

Hypothyroidism: A New Model for Conservative Management in Two Cases

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ABSTRACT: Objective: To review the function, anatomy, physiology, development, hormone synthesis and dysfunction of the thyroid gland. Treatment options are discussed, and 2 case studies of a mind-body therapy (Neuro-Emotional Technique—NET) successfully managing hypothyroid dysfunction are presented. Data Sources: MEDLINE search using key words: thyroid, synthesis, development, anatomy, physiology, hyperthyroidism and hypothyroidism. Data Selection: Eighty-five papers fit the key words and were selected based on relevance to the topic. Papers were selected that contained relevant information on normal and abnormal thyroid function and its management. Data Extraction: Selected papers had to contain information that directly related to the diagnosis, anatomy, physiology and management of hypothyroid conditions. Papers were also selected that described a possible neurophysiological mechanism for the observed treatment effects. Data Synthesis: Objective measures of a new mind-body approach to hypothyroid dysfunction are presented, and its relevance to the biopsychosocial model is discussed. This new treatment is compared to the existing biomedical approaches to treatment. Conclusions: Thyroid dysfunction has been effectively treated with medicine for many years. This paper presents a new therapy that produced objective pre-post changes to hypothyroid dysfunction in 2 cases. This therapy may have potential in future circumstances, with further research recommended to confirm its reliability/validity.

INDEX TERMS: (MeSH): CHIROPRACTIC; PSYCHOLOGY; THYROID GLAND. (Other): BIOPSYCHOSOCIAL MODEL; TREATMENT.

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INTRODUCTION

The thyroid gland acts to regulate many aspects of human function, either directly or indirectly, primarily through its effects on metabolic activity of body tissue. In addition, the thyroid affects function of the heart, the gut and the musculoskeletal system. Due to these widespread effects, hyperthyroidism causes changes in many body systems (generally to increase function), while hypothyroidism results in equally widespread but opposite changes (*i.e.* generally to decrease function). Thyroid disorders are mostly diagnosed between the ages of 30 and 60 years of age, and women are 4 times more likely to be affected than men.

Hypothyroidism

Hypothyroidism results from a deficiency of circulating free thyroid hormones in the body. Causes are multiple and include chronic lymphocytic thyroiditis, iodine deficiency, prolonged treatment with lithium, pituitary insufficiency and metastasising tumours. It may also be the result of ¹⁴²I radioiodine therapy or surgery for hyperthyroidism, where too much thyroid tissue has been destroyed.¹⁻³

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Hypothyroid symptoms affect the whole body. Physically patients may experience lethargy, slowed mental functioning and poor short-term memory.¹⁻⁵ A hoarse voice, due to increased infiltrates in the larynx, is often apparent.¹⁻³ In the cardiovascular system, there is a decrease in contractility and heart rate, with resultant decreased cardiac output, which has implications for fatigue-based complaints, a secondary cause of lack of exercise.^{1-3,5,6} Constipation, anorexia and weight gain are also features of hypothyroidism.¹⁻⁵ Females can suffer from heavy and irregular menses, infertility and loss of libido, while males may suffer from impotence.¹⁻⁴

Hypothyroid patients often suffer from back pain, with the muscles demonstrating sluggish contraction rates and appearing oedematous. Non-pitting oedema of the hands and feet with poor wound-healing is also a feature.¹⁻⁶ The discovery of coarse, broken, yellowing skin, facial puffiness and thick, brittle skin on examination may also alert the practitioner to hypothyroidism.¹⁻⁶ A decrease in body temperature and intolerance to cold are distinctive clinical features of a patient with hypothyroidism.^{1,3-5}

Hyperthyroidism

The review of symptoms of hyperthyroidism is beyond the scope of this research. For further discussion, the reader is directed to Lazarus⁷ and Bailes.⁸

THE THYROID GLAND

Anatomy

The thyroid gland is situated directly below the larynx and anterior to the trachea in the neck.^{1,9} The gland is a bilobulated structure connected centrally by an isthmus,¹⁰ with its shape

resembling a butterfly. Many in the population—50%-75%—have an additional lobe, called the pyramidal lobe, which attaches to the superior aspect of the isthmus. The isthmus usually sits over the 2nd to 4th tracheal cartilage,⁹ which corresponds to the level of the 5th, 6th and 7th cervical vertebrae.¹¹ The gland moves on swallowing due to its attachments by fascia to the trachea and larynx. (This fact is used to determine a thyroglossal cyst).¹¹ In an adult, the normal thyroid is approximately 2-2.5 cm wide and 3-5 cm in length.¹⁰

Because of its high vascularity due to an arteriole plexus supplied by the superior and inferior thyroid arteries, the thyroid gland is dark red to brownish in colour.^{10,11} Embedded in the posterior surface of the thyroid are the parathyroid glands.¹⁰ There are usually 4 of these: one pair superior and one pair inferior. These glands secrete parathyroid hormone, the function of which is to maintain serum calcium levels. It achieves this by promoting intestinal absorption and renal tubular reabsorption, and the release of calcium from bone.¹²

The thyroid gland is composed of many spherical structures, called follicles or acini.¹¹ These follicles are hollow and contain a viscous substance called colloid.¹⁰ The cells of the follicle produce this colloid, which is largely composed of thyroglobulin. Between the follicles are the parafollicular or C (clear) cells.¹⁰

Thyroglobulin is involved in all processes of production of thyroid hormone—from its synthesis to its storage and to its active state. The thyroid is the only endocrine gland that is able to store its hormone; other glands secrete the hormone as soon as it is produced.^{9,10} The thyroid follicles and its colloid are the housing sites for the stored hormone. Three hormones are produced by the thyroid gland: thyroxine (T₄), triiodothyronine (T₃), and calcitonin. Calcitonin is produced by the C cells and plays a role in regulating plasma calcium levels by acting to lower the calcium levels in plasma.^{9,11}

T₄ is the main hormone produced by the thyroid gland, although some T₃ is produced,¹⁹ with 70-80% of the T₃ derived from conversion of T₄ to T₃.¹³⁻¹⁵ This conversion takes place in various parts of the body, including the liver, kidneys, pituitary gland, brain and brown fat, as well as the thyroid gland itself.¹⁶ The conversion uses the enzyme thyroxine 5'-deiodinase types I and II, which causes denaturation of T₄.¹⁶ The normal ranges of the thyroid hormones are 60-180 nmol/l for T₄ and 0.8-2.7 nmol/l for T₃.¹⁷ It is, however, T₃ that produces the main effects, being 4 times more potent than T₄.¹

Development

The thyroid develops from two embryological structures: the thyroid diverticulum and the ultimobranchial bodies.¹⁸ The diverticulum produces the thyroid gland, while the ultimobranchial bodies produce the parafollicular C cells.¹⁸ The thyroid develops from the tip of the foramen caecum.^{9,11} A pit is formed, and the diverticulum elongates to produce the thyroglossal duct.¹⁸ Cells at the distal end of the duct undergo rapid proliferation and expansion to produce the bilobulated gland by 31 days gestation.¹⁸ By 41 days, the thyroglossal duct fragments and atrophies.^{9,18} Around this stage, the diverticulum and ultimobranchial bodies fuse with each other, and the gland has descended into its final position in

the neck.¹⁸ Also around this time, there is differentiation of the follicle cells of the thyroid and the expression of genes that are used in the production of thyroid hormone.¹⁸ By 12-13 weeks, thyroid hormone can be detected in the foetus,^{15,18} due to the thyroid's ability to synthesise T₄ and the pituitary to secrete thyroid-stimulating hormone (TSH/Thyrotropin).¹⁵

The hypothalamo-pituitary thyroid (HPT) axis starts maturing during the second trimester, and by the second half of gestation becomes functional.⁹ T₄ levels reach adult concentrations at around 37 weeks, while at 41 weeks, T₃ levels are lower than in adults, with TSH being slightly higher.^{9,15} Two possible explanations include:

- Stimulation of T₃ is subthreshold.
- Foetal processes that convert T₄ to T₃ are not yet mature.

This seems plausible, as there is a significant increase in T₃ levels after birth due to the liver metabolising T₄ to T₃.

THYROID FUNCTION

Synthesis and Secretion

The colloid of the follicular cells is the site of hormone production within the thyroid.⁹ The process requires both iodine and tyrosine, which are both obtained through diet; tyrosine is also produced by the body.⁹ Iodine, however, is the rate-limiting agent in the process.¹⁹ Iodide is absorbed and concentrated in the follicle cells, having been actively pumped against a large concentration gradient from the extracellular fluid.^{19,20} The iodide is oxidised by thyroperoxidase (TPO) and hydrogen peroxide to produce iodine. As a result of chemical iodination, the iodine attaches to tyrosine to produce monoiodotyrosine and diiodotyrosine (DIT) and form part of the thyroglobulin colloid. Approximately 20% of these then combine to form either T₃ (*via* one MIT and one DIT joining) or T₄ (where two DIT join).^{9,20} This process involves both hydrogen peroxide and TPO.²⁰ The hormone is then stored within the colloid of the follicles until required by the body.⁹

When the body requires the thyroid hormone, it needs to be processed from its stored state to its active form. Since the thyroglobulin is not released into the extracellular fluid, the hormone needs to be cleaved off the MIT/DIT molecule. This process is started when the follicular cell ingests a piece of colloid to form a colloid droplet.⁹ Following ingestion by lysosomes, the thyroglobulin molecule is broken down, freeing the T₃ and T₄ (plus some unbound MIT and DIT).^{9,19} The hormone then passes through the outer membrane of the follicle cell and is released into the bloodstream.^{9,19} The MIT and DIT are recycled after they have been deiodinated by a dehalogenase.¹⁹ Once the hormone is in the bloodstream, only 1% is free and biologically active.⁹ The other 99% are bound to plasma proteins, as are thyroxine-binding globulin (TBG)(70-80%),^{14,20,21} thyroxin-binding albumin (TBA) and thyroxin-binding prealbumin (TBPA).^{14,20,21}

Regulation

The HPT axis regulates the growth and function of the thyroid gland, as seen in Figure 1.^{14,20} This axis deals with the interaction of each of the 3 structures to regulate the amount of thyroid hormone circulating in the body. Thyrotrophin-releasing hormone (TRH) is released by the hypothalamus,

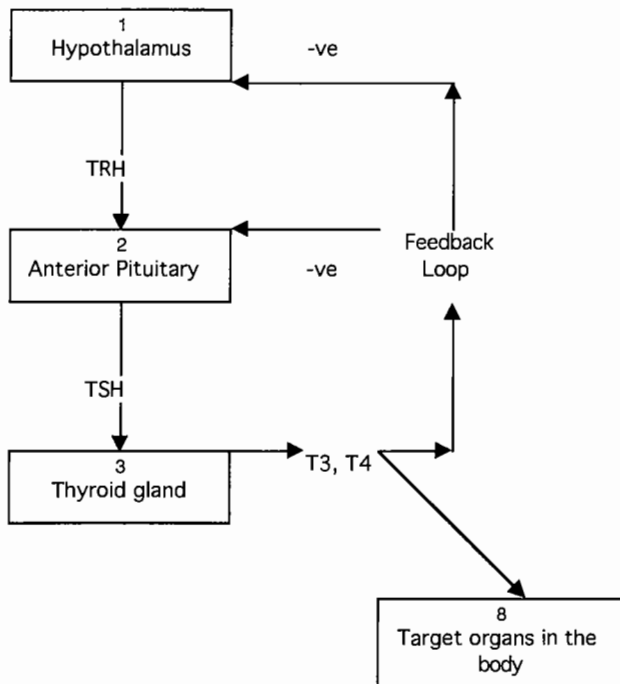


Fig. 1. The HPT axis negative feedback loop. TRH is released by the hypothalamus, which stimulates the anterior pituitary. This releases TSH, which activates the thyroid to produce T_3 , T_4 . When T_3 , T_4 is released into the circulation, it causes inhibition of TSH and TRH production by the anterior pituitary and hypothalamus respectively.

which acts on the anterior pituitary gland.^{11,13} In times of stress, more TRH is produced.¹¹ When “stimulated,” the pituitary produces TSH.^{11,13} As a result of stimulation by TSH, the thyroid increases iodine uptake and increases thyroid hormone production and secretion.^{9,11,13} TSH also causes cell hypertrophy and cell hyperplasia.^{9,11,13}

Regulation of the amount of thyroid hormone in the system is accomplished by a negative feedback system of this HPT axis. Hormone levels are monitored by the pituitary gland, and these levels regulate the amount of TSH produced.⁹ The HPT axis has an inverse log-linear relationship between TSH levels and free T_4 . Small changes for free T_4 levels result in exponential changes in TSH levels in the opposite direction.^{9,20} As a result, TSH levels are the primary diagnostic test used when assessing thyroid function.

Thyroid Function Tests

As mentioned above, when there is a small decrease in free T_4 levels, there is a large increase in TSH levels, an attempt by the body to produce more hormone to address the shortage, although this can take up to 4-6 weeks.²² Conversely, when there is a small increase in free T_4 , there is a large drop in TSH levels to try to reduce the amount of hormone the body is producing.²²

In most cases of abnormal thyroid hormone level, the primary cause is due to thyroid dysfunction, and the TSH level is a reaction to this change.²³ On rare occasions, the level of thyroid hormone is a reactive response to changes in TSH levels, which can occur with pituitary or hypothalamus dysfunction.²³

Due to TSH levels being largely sensitive to circulating hormone levels, the first thyroid function test commonly performed is an assay of TSH levels.²²⁻²⁴ If there is an abnormal TSH level, further tests are then conducted. Free T_4 tests are performed, and then if indicated, free T_3 and microsomal antibody tests are conducted. Microsomal antibody tests identify autoimmune thyroiditis as the underlying cause of the thyroid hormone dysfunction (Hashimoto’s disease).²³

Figure 2 shows the general path of tests used to identify most thyroid hormone dysfunction. TSH levels are first taken. If these are within normal limits there is no dysfunction. If levels are elevated, a decrease in hormone level is suspected, and only free or total T_4 levels are required. If levels of TSH are subnormal, total or free T_4 and T_3 levels are taken, followed by TRH tests to see if the change is due to pituitary or hypothalamus dysfunction. If there are undetectable TSH levels, hyperthyroidism is suspected, and total or free T_4 and T_3 tests are performed. Due to different laboratories having different methods of measuring these levels, they may reference different levels of normal,²³ therefore normal levels for the individual laboratory are always posted with the result of the test. Thus, results have an intra-lab reliability, but not an inter-lab reliability.

For further discussion on the significance of different levels of each test, the reader is directed to Dayan.²⁴

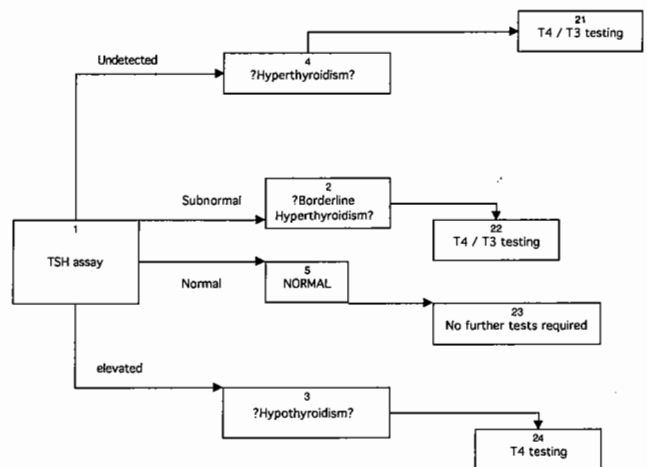


Fig. 2. Typical thyroid function testing cascade (adapted from Ref. 23).

TREATMENT OPTIONS

Hypothyroidism

Administration of thyroid replacement drugs is the standard treatment for hypothyroidism. Thyroid hormone is now synthesised; older preparations were derived from ground cow thyroid glands.²⁵ Treatment aims to achieve a stable serum concentration of T_3 between doses of the drug. Because of its long half-life and conversion to T_3 by the body, Levothyroxine sodium has become the preferred drug. Initially, the patient is prescribed 50-75 μg levothyroxine per day.²⁵ The dosage is slowly increased until the desired levels of hormone are achieved. Most patients with uncomplicated

Table 1

PRE- AND POST-TREATMENT THYROID TEST RESULTS			
	TSH	T ₃	T ₄
Pre-treatment	0.07	2.2	11.6
Post-treatment 1	0.09	4.1	14.2
Post-treatment 2	0.33	—	14.8

primary hypothyroidism require an average dose of about 1.6 µg/kg ideal body weight per day.^{26,27} This typically equates to 120-160 µg/day in males and up to 75-120 µg/day in females.²⁷

The elderly require less levothyroxine and so need to be started at a lower dose, with smaller incremental steps. Rapid increases in heart rate and blood pressure can complicate levothyroxine use and may place patients with cardiovascular problems at risk, therefore a reduced initial dose is given. Some drugs, such as anticonvulsants, phenytoin, carbamazepine, and the antituberculous agent, rifampin, can increase metabolism of levothyroxine, so a higher dose is prescribed for patients using these drugs. Other drugs may interfere with absorption of levothyroxine from the gut, so administration of levothyroxine is spaced at least 4 hours apart from these medications. These drugs include cholestyramine, ferrous sulphate, sucralfate, and aluminium hydroxide antacids.^{1,27}

Monitoring of the thyroid levels takes place every 6-8 weeks until levels normalise, and once this occurs, levels should be evaluated on a yearly basis.⁷ If secondary hypothyroidism is suspected, there needs to be an evaluation of adrenal function prior to initial administration of levothyroxine, as adrenal crisis can be triggered by thyroid hormone in untreated adrenal insufficiency.¹³

Hyperthyroidism

Discussion of treatment options for hyperthyroidism is beyond the scope of this paper. For a review of these options, the reader is directed to Klein²⁸ and Younes.²⁹

CASE STUDIES

Case 1

A 45-year-old Caucasian female initially presented in June 2002 with cervical pain with associated neck and shoulder stiffness. She received manual therapy (chiropractic) for the neck pain and stiffness, which resolved her symptoms.

In June 2002, the patient complained of tiredness and had put on weight (from 61 kg to 67 kg) in the past 4-6 weeks. A blood test for thyroid function was sought. The first test, conducted on 5 August 2002 showed free thyroxine to be 11.6 pmol/l (range 10.0-21.0 pmol/l), free T₃ 2.2 pmol/l (range 2.2-5.3 pmol) and TSH 0.07 mIU/l (range 0.30-4.00 mIU/l) (see Table 1).

Once diagnosed, a mind-body treatment called Neuro Emotional Technique (NET) was commenced to attempt to

Table 2

PRE- & POST-TREATMENT THYROID TEST RESULTS (CASE 2)	
	TSH
Pre-treatment	8.1
Post-treatment 1	3.7
Post-treatment 2	3.0

affect thyroid function. On 23 September 2002, a follow-up thyroid function test was performed that showed improvement in levels. Free thyroxine was 14.2 pmol/l, free T₃ 4.1 pmol/l, and TSH 0.09 mIU/l (this was still low).

NET treatment continued. A third thyroid function test was performed on 4 December 2002 after the patient had reported her energy levels were back to normal and her weight had dropped from 67 kg back to 62 kg. This test showed free thyroxine to be 14.8 pmol/l and TSH 0.33 mIU/l.

Case 2

A 30-year-old female Caucasian computer consultant initially presented on 25 March 1998 with nervousness and depression. A broken left arm in 1997 was the only remarkable medical history. Chiropractic manual therapy regularly helped this complaint.

On 12 October 2001, the patient presented with extreme tiredness and lethargy, stating that she had a thyroid problem, an elevated TSH level that was collected 3 days earlier on 9 October. That test showed TSH levels at 8.1 mU/l (range 0.5-4.15 mU/l).

A treatment using NET produced positive results in the patient: she subjectively reported an immediate improvement in tiredness. The patient was sent for a follow-up thyroid function test the same day (12 October 2001). This test showed TSH levels to be 3.7 mU/l. Another follow-up test performed on 8 May 2002 showed the TSH levels to be 3.0 mU/l (see Table 2).

NEURO EMOTIONAL TECHNIQUE (NET)

Treatment Procedure³⁰

The treatment therapy NET is versatile in its scope. There are 2 usual applications that NET can utilise: the body entry, which is used primarily by manual therapists, and the mind entry, which tends to be used by psychologists and other practitioners. The mind entry is more psychological in nature and is *via* a feeling, a thought or cognition. The body entry is more mechanical in nature, with entry *via* trigger point, muscle strength test or lesions that are commonly referred to by chiropractors as subluxations. A combination of these two methods may also be used. The body entry was used in the two cases described and will be the focus of this description. Another paper by the authors, now in press, discusses the mind entry.

The patient is initially evaluated for an appropriate, intact, healthy muscle that could resist the examiner's mild testing procedure. For the body entry in the cases described, the external rotators of the right upper extremity (teres minor) were chosen. This is the muscle associated with thyroid function (*via* Applied Kinesiology organ/muscle relationships). This muscle group was tested with the patient in the sitting position. The patient was instructed to relax the arm by the side and keep the elbow locked in the side, flexed and held at 90°, and to resist the examiner's pressure on the dorsum of the wrist, directed in an arc toward the midline. The patient was asked to resist by externally rotating the humerus. The author (PB) served as the examining and treating practitioner. The patient practised resisting the examiner's pressure several times until both examiner and patient were comfortable with the procedure.

Using the body method, the patient was asked to concurrently contemplate herself relaxing and to focus on the presenting symptoms while resisting the examiner's pressure. A weakening of the test muscle was observed at this time.

The patient was instructed to maintain contraction of the teres minor muscle, which produced ongoing weakness, while the examiner attempted to counteract the weakness by contacting specific points on the body (meridian access points). Using a traditional acupuncture model, the triple heater meridian would usually be relevant.³⁰ The practitioner performs a procedure known as therapy localisation to identify the relevant meridian to test and treat.³⁰ In this procedure, the practitioner places a hand directly over the meridian access points until the muscle (in this case, the teres minor) tests strong or changes. A positive finding occurs when a muscle that tested weak before stimulation of the access point becomes strong on muscle testing after stimulation of the access point. Such was the case with both patients described in this paper.

Walker³⁰ states that once the meridian has been identified, the practitioner should investigate the psychosomatic (mind-body) connection. In this procedure, the treating practitioner attempts to derive an overview of the mind-body state by attempting to reproduce conditions of psychosomatic responses possibly associated with significant previous life experience. This is achieved *via* a sequential flow of questions asked by the practitioners and the responses observed in the muscle tests performed after answers.

According to Applied Kinesiology theory,³¹ all such meridians are associated with psychological and somatic states. In the case of thyroid dysfunction, the psychological states include muddled thinking, instability, paranoia, feelings of loss and vulnerability, anxiety and emotional instability. One somatic change is said to result in a weakness of an external rotator muscle of the shoulder (teres minor). In a protocol outlined by Walker, the treating practitioner first attempts to localise the dysfunction *via* muscle testing. The psychosomatic dysfunction is then identified by reviewing the patient with a series of questions aimed at identifying the mind state of the patient relevant to specific feelings said to be associated with the dysfunctional meridian.³¹

This process requires the practitioner to explore the original event that evoked the conditioned response to determine why the response is present. The practitioner asks the patient to recall the event when the feeling or situation first occurred. After determining the memory that was associated with a painful or otherwise significant event (*e.g.* an event at age 27 that caused a state of fatigue and stress similar to the present complaint), the practitioner then attempts to relate the event that first occurred to the present by exploring why there is a retained psychosomatic response that is operating as almost a post-traumatic event. Thus, the practitioner attempts to determine which and why recent events have exacerbated a conditioned response that has been created by the first "trauma." The phenomenon of "extinction" to this specific stimulus has not occurred. As a result, an ongoing "associated conditioned response" to this stimulus will be observed until extinction of the stimulus is achieved. According to Walker, such a stimulus is inappropriate and stressful to the patient, and may be associated with painful somatic dysfunction.³⁰

Once the original event is identified, the patient is muscle tested before and after she ponders the mind state associated with the psychosomatic changes, and is then screened about whether the psychosomatic effects result from a loved one, someone that loved or was loved by the patient, or by something relevant to money, finance, job and career. Finally, the patient is investigated from the point of view of any significant specific role, such as parent, sibling, breadwinner, patient, sports person, worker, *etc.* With all questions she is asked if she could recall the feelings and to ponder them. The patient is then asked to perform a muscle test with the thoughts uppermost in her mind, and a weak muscle test is said by Walker to be indicative of a "problem."

Once a scenario is determined, the practitioner attempts to tie together the findings into a summary, which should result in a sentence that describes the salient aspects of the story relevant to the patient. With this story uppermost in her mind, the patient again undergoes a muscle test. It would be expected that the previously strong muscle (in these cases, the teres minor), would become weak.

In addition to the weakening muscle, positive feedback can be obtained by observing the patient for subtle sympathetic nervous system changes that often accompany weakness on muscle testing. Questioning by the practitioner will attempt to localise the time line of the first occurrence of these feelings. When asking the patient to remember the response associated with the first incidence, the practitioner should note other responses, such as hot flushes, sweating palms, slouched posture, holding back of tears, dilated pupils, withdrawal, *etc.* All of the preceding signs are said by Walker to be associated with identification of the original process that set up the psychosomatic conditioned response.

Treatment involves the patient holding the meridian access point (in this case, by touching the point over the thyroid), and holding the cranium (over the frontal lobe), with the practitioner simultaneously providing a somatic stimulus to the spinal segment said to be associated with the dysfunction meridian or organ whilst contemplating.^{31,32}

Walker associates the triple heater meridian with anxiety, paranoia, muddled thinking, emotional instability and the C1, C4 and C7 spinal segments. A positive response in this region indicates that the emotions may be associated with the concept being pondered. While the patient ponders the summary statement, a manually-assisted, short-lever adjustment instrument was used to provide a minor mechanical (somatic) force in the posterior to anterior direction applied along the facet plane to both transverse processes of C1, C4 and C7, segments associated with the triple heater meridian, during inhalation, breath-holding and exhalation.³⁰

The practitioner then confirms the procedure by retesting the meridian entry point to show that the muscle (in these cases teres minor) tests strong after challenge. The reader will note that this process results in a direct feedback as to the “success” of the procedure—a feedback that is immediately apparent to the patient, therefore resulting in conscious feedback of the psychosomatic connection. The treatments are often followed up with additional nutritional or homeopathic support using a similar testing procedure.

DISCUSSION

Walker founded the treatment therapy Neuro Emotional Technique (NET) after becoming frustrated that some patients achieved good results with traditional chiropractic methods, whilst others with similar complaints achieved little or no result.³⁰ He developed the technique after noting that emotions appeared to be a large component in such recalcitrant conditions. He drew upon the meridian system of Eastern philosophy, as well as Applied Kinesiology principles developed by Goodheart³¹ to develop a technique to address the emotional component of physical conditions.³⁰

The orthodox concept of emotions has been that they cause no neurophysiological change. The biopsychosocial model by Engel³³ challenged this concept. The model proposes that not only are physical factors able to cause psychological dysfunction, but that psychosocial factors can also cause physical dysfunction. The biopsychosocial model is accepted by the medical profession in cases of functional bowel,^{34,35} cardiovascular³⁶ and chronic pain syndromes,³⁷ as well as other chronic illnesses.³⁸⁻⁴² Thus, there is a framework in place that, as proposed by Walker, emotional experiences in the presence of physical and neurological trauma can cause a meridian imbalance that presents as a physical condition that does not resolve.³⁰ NET uses the concept that not only can psychosocial issues cause physical dysfunction, but that through treatment they can also serve as a source of relief to physical conditions.

This possible link between the effects of psychosocial factors on physical conditions can be explained by research into memory and conditioned responses.⁴³⁻⁴⁶ Conditioned responses are “learned” responses that occur with a stimulus that does not normally provoke such a response.

Pavlov first demonstrated the classical conditioned response.⁴³ He presented a dog with food (primary stimulus), which caused the dog to salivate (unconditioned response, *i.e.* the response that usually occurs with that stimulus). At the same time, a bell was rung (secondary stimulus). Over consecutive trials, the amount of food was reduced until no food was present when the bell was rung. The dog still

salivated (conditioned response; *i.e.* a response that had been “learned” for that stimulus), as it associated the bell with food.

Pavlov’s experiment demonstrated how a secondary stimulus could reproduce a primary unconditioned response when the secondary stimulus is initially coupled with the primary stimulus. In the same way, an emotion (secondary stimulus) can be associated with the unconditioned response of a primary stimulus. Rolls⁴⁴ conducted research on the role of a rewards/punishment system in the brain memory systems, which produced results similar to the conditioned response described above. He showed that when associated with a primary reinforcer, a response could be associated with a stimulus.

The formation of memory is a complex issue that is not fully understood, however we know that the hippocampus and amygdala play a large role in its formation.^{45,46} Research has shown that damage to the hippocampus causes amnesia, both retrograde and anterograde.^{47,48} The amygdala, along with the adrenergic system, is involved in the consolidation of memory and involved in the experiencing of emotion, particularly anxiety.^{45,46} Thus emotions can play a role in memory formation. Experiences with strong emotional content are more strongly remembered.^{46,49} In many cases, it is not the specific experience, but the emotions felt in association with that experience, that are vividly recalled.⁴⁴

The premise of NET is that it identifies these psychosocial factors that cause dysfunction *via* neurological/meridian imbalances and attempts to “correct” them. To address these imbalances, NET uses several methods. These include informing patients of their condition and how psychosocial factors can induce it, and educating patients on the mind-body connection and how this interaction can be not only the “cause” of the condition, but also a therapy.

A further component of NET therapy is the use of cognitive restructuring. This involves aiding the patient to identify the causative issue, confronting the associated issues surrounding it, and finally developing appropriate strategies to respond to these issues. This form of therapy has been used with success in many conditions, including post-traumatic stress disorder⁵⁰ and chronic fatigue syndrome,⁵¹ and as a coping tool in cancer therapy⁵² and rheumatoid arthritis.⁵³ A crucial aspect paramount to the success of the therapy lies in the identification of the initial causative issue, as this is the original source of the meridian/psychosocial imbalance. If this is not identified and addressed, the condition will probably return. Sometimes, to help the patient focus on this recall, deep-breathing techniques can be utilised.

The biopsychosocial model can explain non-mechanical or visceral complaints (“Type O” disorders), which have been treated with anecdotal success by chiropractic therapy. In the case of studies described in this paper, thyroid dysfunction (a “Type O” condition) was successfully treated with the identification of a meridian imbalance related to anxiety. As described above, the premise is that there exists a psychosocial component to the physical presentation of the visceral complaint. Functional cardiovascular and functional bowel disorders are two conditions (among others) that have long been identified as having social and psychological factors as confounders.^{36,41,42} Chronic pain

syndromes have also been shown to have a psychosocial component.^{37,54,55} Research has shown that depressive mood states and distress can be causative or prolong the natural history of chronic pain states.⁵⁶ Herein lie the most likely explanations for the positive results seen by physical therapists when treating patients with thyroid dysfunction in the case reports above. This is also the likely explanation for other "Type O" disorders successfully treated by manual medicine practitioners. Therapy that achieves a good result has addressed the associated psychosocial component to the condition as well as the physical component.

CONCLUSION

NET has produced a successful clinical outcome demonstrated by objective pre- and post-treatment measurements in two cases of hypothyroidism without a need for medication. A spontaneous remission of the condition may explain the improvement, but given the chronicity of the complaint and the time frame of improvement after implementation of therapy, we hypothesise that the improvement resulted from the NET therapy used in the treatment. Since this is a case report, not a controlled trial for hypothyroid treatment, any conclusion drawn must be carefully considered. We therefore recommend that a randomised controlled trial of the effect of NET therapy in hypothyroid patients be conducted to ascertain if this phenomenon is real under controlled conditions, or purely coincidental.

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