MONOGRAPH

Thyroid Stimulating Hormone: An Update to TSH Ranges

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Abstract

Thyroid dysfunction can lead to the disruption of metabolism characterized by a wide variety of symptoms. Within an elaborate feedback system, the hypothalamus, pituitary, and thyroid glands work together to maintain optimal levels of circulating thyroid hormones. The enduring question has become how to detect early changes in thyroid metabolism and address them before dysfunction progresses to disease.

The standard screening test for thyroid dysfunction is thyroid stimulating hormone (TSH), a hormone produced in the pituitary with direct effects on the thyroid gland. A reduction in circulating thyroid hormone will lead to an increase in TSH, while an increase in thyroid hormone will decrease TSH. An abundance of research suggests that the current range of values used to evaluate TSH are too broad and fail to identify thyroid dysfunction. Instead, clinicians are calling for a narrower optimal range for TSH that will better reflect optimal health and metabolism. Utilizing an optimal range for TSH of 1-2 mU/L will allow the clinician to identify very early signs of potential dysfunction.

Introduction

The ultimate goal for any clinician is to identify and help resolve issues presented by their patients. The ultimate challenge may be to pinpoint exactly what underlies the disorder at hand. Accurate assessment of thyroid function is especially important given its essential role in vascular integrity, energy expenditure, and lipid and glucose metabolism.[1] Insufficiency of thyroid hormone is directly associated with insulin resistance, dyslipidemia, diastolic dysfunction, diastolic hypertension, and endothelial dysfunction.[2]

Early thyroid abnormalities may not be suspected or detected using current clinical and laboratory guidelines. Dysfunction may then be allowed to progress until conspicuous symptoms and lab abnormalities reveal overt pathology. Reevaluating how we assess thyroid function allows the clinician to identify trends toward disease and apply early interventions to help maintain optimal health. Current research supports revision of reference ranges for thyroid stimulating hormone (TSH) in order to detect and address early dysfunction.

This review will address

- ✓ Assessing thyroid function
- ✓ Hypothyroidism and hyperthyroidism signs, symptoms, and risk factors
- ✓ Subclinical thyroid disease
- ✓ Laboratory evaluation of thyroid function with a focus on TSH
- ✓ Standard and optimal reference ranges for TSH
- ✓ Brief overview of thyroid replacement therapy
- ✓ Optimal Takeaways

Assessing Thyroid Function

In the past, a thyroid disorder may not have been diagnosed until serious consequences such as exophthalmos, goiter, or physiological disturbance had occurred. Indeed, undiagnosed hypothyroidism may have progressed to a debilitating stage characterized by psychological, neurological, and physiological decline before a diagnosis was made. In some cases, severe hypothyroidism or "myxedema" was even fatal.[3] Now clinicians are able to identify early signs of hypothyroidism (insufficient production of thyroid hormones) and hyperthyroidism (excess production of thyroid hormone) and address them before dysfunction progresses.

Hypothyroidism

Hypothyroidism is particularly prevalent, affecting an estimated 5 out of 100 residents in the United States, with many individuals going undiagnosed. The most common cause of primary hypothyroidism worldwide is iodine insufficiency. However, in the United States the main cause is autoimmune Hashimoto thyroiditis. Hashimoto's causes increased turnover of iodine, chronic inflammation, T-cell lymphocyte infiltration, and fibrosis of the thyroid. It is also associated with papillary cancer and is strongly associated with lymphoma.[4] Additional causes of hypothyroidism include medications, radiation, and thyroid surgery.

Inability of the thyroid to produce adequate thyroid hormone is considered primary hypothyroidism. If pathology is related to the hypothalamus or pituitary gland, the condition is considered secondary hypothyroidism, though this form is relatively rare.[5]

Risk Factors for Hypothyroidism Include:[6] [7] [8]

- ✓ Autoimmune disorders
- ✓ Celiac disease
- ✓ Diabetes, type 1 or 2
- ✓ Family history of thyroid disease
- ✓ History of head and neck radiation
- ✓ Lupus
- ✓ Mercury exposure
- ✓ Pernicious anemia
- ✓ Pregnancy in past six months
- ✓ Previous thyroid issue such as goiter
- ✓ Rheumatoid arthritis
- ✓ Sjogren's syndrome
- ✓ Thyroid peroxidase antibodies
- ✓ Turner syndrome
- ✓ Women over age 60

Left untreated, the metabolic deterioration that occurs with thyroid deficiency can contribute to cognitive dysfunction, hyperlipidemia, hypertension, neuromuscular dysfunction, and infertility.[9]

Hypothyroidism Signs and Symptoms[10] [11] [12] [13]

- ✓ Anxiety, depression
- ✓ Cognitive changes, memory loss
- ✓ Cold intolerance
- ✓ Congestive heart failure
- ✓ Constipation
- ✓ Decreased sweating
- ✓ Decreased thyroid hormones
- ✓ Difficulty losing weight
- ✓ Dry skin
- ✓ Emotional lability
- ✓ Fatigue
- ✓ Fertility issues
- ✓ Forgetfulness
- ✓ Hair loss, thinning hair
- ✓ Headaches
- ✓ Hyper-cholesterolemia
- ✓ Joint and muscle pain
- ✓ Hyponatremia
- ✓ Fatigue, lethargy
- ✓ Memory problems
- ✓ Menstrual irregularities
- ✓ Mental slowness
- ✓ Myxedema (severely compromised metabolism)
- ✓ Prolonged QT interval
- ✓ Psychosis
- ✓ Recurrent infections
- ✓ Sensitivity to cold
- ✓ Skin changes, dry skin
- ✓ Sleep disturbance
- ✓ Slowed heart rate
- ✓ Voice changes
- ✓ Rare symptoms include rhabdomyolysis, ascites, pericardial effusion

Differential Diagnoses[14]

Signs and symptoms of hypothyroidism can overlap with other disorders that may need to be ruled out during clinical workup.

- ✓ Addison's disease
- ✓ Anemia
- ✓ Chronic fatigue syndrome
- ✓ Depression
- ✓ Dysmenorrhea
- ✓ Erectile dysfunction
- ✓ Euthyroid sick syndrome
- ✓ Familial hypercholesterolemia
- ✓ Goiter
- ✓ Infertility
- ✓ Iodine deficiency
- ✓ Riedel thyroiditis
- ✓ Subacute thyroiditis
- ✓ Thyroid lymphoma

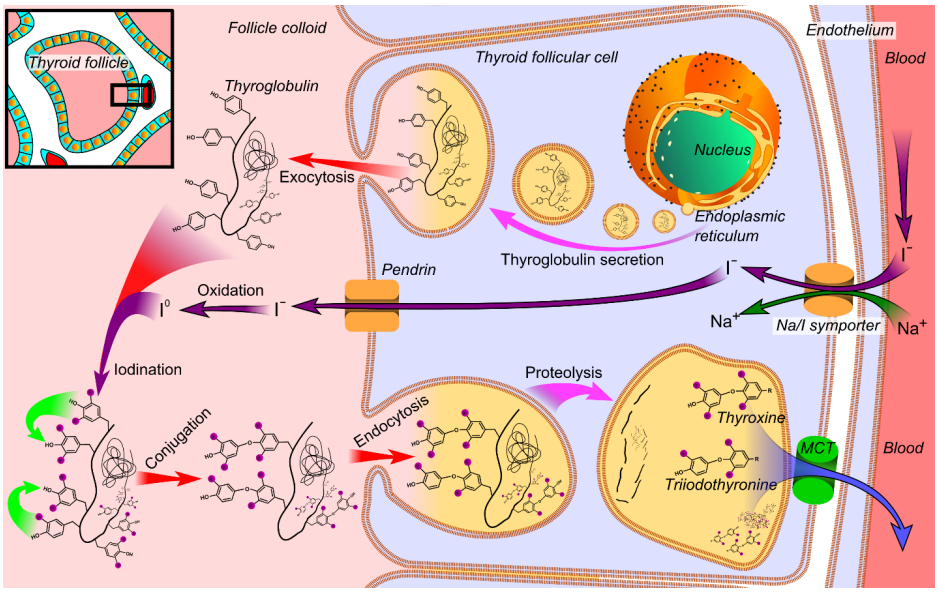
Hyperthyroidism

Hyperthyroidism is not as prevalent as hypothyroidism, though it is significantly more prevalent in women than men. The most common cause of hyperthyroidism in Western countries is autoimmune Graves' disease, especially in younger individuals. Other causes include toxic multinodular goiter, iodine-induced hyperthyroidism, thyroid adenomas, subacute thyroiditis, postpartum thyroiditis, and factitious thyroiditis (due to excess pharmaceutical thyroid hormone).[15]

Hyperthyroidism Signs and Symptoms[16]

- ✓ Diaphoresis
- ✓ Diarrhea or increased GI motility
- ✓ Diffuse enlargement of the thyroid, non-painful goiter
- ✓ Fatigue
- ✓ Heat intolerance
- ✓ Hypermetabolism
- ✓ Increased thyroid hormones
- ✓ Irritability
- ✓ Menstrual irregularities
- ✓ Muscle weakness
- ✓ Palpitations
- ✓ Nervousness
- ✓ Racing heartbeat
- ✓ Skin warm, thin, moist
- ✓ Sweating
- ✓ Thyrotoxicosis
- ✓ Tremors
- ✓ Weight loss

Signs and symptoms of hyperthyroidism may be due to thyrotropin-secreting pituitary adenoma though this is a rare phenomenon.[17]



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Influencing Factors

Thyroid hormone and TSH concentrations may be influenced by factors unrelated to thyroid disease including acute or chronic illness, age, gender, body mass index, ethnicity, iodine status, and type of assay used.[18] [19] [20] Long-term use of some medications can influence levels of thyroid hormones and TSH as well.

Common Causes of Abnormal TSH[21]

Increased TSH

- ✓ Antithyroid medication
- ✓ Congenital hypothyroidism
- ✓ Lithium
- ✓ Pituitary TSH-secreting tumor
- ✓ Potassium iodide, large doses of iodine
- ✓ Primary hypothyroidism
- ✓ Severe and chronic illness
- ✓ Thyroid agenesis
- ✓ Thyroiditis
- ✓ TSH injection

Decreased TSH

- ✓ Aspirin
- ✓ Dopamine
- ✓ Heparin
- ✓ Hyperthyroidism
- ✓ Pituitary dysfunction
- ✓ Secondary hypothyroidism
- ✓ Severe illness
- ✓ Steroids
- ✓ T3

Age-related changes may be anticipated as well. For example, after age 30-39, the 97.5th percentile of serum TSH may increase by 0.3 mU/L.[22] TSH levels are also affected by pregnancy and specific guidelines are provided according to trimester. However, even these guidelines continue to be debated and adjusted.[23] [24]

Intestinal dysbiosis and alterations in bacterial composition may affect thyroid hormone levels and TSH production due to the role microbiota play in availability of nutrients and metabolism of thyroid hormones.[25] [26]

An individual may even have a unique set-point for thyroid hormones at which thyroid function is optimized. This range can be influenced by genetic factors.[27] Ideally, assessment of lab results, symptomatology, history, and overall clinical presentation will be used in determining target ranges.[28]

As always, a comprehensive clinical assessment is warranted before determining presence of disease, clinical goals, or treatment for each patient. Individual variations in TSH, T3, and T4 should be observed over time in order to determine each individual's optimal care plan.[29]

Subclinical Thyroid Disease

Subclinical thyroid disease is characterized by an abnormal TSH accompanied by a standard normal free thyroxine (T4). Serum TSH will be low at 0.1-0.45 or elevated at 4.5- 10 mU/L.[30] Subclinical disease is associated with increased risk of morbidity, biochemical abnormalities, and physiological impairment. Undetected thyroid dysfunction can contribute to metabolic and organ complications, and progressively worsen existing metabolic syndrome, diabetes, kidney dysfunction, and heart failure prognosis.[31] [32]

Subclinical hypothyroidism (SCHypo), characterized by elevated TSH with normal free T4, occurs in 4-10% of the developed world, while overt hypothyroidism is found in approximately 5%.[33] Generally, if TSH above 4 mU/L persists after repeated measurements with a normal free T4, subclinical hypothyroidism is considered.[34]

Determination of subclinical hypothyroidism depends on stable thyroid function (greater than 2 weeks or more), the absence of acute or critical illness, and a normal hypothalamic-pituitary-thyroid axis.[35]

Traditionally, treatment of subclinical hypothyroidism may not be considered until TSH exceeds 10 mU/L.[36] However, a TSH of greater than 10 mU/L may already have debilitating consequences, including increased risk of heart failure and cardiovascular mortality.[37]

Subclinical hypothyroidism itself is associated with elevated cholesterol, blood pressure, and homocysteine; congestive heart failure; fatal and non-fatal coronary artery disease; fatal stroke; and decreased muscle strength and exercise capacity. It may be asymptomatic but can also present with a range of symptoms associated with hypothyroidism including:[38]

- ✓ Integumentary: Dry skin, hair loss, loss of outer 1/3rd of eyebrows, facial puffiness
- ✓ Gastrointestinal: Constipation, dysphagia, loss of appetite, weight gain, cholelithiasis
- ✓ Cardiovascular: Diastolic hypertension, bradycardia, pericardial effusions
- ✓ Neurological: Decreased attention span, pseudodementia, mononeuropathies (most commonly carpal tunnel syndrome)
- ✓ Musculoskeletal: Muscular weakness, cramps, stiffness, fatigue
- ✓ Reproductive: Irregular periods, decreased libido

Evaluation of data from 9020 participants in a series of NHANES studies revealed a U-shaped association between TSH and risk of all-cause mortality. Researchers found that individuals with subclinical hypothyroidism and 'high-normal" TSH between 1.96-5.6 mU/L had increased risk of cardiovascular disease and increased all-cause mortality. Unfortunately, the "high-normal" category was not broken down to further differentiate ranges in this evaluation. A "low-normal" TSH of 0.34-1.19 mU/L was associated with increased risk of all-cause mortality as well. Those with a "middle-normal" TSH of 1.2-1.95 mU/L had the lowest risk of all-cause mortality.[39]

A 2018 meta-analysis of 21 studies comprising 2192 randomized adults receiving thyroid hormone treatment for subclinical hypothyroidism found that therapy reduced mean serum TSH to 0.5-3.7 mU/L versus 4.6-14.7 mU/L with placebo. However, overall results suggest that this shift in TSH did not translate into improvement of quality of life or thyroid-related symptoms.[40] It is possible that treatment may have brought TSH into "normal" lab range but not optimal lab range.

Subclinical hypothyroidism may be overlooked despite its potential complications including "infertility, pregnancy complications, psychiatric illness, neuromuscular symptoms, cardiac dysfunction, and mortality." [41]

In fact, subclinical and overt hypothyroidism are so closely linked to heart disease and heart failure that a TSH screening is recommended for all individuals newly diagnosed with heart failure or type 1 diabetes.[42]

Approximately 50% of individuals with subclinical hypothyroidism will progress to primary hypothyroidism within 20 years.[43]

Subclinical Thyroid Disease and Cardiometabolic Disorders

Subclinical *hyper*thyroidism may have low or undetectable TSH with free T4 and free T3 within standard range. It has been associated with atrial fibrillation, as well as coronary heart disease events and mortality.[44]

Subclinical *hypo*thyroidism, with elevated TSH and normal free T4, is associated with insulin resistance and metabolic syndrome. It is also associated with individual components of metabolic

syndrome including central obesity, hypertension, hypertriglyceridemia, and low HDL as demonstrated in meta-analysis of 19 studies comprising 79,727 subjects.[45] Another meta-analysis of 11 studies comprising over 55,000 individuals demonstrated an increased risk of cardiovascular events and fatality in those with subclinical hypothyroidism, especially as TSH increased.[46]

An early double blind placebo-controlled study demonstrated that thyroid replacement therapy in subclinical hypothyroidism significantly reduced total and LDL cholesterol and apolipoprotein B-100. Therapy also significantly improved clinical symptomatology.[47] In another trial, treatment with levothyroxine (synthetic T4) for 18 months normalized elevated cholesterol, homocysteine, and blood pressure in women with subclinical hypothyroidism.[48]

It is clear that further investigation beyond "normal standard ranges" can reveal underlying metabolic dysfunction that should be addressed before significant disease sets in.

Laboratory Evaluation of Thyroid Function

Typical signs and symptoms associated with hypo- and hyper- thyroid disease are not specific enough to be diagnostic. Therefore, biochemical evaluation must be utilized.[49] [50]

Thyroid disorders may be evaluated using a variety of laboratory tests including[51]

- ✓ Thyroid stimulating hormone (TSH), also called thyrotropin or pituitary thyrotropin
- ✓ Triiodothyronine (T3), the active form of thyroid hormone, total and free levels should be evaluated
- ✓ Thyroxine (T4), basically inactive hormone. Total and free levels should be evaluated.
- ✓ Antibodies to thyroglobulin, thyroid peroxidase, and TSH receptors

Thyroid Stimulating Hormone (TSH)

Serum TSH is considered the best initial screening marker for thyroid dysfunction in most individuals.[52] Evaluation of free T4 and anti-TPO antibodies should be considered in screening as well, especially if initial TSH levels are abnormal.[53] Further evaluation of free hormone levels will provide information about the nature of the dysfunction.

TSH is produced in the anterior pituitary under the regulation of thyrotropin releasing hormone (TRH) from the hypothalamus. Circulating TSH stimulates the thyroid gland to secrete T4, ~100-125 nmol per day, as well as small amounts of T3. Circulating T4 will be enzymatically converted to active T3 in the periphery by deiodinases. Increasing levels of T3 will then feedback and reduce production of TRH, TSH, and T4. This feedback loop, and its associated organs, must be in balance and functional for thyroid status to remain normal. In hypothyroidism, production of T4 declines and increasing TSH will promote hypertrophy and hyperplasia of thyroid tissue, leading to increased T3 production.[54]

TSH is a sensitive marker of thyroid dysfunction as long as pituitary and hypothalamus function are normal.[55] The feedback loop between TSH and thyroid hormones is a sensitive one. Small changes in TSH reflect small changes in free T3 and free T4, and greater changes in TSH reflect greater changes in free T3 and free T4.[56]

TSH also appear to regulate production and activity of deiodinase enzymes and conversion of T4 to T3. Increased conversion will alter the ratio of free T3 to free T4 (FT3/FT4) and provide further clues to underlying metabolic changes. A cross-sectional study of 132,346 adults examined the relationship between TSH, FT3, FT4, and metabolic dysfunction. Even though levels of TSH, FT3, and FT4 were within standard range, the FT3/FT4 ratio was positively associated with TSH and positively associated with risk of insulin resistance and metabolic syndrome. Researchers suggest

that FT3/FT4 ratio may be a more effective predictor of metabolic syndrome than TSH, indicating that more in depth assessment of thyroid hormones may be prudent in apparently euthyroid subjects.[57]

In addition to aberrant thyroid-related biomarker levels, additional biochemical abnormalities observed with overt hypothyroidism can include anemia, hyperlipidemia, and elevations in hepatic enzymes, uric acid, BUN, creatinine, and serum creatine kinase. [58]

TSH Standard Reference Range

Quest Diagnostics TSH[59]	≥20 Years	0.40-4.50 mU/L
Labcorp TSH[60]	>10 Years	0.45-4.5 mU/L
Mosby's[61]	Adult:	2-10 mU/L

The standard reference range for TSH has evolved over time. The lower limit, ~2.5th percentile, is between 0.2 and 0.4 mU/L. The accepted upper limit for TSH has decreased from an historical 10 to 5-7 to 4.5 mU/L,[62] [63] and finally to 4.12 mU/L based on NHANES III data. [64] However, even the NHANES III upper limit was thought to be skewed higher due to occult thyroid dysfunction that went undetected.[65]

Practitioners recognize that the current standard reference range may be too broad to reliably identify hypothyroidism and a TSH within standard range does not rule it out. Research confirms these observations.

- ✓ A 2013 literature review reveals significant risk of cardiovascular and metabolic outcomes with a TSH in the upper end of standard lab range including positive associations with[66]
    - Blood pressure
    - Cholesterol and lipid levels
    - Cardiovascular mortality in women (possible)
    - BMI
    - Metabolic syndrome
- ✓ Symptomatic patients with a TSH between 1.33 and 2.65 mU/L were found to be hypothyroid upon further evaluation of total and free T4 and T3.[67]
- ✓ Antithyroid antibodies are more prevalent when TSH levels rise above 2.5 mU/L.[68]
- ✓ Median values for TSH range from 1-1.5 mU/L and a significant percentage (~30%) of individuals with a value above 3 mU/L have autoimmune thyroid disease.[69]
- ✓ A cross-sectional study of 1333 subjects found levels of 2.5-4.5 mU/L to be associated with obesity, hypertriglyceridemia, and increased risk of metabolic syndrome. Individuals in this group had: [70]
    - Significantly higher BMI
    - Significantly higher fasting triglycerides
    - A 1.7 fold increase for fulfilling metabolic syndrome criteria
    - A TSH below 2.5 mU/L was associated with a more favorable metabolic profile

It is important to recognize that while labs may be considered "normal," an individual's health may be far from normal.

What Is an Optimal TSH?

An optimal TSH should reflect optimal functioning of the hypothalamus-pituitary-thyroid axis.[71]

Although the commercial standard reference range for adults is currently ~0.45-4.5 mU/L, the therapeutic TSH target range for thyroid hormone treatment is lower at 0.3-3.0 mU/L.[72]

Some clinicians call for an even narrower reference range for TSH. Accumulating research suggests levels between 0.4 and 2 mU/L may prevent progressive derangement of TSH values.[73] However, other researchers note that suppression of TSH below 1 mU/L may compromise bone density and contribute to an increased risk of vertebral fracture and is not advised.[74]

A prospective survey indicates that actual thyroid dysfunction was lowest with a serum TSH between 1 and 1.9 mU/L; incidence of hyperthyroidism increased when serum TSH fell below 1 mU/L and hypothyroidism increased as TSH increased above 1.9 mU/L.[75]

The upper TSH limit has garnered the most attention.

Research suggests that more than 95% of healthy individuals maintain a TSH level less than 2.5 mU/L.[76] Officially adjusting the upper limit from ~ 4-4.5 mU/L to 2.5 mU/L is being considered in wider clinical circles.[77] [78] [79] [80] [81] [82]

The National Academy of Clinical Biochemists (NACB) specifically recommends an upper limit of 2.5 mU/L for TSH even without clinical evidence of thyroid disease.[83] This upper limit is currently used by many clinicians as a guide for monitoring thyroid hormone balance and sufficiency. It is important to determine whether "normal" TSH values between 2.5-4.12 mU/L reflect early hypothyroidism so that early intervention can be initiated.[84] Asymptomatic individuals with a TSH between 3-4.5 mU/L and above, should be monitored carefully for progression of disease, especially if anti-TPO antibodies are present.[85]

A TSH above 2.5 mU/L in hypothyroid patients is associated with depression. A 2017 review of depressive symptoms was conducted on hypothyroid patients on levothyroxine who maintained TSH levels of 0.5-5 mU/L. Depression and hypothyroidism share similar clinical features including fatigue, mental sluggishness, forgetfulness, and emotional liability. Severity of depressive symptoms correlated with TSH levels. Researchers reiterate that 95% of a healthy population has a TSH of less than 2.5 mU/L with mean normal TSH levels of 1.18-1.4 mU/L.[86]

As revealed in this review, an abundance of research suggests that the standard upper cutoff for TSH should be changed to 2.5 mU/L. However, research also demonstrates that an optimal range for TSH of 1-2 mU/L would be a better screening tool for identifying early thyroid dysfunction.[87] [88] [89] [90] [91] [92] [93] [94]

Thyroid Hormone Replacement

Currently, standard allopathic treatment of primary hypothyroidism is replacement therapy with levothyroxine, a synthetic form of T4 introduced in the 1970s. Levothyroxine replaced the use of desiccated thyroid extract from animals that had been used for decades prior. Monotherapy with levothyroxine, which provides T4 only, may not be effective for some individuals and addition of T3 may be warranted, especially if conversion of T4 to T3 is compromised.[95]

Naturopathic and functional clinicians may opt for thyroid extracts as a more natural approach to therapy. Monitoring is warranted as these preparations may contain higher or unstandardized amounts of T3 which may increase risk of atrial fibrillation and thyrotoxicosis in some individuals. Preparations may contain variable amounts of T3, T4, and other iodinated compounds such as diiodotyrosine and monoiodotyrosine, which may facilitate weight loss.[96]

An NIH randomized, double-blind crossover study of 14 hypothyroid subjects revealed that those receiving T3 in place of T4 experienced significant weight loss and improvement of serum lipid parameters despite both treatment groups achieving a TSH between 0.5-1.5 mU/L.[97] [98] However, long-term T3 monotherapy is not routinely recommended.

Overview of Thyroid Hormone Replacement[99]

- ✓ Rule out iodine deficiency as cause of hypothyroidism and resolve if present
- ✓ Levothyroxine (synthetic T4) and natural options Armour® Thyroid and Nature-Throid® (thyroid glandular products containing T4 and T3) are FDA approved thyroid replacement therapies
- ✓ Reevaluate TSH and free T4 every 4 to 8 weeks until stable, then 6 month and finally 12 month evaluations would be appropriate.
- ✓ Monitor for angina and atrial fibrillation in cardiac patients
- ✓ Adrenal insufficiency can be exacerbated with thyroid replacement therapy.

Goals of Thyroid Hormone Replacement[100]

- ✓ Normalization of thyroid hormone parameters and resolution of symptoms
- ✓ Improvement of associated cardiovascular risk factors including dyslipidemia, hypertension, endothelial dysfunction, and insulin resistance.
- ✓ Moderation of therapy to avoid thyrotoxicosis and atrial fibrillation
- ✓ If desiccated animal thyroid preparations are used, purity and potency of T3 content should be ensured.

Optimal Takeaways

Based on current knowledge and clinical research, it is clear that current standard lab ranges for TSH may fail to identify individuals with underlying metabolic and thyroid dysfunction, including subclinical thyroid disease. Evaluating TSH within a narrower optimal range will help identify and clinically support these individuals and, ideally, return metabolic and thyroid function to an optimal level. The following takeaways will assist the clinician in addressing thyroid health:

- ✓ Thyroid dysfunction manifests as hyperthyroidism (excess production of thyroid hormone) or hypothyroidism (insufficient production of thyroid hormone), with hypothyroidism being the more prevalent disorder.
- ✓ Impairment of thyroid function will have profound effects on physiology due to its involvement in energy metabolism, vascular integrity, and cardiometabolic health.
- ✓ Symptoms of thyroid dysfunction can mimic other disorders so biochemical confirmation is warranted.
- ✓ Thyroid stimulating hormone (TSH) is the best initial screening tool for identifying thyroid dysfunction. Additional testing will be warranted to further explore the nature of dysfunction.
- ✓ Each individual's history, symptomatology, and laboratory profile must be taken into account when determining the best target range and therapeutic goals for that person.
- ✓ Significant research indicates that levels of TSH above 2.5 mU/L suggest that thyroid dysfunction is present and progressing.
- ✓ An optimal range for TSH of 1-2 mU/L will identify individuals who may be trending toward dysfunction. Individuals outside of this optimal range should be assessed further and monitored for thyroid and metabolic changes.

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