	0.4-4.2 mIU/L	4.5-12.0 ug/dL	71-180 ng/dL
Pre-Chiropractic	2.30 mIU/L	6.2 mcg/dL	107 ng/dL
Post-Chiropractic	1.86 mIU/L	6.1 ug/dL	197 ng/dL

Table 3. Pre and Post Chiropractic Adjustment Thyroid Test Results

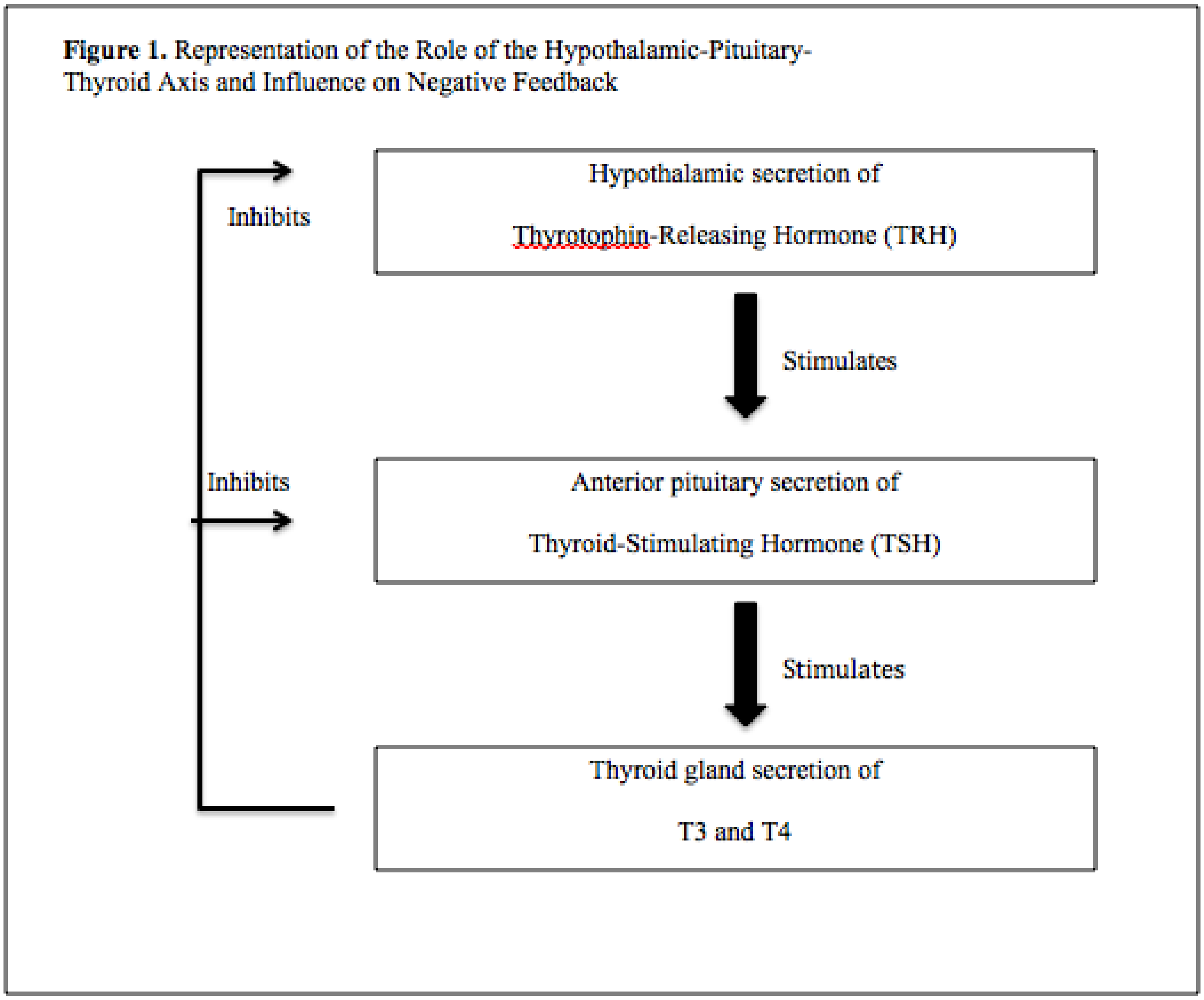

Figure 1. Representation of the Role of the Hypothalamic-Pituitary-Thyroid Axis and Influence on Negative Feedback Diagram courtesy of Kathleen Delander 2016. Used with permission

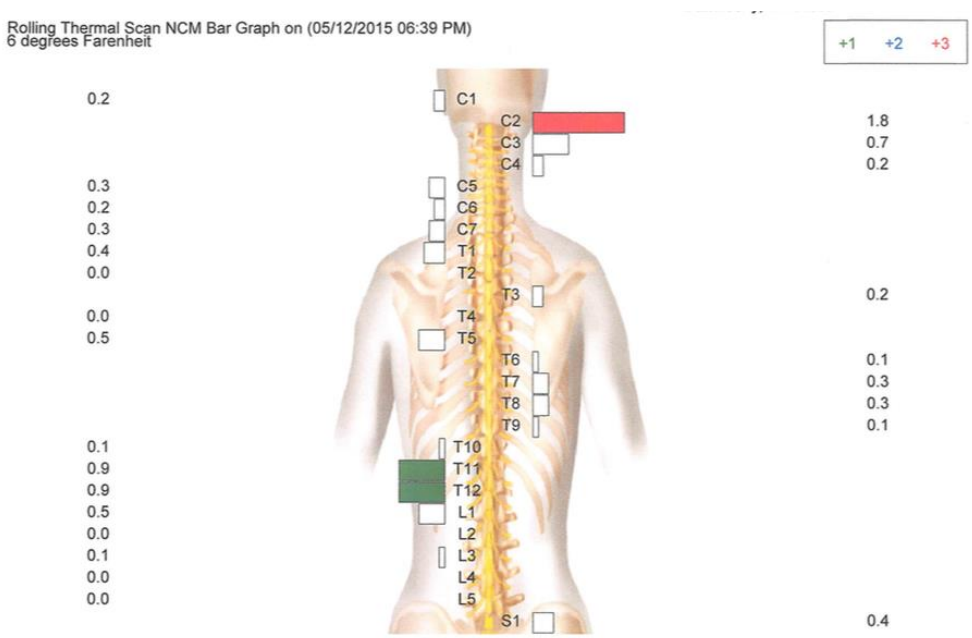

Figure 2A. Rolling Paraspinal Thermography Scan: Initial Visit

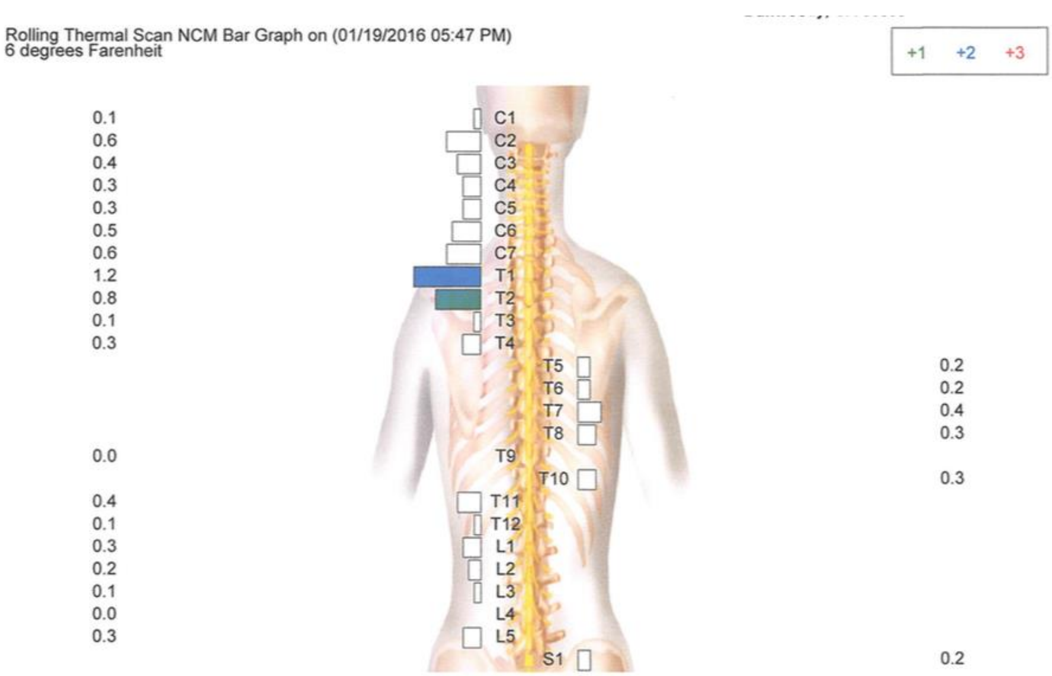

Figure 2B. Rolling Paraspinal Thermography Scan: Eight-Month Re-evaluation