

Functional Thyroid Disorders, Part I

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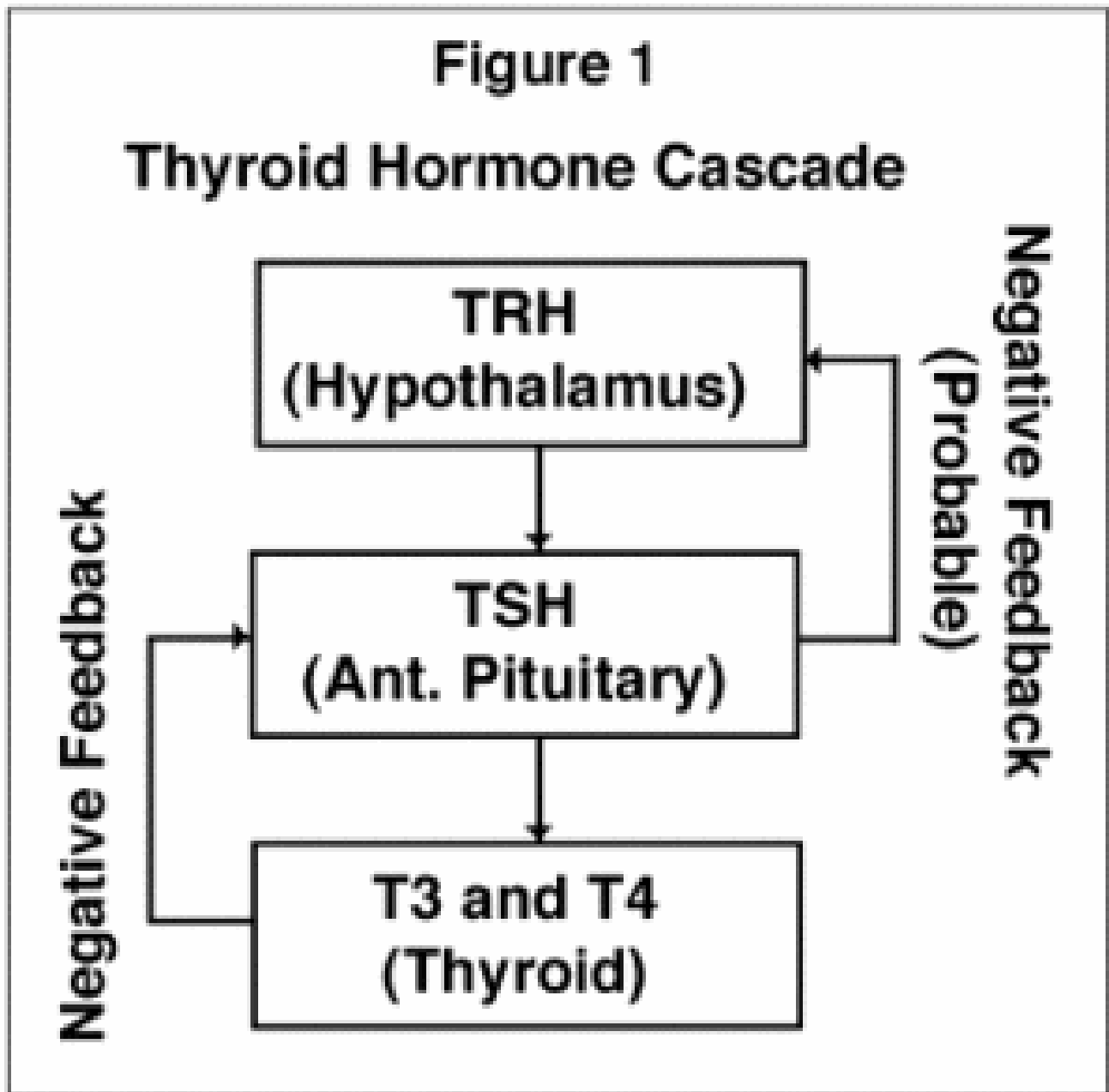
Thyroid disorders are among the most commonly encountered metabolic disorders in private practice. The strong tendency for thyroid disorders to cause musculoskeletal symptoms make the understanding of these disorders even more critical to the practicing chiropractor.

In this two-part article, I will attempt to provide the reader with an overview of thyroid disorders, along with suggestions on proper diagnosis and treatment. I will explain why conventional medicine does not recognize many cases of thyroid disease, and why many patients diagnosed with thyroid disease do not respond to conventional therapy. Part I will provide information on hypothyroidism, with Part II providing information on hyperthyroidism and thyroid cancer. A follow-up article on fibromyalgia will discuss the current state of research and treatment of this complex metabolic disorder, including the strong role that thyroid dysfunction plays in many cases of fibromyalgia and fibromyalgia-like disorders.

The thyroid gland is located in the anterior neck region and normally weighs approximately 15-20g. It consists of two lobes: one on either side of the trachea in the lower neck, and a connecting portion called the isthmus, giving the entire gland an H-shaped appearance. The thyroid gland is responsible for synthesizing several hormones that have vast effects on overall body metabolism. It is unique among endocrine glands in that large amounts of hormones are created and stored in the thyroid and then released very slowly.¹ Iodine ingested from food and water is concentrated by the thyroid gland and combined with the amino acid tyrosine in various chemical configurations to create the active thyroid hormones triiodothyronine (T3) and thyroxine (T4). The numbers 3 and 4 are used to identify the hormones that are related to the number of sites iodinated on the hormone structure. All reactions necessary for the formation of T3 and T4 are influenced and controlled by thyroid stimulating hormone (TSH) produced in the pituitary gland.

TSH secretion is controlled by a negative feedback mechanism modulated by circulating levels of T4,

and to a lesser degree T3. Decreased serum levels of T3 and T4 result in an increase in TSH production and release by the anterior pituitary gland. Conversely, increased levels of T3 and T4 result in a reduction of TSH formation and release. TSH secretion is also influenced by the increased production and secretion of thyroid releasing hormone (TRH) produced in the hypothalamus^{2,3}. The resulting thyroid hormone cascade is summarized in Figure 1.



The overwhelming majority (99%) of circulating thyroid hormones are bound to serum proteins, mostly thyroid-binding globulin (TBG). The small percentage of free thyroid hormones, those not bound to TBG, act as the active form, since only the free hormones can enter cells and bind to the nuclear hormone receptor. The nuclear hormone receptor regulates DNA control of oxidative processes, thereby altering metabolism throughout the body.⁴ Generally speaking, lower levels of thyroid hormone entering cells will slow their metabolism, while higher levels will increase their metabolism.

Prevalence rates of thyroid disorders in general vary depending on the study and generally show increased rates in women.⁵ In a literature review by Helfand and Crapo, the prevalence of new cases of overt hypothyroid and hyperthyroid was estimated to be 0.5% of the U.S. population.⁶ A German study reported a prevalence of overt hypothyroidism of 0.03% and overt hyperthyroidism of 0.9% in a healthy population of workers.⁷ These rates may be significantly skewed to the low side, since in this study of healthy workers, women were under represented (94% of the subjects were men. Both of the previously mentioned prevalence studies were looking for the prevalence of "overt" thyroid disease, rather than the much more prevalent low-grade "functional" thyroid disorders that are more likely to present to the chiropractic office. Studies by Rallison, et al., demonstrated a clear increase in prevalence of thyroid disorders with age.⁸

Hypothyroidism

Various types of hypothyroid states exist: primary (disease of the thyroid gland itself); secondary, aka "pituitary" (due to lack of TSH production); late Hashimoto's thyroiditis (auto-immunity against the thyroid); iodide deficiency goiter; genetic thyroid enzyme defects; conversion of T4 to T3 defects; thyroid receptor insensitivity; and drug induced hypothyroidism, i.e., lithium, sulfonamides, phenylbutazone, oral contraceptives, etc.^{2,4} All of these disorders can result in a functional hypothyroidism and a very similar clinical presentation.

Lowered function of the thyroid gland, regardless of the cause, can result in profound physiologic effects throughout virtually all systems of the body, due to the effects of lowered temperature on enzyme function. General signs and symptoms include fatigue, weakness, cold intolerance, low-morning axillary temperatures (normally 97.8 degrees F to 98.2 degrees F, taken for 10 minutes before getting out of bed and averaged over five days), weight changes (usually weight gain) and depression.⁴ (See Table 1).

Table 1

Hypothyroid S/Sx's

- | | |
|----------------------|----------------------|
| • fatigue, lethargy | • weakness |
| • weight gain | • muscle cramp |
| • dry, coarse skin | • arthralgias |
| • cold intolerance | • paresthesia |
| • swelling of hands | • memory loss |
| • legs and face | • carpal tunnel syn. |
| • chronic infections | • constipation |
| • postnasal drip | • dysglycemia |

All female patients complaining of fatigue should have axillary temperatures taken, particularly if they are also complaining of muscle tenderness and/or other musculoskeletal symptoms. Common musculoskeletal signs and symptoms include muscle pain, stiffness, muscle cramping, muscle weakness, paresthesias, arthropathy and sluggish deep-tendon reflexes.⁹ Other reported musculoskeletal-related symptoms associated with hypothyroidism include adhesive capsulitis of the shoulders, proximal myopathy, carpal tunnel syndrome, and polyneuropathy, which tends to be primarily sensory but sometimes shows motor weakness as well.^{10,11} The incidence of musculoskeletal symptoms with hypothyroidism has been reported by Khaleeli et al. to be as high as 30-80 percent, depending on the special interests of the diagnosing physician.¹² Many of these musculoskeletal symptoms are thought to result from myxedematous infiltration of ligaments and muscles.

As the primary interest and expertise of the average chiropractor is musculoskeletal conditions, the yield for the chiropractor in picking up the musculoskeletal-related symptoms of hypothyroidism, and indeed hyperthyroidism, should be expected to be at the higher end of the range reported by Khaleeli. This is extremely important since it is precisely these musculoskeletal symptoms, which may drive the patient to the chiropractor initially. In fact, the listing of these symptoms reads surprisingly like the typical chiropractic yellow page advertisement. Many of these patients will likely be unaware that they

have a thyroid condition, or that their thyroid condition is causing the musculoskeletal symptoms. This provides an opportunity for the astute chiropractor to make the initial diagnosis, fulfilling the role of a portal of entry physician, while at the same time allowing the patient to avoid the profound, and often life-altering, complications of hypothyroidism by being directed to further work-up and proper treatment.

The most useful serum laboratory tests to detect hypothyroidism are total T4, the free thyroxine index (FTI), and thyroid stimulating hormone (TSH).^{1,13} Laboratory findings include a low or low normal T4. TSH is usually increased in primary hypothyroidism as the pituitary attempts to increase thyroid output, but it is low or normal in secondary hypothyroidism (aka pituitary insufficiency). Serum T3 is an unreliable test to detect hypothyroidism. Serum thyroperoxidase and thyroglobulin antibody titers are high only in cases of autoimmune processes causing hypothyroidism, such as in Hashimoto's thyroiditis. Other associated lab findings of hypothyroidism may include hypercholesterolemia, increased liver enzymes, increased creatine kinase, hypoglycemia, albuminuria and anemia.^{1,4,13} (See Table 2.)

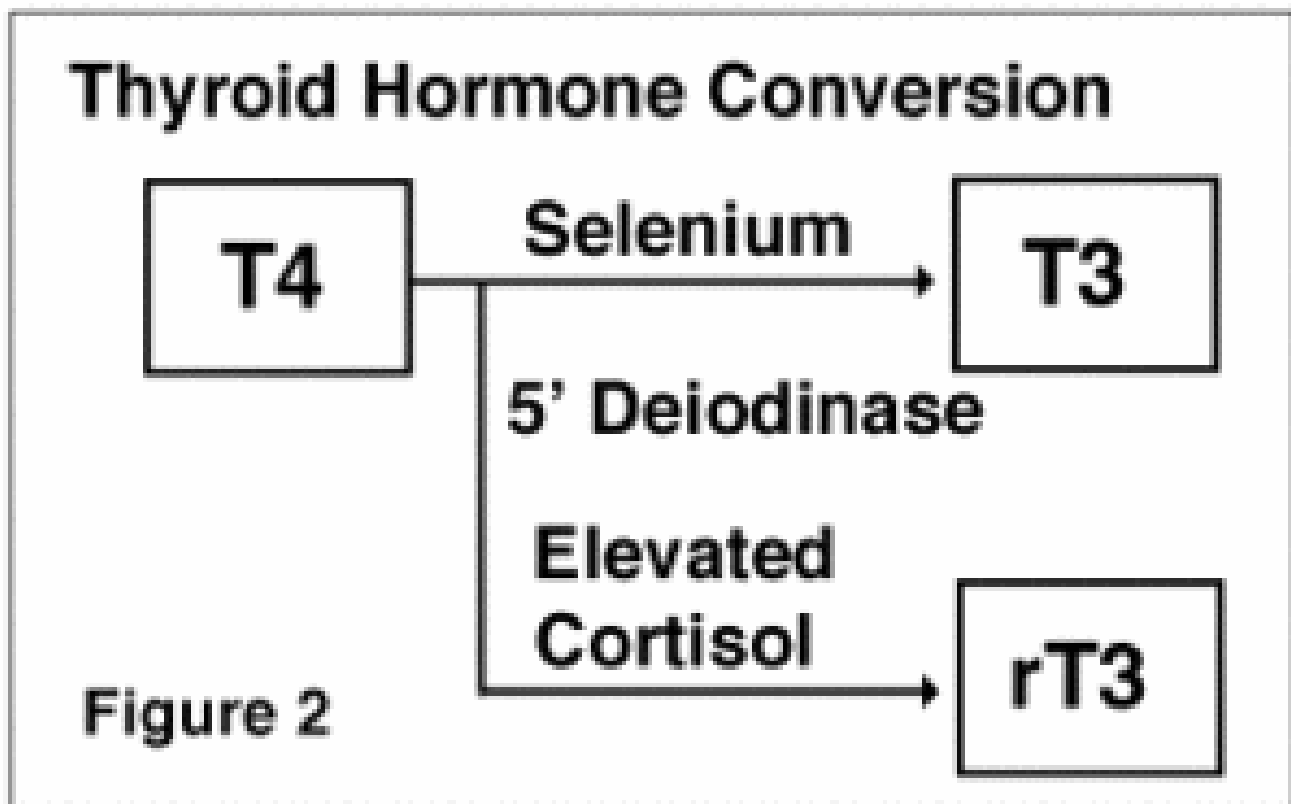
Table 2: Laboratory findings in common thyroid disorders										
Thyroid Disorder	T4	T3	rT3	T3 resin uptake	Free T4 index	Free T4	TSH	TRH test	Thyroglobulin	TRAb
Hypothyroidism "primary"	L	L	L	L	L	L	H	H	N	-
"secondary (pituitary)"	L	L	L	L	L	L	N/L		N	-
Hyperthyroidism	H	H	H	H	H	H	L	L	N/H	-
Grave's disease " (thyrotoxic phase)"	H	H	H	H	H	H	L	L		+
Hashimoto's thyroiditis	V	V	V	V	V	V	V	V		++
Euthyroid sick syndrome, conversion disorders, peripheral receptor resistance	N/L	N/L	H	N/L	N/L	N/L	V	N/L		-
T4 = thyroxine; T3 = triiodothyronine; rT3 = reverse triiodothyronine; TSH = thyroid-stimulating hormone; TRH = thyroid-releasing hormone; TRAb = thyrotropin-receptor antibodies; H = high; L = low; N = normal; V = variable; - = negative; + = positive										

Common nonthyroidal diseases, including surgical stress and serious infection, can result in what appears to be hypothyroidism on laboratory testing. This clinical entity is termed "euthyroid sick syndrome" and must be ruled out primarily by historical information and subtle differences in laboratory results as compared to true hypothyroidism.¹⁴

Serum studies often miss cases of mild hypothyroidism, because hypothyroid patients tend to have low blood volume, which produces a concentration effect, resulting in thyroid hormones being interpreted

as normal when they are low. The TSH test, which would be expected to be high in cases of primary hypothyroidism, can also be falsely interpreted as normal since a hypothyroid state can produce adverse cellular effects upon the pituitary, resulting in decreased TSH production. The urinary excretion test of metabolites of thyroid and adrenal hormones, as well as electrolytes,¹⁵ provides an alternative method of checking the function of the thyroid and adrenal glands. This test is preferred by many functionally minded clinicians for its ability to pick up milder cases of hypothyroidism, and its ability to relate thyroid to adrenal function. However, this test is quite expensive and has a very long turnaround time. It is important to evaluate adrenal status, since adrenal and thyroid function are so interdependent.

A study which is much more cost effective than the Barnes test to evaluate adrenal function is the "adrenal stress index," a urinary and salivary test which measures cortisol at several times during the day, as well as DHEA. This test is offered by many functional medicine labs. Adrenal function is critical in the conversion of T4 to T3, which is 10 times more active than T4, in peripheral tissues. This conversion of T4 to T3 is influenced by adrenal cortisol, iron, selenium, B12 and magnesium. Too much cortisol in the system can induce a conversion of T4 to an improper form of T3 called reverse T3 (rT3), as shown in Figure 2.



Reverse T3 is not recognized by peripheral thyroid hormone receptors and has little to no activity on cellular metabolism. Too little cortisol, or other nutrients listed above, can result in a slowed conversion of T4 to T3. Therefore, borderline hypoadrenalism or hyperadrenalism can result in functional hypothyroidism and persistently low axillary temperatures, even when the patient is on significant dosages of exogenous thyroid hormones. Adrenal dysfunction can be addressed with stress reduction, proper sleep, the use of nutrients such as vitamin C, vitamins B5 and B6 and adaptogenic

herbs, such as ginseng compounds, licorice root (*glycyrrhiza glabra*), withania (*ashwagandha*) and others.^{16,17}

Stressful physiological conditions, such as pregnancy and trauma, can induce a hypercortisol condition and are commonly determined during the medical history to correlate with the onset of the patient's thyroid-related symptoms. Often, in cases such as this, when adrenal function is normalized, the thyroid follows suit spontaneously. Depressed liver function, due to a toxic or sluggish liver, can also influence the conversion of T4 to T3 via cytochrome P450 activity, resulting in low T3 levels (see my previous article on "Functional Medicine" for more information on systemic toxicity and hepatic function (*DC*, Vol 17, No.8, April 5, 1999, or at <http://www.chiroweb.com/archives/17/08/04.html>). In general, the use of a combination of urinary excretion tests and morning axillary temperatures is far superior to serum studies alone, which miss many cases of mild hypothyroidism.

The current conventional medical treatment of choice is hormone replacement therapy with the drug Levothyroxine (Synthroid) which contains a synthetic version of T4, which is then converted in the body to T3, the most active of the thyroid hormones.⁴ This certainly presents problems in patients with hypothyroidism due to conversion defects. For some patients with very mild hypothyroid states, nutritional supplementation to support thyroid function and proper metabolism of thyroid hormones includes; L-tyrosine, iodine, selenium, B-vitamins and thyroid glandular concentrate. This may provide enough therapy to avoid the need for hormone replacement therapy, or at least lessen the dosage required.^{16,17,18} Dosages of these nutrients must be adhered to strictly, since excessive thyroid glandular concentrate has been anecdotally reported to trigger autoimmune-type thyroid disorders in some cases, and iodine supplementation greater than 150 mg/day can suppress thyroid function (see Table 3).

Table 3:
**Suggested nutrition supplementation
program for mild hypothyroidism**

L-Tyrosine	500 mg b.i.d.
Atlantic Sea Kelp (Iodine Source)	150-200 mg per day
Thyroid Glandular	250-500 mg per day
Multivitamin and Mineral Complex	As directed on label
B-Complex	100 mg t.i.d.

Monitoring with periodic laboratory assessment of thyroid function and axillary temperatures is required to assess the success, or lack thereof, of nutritional therapy. Unfortunately, most cases of hypothyroidism will require the use of hormone replacement medication. An alternative to the commonly used pharmaceuticals, such as Synthroid, Levothyroid, and Levoxyl, which only contain L-thyroxine (T4), is Armour thyroid. Armour thyroid, unlike the previously mentioned thyroid glandular nutritional supplements, which contain no active hormone, is a pharmaceutical preparation of purified desiccated pork thyroid tissue, which contains significant levels of both T3 and T4. It commonly provides a smoother onset, less toxicity, and a better clinical and symptomatic response than synthetic compounds. This is particularly true in those patients suffering from T4 to T3 conversion disorders, who often do not fair well on synthetic T4 (Synthroid). Armour thyroid is available by prescription only. To find a prescribing physician in your area aware of functional thyroid disorders, and who dispenses Armour thyroid, call the Broda Barnes Foundation.¹⁵ In part II of this article, we will discuss hyperthyroidism and thyroid cancer.

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