RHETT WOMEN'S CENTER

INFORMED CONSENT FOR THYROID HORMONE SUPPLEMENTATION THERAPY

This form is an "Informed Consent Form." Its purpose is to inform you about the thyroid hormone replacement therapy that your provider(s) (physician/nurse practitioner/physician's assistant) has/have recommended for you. You should read this form carefully and ask any questions before you decide whether or not to give your consent for this therapy.

As with all treatments, there are potential risks and benefits of both treatment and from forgoing treatment. Treatment carries the potential risk of unsuccessful results, complications and injury from both known and unforeseen causes. There is no warranty or guarantee made as to a result or cure. You have the right to be informed of such risks as well as the nature of the treatment, the expected benefits or effects of such therapy, the available alternative methods of treatment and their risks and benefits, and the controversies regarding the most appropriate diagnosis and treatment of low or suboptimal thyroid hormone levels.

The Principals of Medical Ethics adopted by the American Medical Association in 1980 states that a physician shall continue to study, apply, and advance scientific knowledge, make relevant information available to patients, colleagues, and the public. An essential component of informed consent requires that in the absence of medical certainty, patients have the opportunity to choose among medically indicated treatments. The American Medical Association's code of ethics states, "The principle of patient autonomy requires that competent patients have the opportunity to choose among medically indicated treatments and to refuse any unwanted treatments." Because choice can only be preserved by understanding and acknowledging divergent viewpoints on treatment options and providing those treatment options, this document, along with the discussion with your physician, is designed to provide you with such information.

Background: You have been diagnosed with a relative or absolute deficiency of thyroid hormone and may potentially benefit from thyroid hormonal supplementation. Your doctor has recommended treatment with oral thyroid hormone replacement therapy(ies). The goal is to provide you with the most up-to-date therapy options. You need to be sure you understand the reason that this therapy is being prescribed, the potential risks of therapy, and the potential risk of no treatment.

We also feel it is important that you know there are significant controversies regarding the best method to diagnosis low thyroid levels and the best methods of treatment. There is also controversy surrounding the most appropriate way to decide proper therapy and dosage, as well as the best way to monitor therapy. This is especially true when "standard" blood tests look "normal". You may consult another doctor who does not agree with the therapy that you have been prescribed. This document provides extensive information that will be summarized by your provider so that you understand the basis for the diagnosis and the treatment method. It is also vital that you understand the potential risks and benefits of treatment, as well as risks of not treating.

Do not undergo therapy until you have reviewed this document with your provider, and thoroughly understand the potential risks and benefits of treatment and have all your questions answered. The diagnosis and treatment used may be considered non-conventional, complementary or alternative and other health care providers may disagree with the need for treatment, the method of treatment (including medication dosing), and/or the methods of monitoring therapy. You agree to undergo testing as recommended by your provider and report any potential side-effects immediately.

The article entitled Controversies in the Diagnosis and Treatment of Hypothyroidism written by Dr. Holtorf, attached to this document, outlines the controversies involving the diagnosis and treatment of low thyroid. It is designed to inform you about the controversies and to ensure you are able to make an informed decision regarding treatment of your thyroid.

This disclosure is not meant to scare or alarm you. It is simply an effort to make you better informed so that you may give or withhold your consent to the procedure with treatment.

Therapeutic Basis: Based on clinical criteria and symptoms, serologic analysis and/or metabolic/physical testing, patients may demonstrate the presence of low or suboptimal thyroid hormone levels and may benefit from therapy with thyroid replacement/supplementation/optimization. Thyroid hormone replacement therapy can be used to augment thyroid hormone levels in a number of conditions where diminished levels of free T3 and or T4 are shown to be suboptimal. Thyroid hormone replacement therapy is shown to be beneficial for a thyroid deficiency caused by a relative reduction in

the secretion of thyroid hormones from the thyroid gland (either due to primary thyroid illness or from hypothalamic/pituitary dysfunction) and from low tissue or cellular levels caused by dysfunctions in the local control of thyroid activation and transport at the cellular level. Thyroid hormone works at a cellular level to stimulate diverse metabolic activities in most tissues, leading to an increase in energy and basal metabolic rate. Thyroid hormone is necessary for the proper functioning of other glands and organs. Cellular levels cannot be tested directly so estimates are based on serologic, clinical criteria (systems) as well as metabolic and physical testing.

Thyroid hormones may be used alone, or in conjunction with one another, based upon the patient's individualized needs. After review of your serologic analysis, clinical history, metabolic and physical testing, presentation and reported symptoms, your provider is recommending thyroid replacement. This can be T4, T3 or a combination of the two.

Objectives: The goal of thyroid hormone replacement therapy is to optimize hormone levels and to reduce symptoms associated with low cellular levels of these hormones.

Potential Risks: Adverse side effects of any thyroid hormone replacement can include rapid heartbeat, irregular heartbeat, chest pain or tightness, shortness of breath, nervousness, irritability, sleeplessness, tremors, excessive sweating, heat intolerance, weight loss, hair loss, or changes in menstrual periods. Like exercise, which is healthy but can trigger a heart attack or death in someone with underlying heart disease, thyroid replacement is also usually heart healthy but can unmask a heart attack or abnormal rhythm.

If you have a history of heart palpitations or have ever been diagnosed with a heart/cardiac condition, notify your provider before beginning or increasing the dose of any thyroid replacement therapy. Stop taking your thyroid replacement if any symptoms occur and call our office at 843-375-2210. If you are currently taking any thyroid hormone prescribed by another physician, discuss this medication with your provider at Rhett Women's Center prior to initiating any additional thyroid replacement.

Studies show that thyroid hormone replacement is not likely to cause osteoporosis when appropriately monitored, but if the thyroid dose is too high for an extended period of time, it could worsen bone loss. Testing can be done to monitor the amount of bone breakdown as well as undergoing periodic DEXA scans to monitor bone mineral density.

Optimal thyroid levels during pregnancy are essential. Although there is no conclusive data showing that straight T3 is harmful during pregnancy, there is also little data on the safety of straight T3 during pregnancy. Notify your provider if you are pregnant, suspect that you have become pregnant, or if you are planning to become pregnant during this therapy.

Potential Risks of Not Treating may include, but not limited to: Low levels of thyroid can cause, contribute to or be associated with fatigue, depression, heart disease, high cholesterol, chronic fatigue syndrome, fibromyalgia, weight gain, irritable bowel syndrome, cold intolerance, body aches, thinning hair or hair loss, dry skin, heavy periods, premenstrual syndrome, cold extremities, water retention, constipation, muscle cramps, stiff or painful joints, hoarse voice, poor immunity and diminished sweating.

As with other therapies, the response to thyroid hormone replacement/supplementation can vary significantly, you agree to discuss any change in your therapy with your prescribing provider.

By signing this informed consent document, you agree that:
You have been given an opportunity to ask questions about your condition, about conventional "standard" methods of diagnosis and treatment, about integrative, alternative and complementary forms of diagnosis and treatment, about the risks of treatment and the risks of non-treatment, and the risks and hazards involved, and believe that you have sufficient information to give this informed consent.

You certify that this form has been fully explained to you, that you have read it or have had it read to or explained to you and that you understand its contents.

You agree not to undergo any treatments unless you fully understand the treatment and have discussed possible risks and benefits and call or come into the office to ask any questions about the controversies, risks and benefits of treatment (and not treating) and not continue treatment until all your questions are answered.

Reference Article:

Article originally published on Medical Law Perspectives

While the standard of care for most thyroid diseases has little controversy and is supported by a consistent consensus among practitioners, there is significant controversy regarding the essentially two standards of care for the diagnosis and treatment of hypothyroidism. The old standard of care for the diagnosis of hypothyroidism was based on a simplistic model that requires a patient to have a high TSH in order to be diagnosed with hypothyroidism, while a normal TSH indicates euthyroidism (normal tissue thyroid levels) and a suppressed TSH indicates hyperthyroidism (too much thyroid hormone). The problem is that over the last several years, hundreds of studies have demonstrated that this old standard missed the presence of hypothyroidism in the majority of patients, especially those who suffer from chronic illness such as depression, diabetes, obesity, chronic fatigue syndrome, fibromyalgia, or any other condition associated with chronic inflammation as well those with a significant exposure to plastics, pesticides, or other toxins, which potentially includes the majority of the population.

The majority of physicians believe that there must be an elevated TSH in order to make the diagnosis of hypothyroidism. If a patient meets this narrow definition of hypothyroidism, the old standard method of treatment involved treatment with inactive thyroid hormone (T4), which is generally titrated up until the TSH is normalized. The patient is then felt to be euthyroid (have normal thyroid levels). This method, which is practiced by the majority of physicians, is criticized as suboptimal, at best. Additionally, this simple treatment strategy is not followed by a very large (and growing) respectable minority of physicians. Because a significant percentage of physicians are not following the standard guidelines used by the majority of physicians, this respectable minority should not be considered to be practicing outside the standard of care for deviating from the standard diagnostic and treatment guidelines for hypothyroidism. Many alternative methods of diagnosis and treatment of hypothyroidism are evidence-based and backed by numerous published and peer-reviewed studies and taught at numerous CME approved major medical conferences.

While a review of the latest (2014) American Thyroid Association (ATA) Guidelines for the Treatment of Hypothyroidism is beyond the scope of this comment, the large respectable minority of physicians believe that the guidelines are based on a number of flawed premises. One is that the TSH level is an accurate measure of the thyroid activity in the cells of the body. The TSH level is an accurate marker for the T3 level in the pituitary, but hundreds of studies demonstrate that this age-old dogma is only accurate for a theoretical completely healthy patient with no chronic illness, including obesity, depression, stress, and the conditions listed above. Hundreds of studies demonstrate that with any physiologic or emotional stress, the T3 level in the pituitary increases (reducing TSH levels) while the T3 levels in the cells of the rest of the body are decreased. This results in secondary/tertiary hypothyroidism (hypothalamic/pituitary dysfunction), where a low TSH is associated with low peripheral thyroid cellular concentrations if any of the above conditions exist. The ATA Task Force (authors of the latest guidelines) admittedly state that the guidelines do not pertain to those with secondary/tertiary hypothyroidism, but do not state that the majority of the population have some degree of secondary/tertiary hypothyroidism. See: Holtorf, K. Peripheral Thyroid Hormone Conversion and Its Impact on TSH and Metabolic Activity. J Restor Med 2014;23:30-52. Holtorf, K. Thyroid Hormone Transport into Cellular Tissue J Restor Med 2014;3(1):53-68. Schwartz E, Holtorf K. Hormone replacement therapy in the geriatric patient: Current state of the evidence and questions for the future: Estrogen, progesterone, testosterone, growth hormone and thyroid hormone augmentation in the geriatric clinical practice: Part 1. Clinics in Geriatric Medicine 2011;27:541-559. Schwartz E, Morelli V, Holtorf K. Hormone replacement therapy in the geriatric patient: Current state of the evidence and questions for the future: Estrogen, progesterone, testosterone, growth hormone and thyroid hormone augmentation in the geriatric clinical practice: Part 2. Clinics in Geriatric Medicine 2011;27:561-575.

The ATA Task Force's literature review may have missed pertinent studies. For instance, studies that showed dramatic results with straight T3 (rather than the recommended standard treatment with T4) appear to have not been considered. The ATA Task Force used a team of translational scientists to translate basic science into clinical relevance, but they may not have considered the local control of thyroid activity, conversion of T4 to T3 (deiodinase activity), thyroid transport, and the many studies that show why it is impossible to get normal levels of T3 in the tissues if only T4 replacement is used, all of which forms the basis of the alternative treatment regimens used by a large respectable minority of physicians.

The ATA Task Force discusses the fact that much is being learned about how genetic defects in deiodinases (T4 to T3 conversion) may lead to poor patient satisfaction with T4 replacement. However, the task force did not discuss the dramatic effect that occurs on deiodinases (in addition to genetic effects) with a wide-range of chronic illnesses, all resulting in reduced cellular levels of T3. The conclusions of the task force are probably correct for a theoretical patient that has no illness, is not overweight, is not stressed, has no depression, and has never dieted, which makes the guidelines inaccurate for all those except the healthiest individuals.

A few notes on the ATA Task Force's major recommendations: regarding the recommendation on the use of T4 in those with depression and have a normal TSH, the task force recommended against treatment, stating that due to a paucity of evidence treatment success was not assured. The task force may not have considered numerous studies, including one of the largest studies ever done on the treatment of depression, which included the use of T3 in those with a normal TSH. With over 4000 patients, The Star*D Report is the largest trial comparing antidepressant effectiveness for depression. It found that 66% of patients fail to respond to antidepressants or have side-effects severe enough to discontinue use. Of those who do respond, over half will relapse within one year. The trial found that T3 was effective even when other medications-such as citalopram (Celexa), bupropion (Wellbutrin), sertraline (Zolft), venlafaxine (Effexor), or cognitive therapy-were not. Thyroid replacement with T3 was shown to be 50% more effective, even with the less than optimal dose of 50 mcg, under direct comparison with significantly less side effects than commonly used therapeutic approaches with antidepressants. See: National Institutes of Mental Health, Questions and Answers about the NIMH Sequenced Treatment Alternatives to Relieve Depression (STAR*D) Study-All Medication Levels (November, 2006).

The task force did not mention that in the International Journal of Neruopsychopharmacology, published a double blind placebo control trial of 50 patients with normal thyroid function as defined by a normal TSH (1.5 +/- 0.8). The patients were randomized to receive 25 mcg of T3 or placebo in addition to antidepressant therapy. The study found almost a two-fold increase in response rate with T3 and a 4.5 times greater likelihood of experiencing a positive response at any point over a six-week period with the addition of T3. Side effects were higher in the placebo group on 10 out of 11 criteria including a significant increase in nervousness with the placebo group. See: Posternak M et al, A pilot effectiveness study: placebo-controlled trial of adjunctive L-triiodothyronine (T3) used to accelerate and potentiate the antidepressant response, International Journal of Neruopsychopharmacology (February, 2008).

The task force did not mention a study by Tammas Kelly and Daniel Lieberman that investigated the effectiveness of T3 for the treatment of biopolar disorder in 160 patients that had failed to respond to an average of 14 medications used to treat their bipolar disorder. T3 was found to be well tolerated and 84% experienced significant improvement and 33% had a full remission. Again, this is in patients who had not previously responded to numerous medications. One patient who was switched to standard T4 therapy for cost reasons experienced a return of symptoms, which resolved with the reintroduction of T3. See: Tammas Kelly and Daniel Z. Lieberman, The use of triiodothyronine as an augmentation agent in treatment-resistant bipolar II and bipolar disorder NOS, Journal of Affective Disorders 116 (2009) 222–226.

The ATA guidelines state that a suppressed TSH indicates overtreatment and there is significant and dramatic risk if the TSH is suppressed when on thyroid replacement, including atrial fibrillation (a-fib) and osteoporosis. These risks are claimed to be grossly overstated and disputed by a large respectable minority of physicians who state that such risks are not supported by the medical literature. For instance, the perceived risk of a-fib with a suppressed TSH is essentially based on one commonly cited study by Sawin. See: Sawin CT, Geller A, Wolf P, et al. Low serum thyrotropin concentrations as a risk factor for atrial fibrillation in older persons. NEJM 1994;331(19):1249-52. The ATA guidelines state, “For example, in one study, patients > age 65 with serum TSH levels <0.1 mIU/L, the majority of whom were taking L-T4, had a three-fold increase in the risk of atrial fibrillation over a 10 year observation period compared to euthyroid controls.” See: Jonklaas J, Bianco AC Bauer AJ, et al. Guidelines for the treatment of hypothyroidism: Prepared by the American Thyroid Association Task Force on Thyroid Hormone Replacement. Thyroid 2014;24(12):1670-751. This statement is criticized as being incorrect and grossly misleading. As an initial criticism, only 5.7% of study individuals were on thyroid replacement, not the majority, as stated. Of the 115 patients on thyroid replacement (compared to 1892 not on thyroid replacement), 36 (31%) had a suppressed TSH. There were, however, no cases of atrial fibrillation in these patients. This data thus indicates that there is no increased risk of a-fib among people who have a suppressed TSH on thyroid hormone replacement and actually shows protection against a-fib if the TSH is suppressed with those on thyroid hormone replacement. The findings support the known increase risk of a-fib in those with chronic illness, including cardiovascular disease and congestive heart failure, due to secondary and tertiary hypothyroidism.

The task force also did not mention studies that demonstrate that hypothyroidism significantly increases the risk of a-fib and that the use of T3 in those with low serum T3 reduces the risk of a-fib. See: Klemperer JD, Klein IL, Ojamaa K, et al. Triiodothyronine therapy lowers the incidence of atrial fibrillation after cardiac operations. Ann Thorac Surg 1996;61:1323-9. The same can be said of the risk of osteoporosis in those on thyroid hormone replacement with a suppressed TSH. The ATA guidelines do not cite the largest and most rigorous studies and meta-analyses that demonstrate an extremely low risk of osteoporosis with even TSH suppressive doses of thyroid replacement. In fact, the risk of osteoporosis is much higher with the simple use of antidepressants when compared to those with suppression of TSH with thyroid replacement.

In summary, the diagnosis and treatment of hypothyroidism has at least two major standards of care. Because of this, the large but minority group of physicians who do not follow the standard guidelines should not be considered to be practicing outside the standard of care.