

Chapter 7

Safely Getting Well with Thyroid Hormone

ONE OF THE WORST DISASTERS in the history of medicine is the endocrinology specialty's modern guidelines for diagnosing and treating patients whose bodies are under-regulated by thyroid hormone. These guidelines and the beliefs they're based on have caused a worldwide public health crisis. It involves the chronic illness of scores of millions of people and the premature deaths of incalculable numbers more. Each year, billions of dollars are spent for drugs intended to control patients' chronic symptoms; the drugs are largely ineffective and often induce adverse effects in the patients. Researchers who fail to recognize that the cause of the patients' symptoms is under-regulation by thyroid hormone do studies looking for other causes. Since they are blinded to the real culprit, their fruitless efforts squander billions of research dollars.

We learned of this public health disaster through our study of fibromyalgia patients. Our research taught us the main underlying cause of most of the patients' fibromyalgia symptoms: under-regulation of their bodies by thyroid hormone. After learning this, we developed metabolic rehab, a treatment method that helps patients recover normal metabolism. The treatment involves the use of thyroid hormone, but in ways that differ from the method the endocrinology specialty has imposed on most doctors and patients.

Most patients who undergo conventional thyroid hormone therapy remain ill. In stark contrast, some 85% of patients who undergo metabolic rehab fully and lastingly recover. In this chapter, we explain how patients use thyroid hormone in metabolic rehab.

MOST HYPOTHYROID PATIENTS DISSATISFIED WITH T₄-REPLACEMENT THERAPY

T₄-replacement therapy is defined as the treatment of hypothyroid patients with the thyroid hormone T₄, adjusting their doses

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according to their TSH levels.^{[85][89][101][212]} Some advocates of T₄-replacement therapy argue that most patients are satisfied with the treatment. Among those expressing this view is endocrinologist Anthony Toft. In 1999, he wrote of the use of T₄ alone, “It should not be forgotten that the majority of patients taking a dose of thyroxine that satisfies the recommendations of the American Thyroid Association have no complaints about their medication.”^[391]

Not surprisingly, Toft provides no scientific evidence to back up his claim. The reason is that no scientific evidence supports his view. On the contrary, substantial evidence shows that enormous numbers of hypothyroid patients are disappointed with their T₄-replacement therapy.

We receive thousands of requests for information through our website question-and-answer service at <www.drLOWE.com>. One of the most common questions we receive comes from hypothyroid patients being treated with T₄-replacement therapy. Why, they ask, do I still suffer terribly from hypothyroid symptoms? Our answer is that T₄-replacement is the least effective approach to thyroid hormone therapy.^{[210][211]} Over the past fifteen years, we have carefully evaluated the relative effectiveness of different types of thyroid hormone. Our evaluations have led us to a firm conclusion about T₄-replacement: It enables some patients to recover from their hypothyroid symptoms. For most, however, it provides modest improvement at best. Many patients don't benefit from it at all no matter how high they raise their dose of T₄.

Toft's opinion is contradicted by the popularity of websites that provide information on alternative approaches to thyroid hormone therapy. During 2002-2003, we've had an average of 22,000 visitors to <www.drLOWE.com>^[396] each month, most of whom are hypothyroid patients dissatisfied with the kind of thyroid hormone therapy Toft advocates. Other popular websites are Mary Shomon's thyroid information site^[394] and Thyroid-UK.^[395] Totaled, hundreds of thousands of dissatisfied patients visit these sites and others, looking for information with which they can recover the health they've lost through T₄-replacement therapy.

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Studies also contradict Toft's opinion. The Thyroid Foundation of America conducted a survey of patients whose thyroid glands had been removed. The majority were dissatisfied with their treatment.^[11,p.153]

Also, in 2003, thyroid patient advocate Mary Shomon conducted a survey of more than 850 patients. Over half were dissatisfied with their thyroid hormone therapy. Results of the survey showed that many thyroid patients still suffer from symptoms even though they're being treated with thyroid hormone. Table 1 gives the percentages of current symptoms reported by the thyroid patients who responded to Mary's survey.^[389]

Symptom	Percentage still suffering from
• Fatigue	92%
• Inability to lose weight despite diet/exercise	65%
• Feel sluggish & lethargic	62%
• Trouble concentrating	60%
• No sex drive	58%
• Pains, aches, stiffness	51%
• Depression	45%
• Hair loss	43%
• Eyes dry & light sensitive	38%
• Strange feeling in neck or throat	38%

Interestingly, Dr. Toft has waffled in his viewpoint. At the 21st Joint Meeting of the British Endocrine Societies, April 2002, he made a concession that many thought they would never hear from him.^[390] He cited studies showing that restoring normal levels of thyroid hormone in the cells of hypothyroid rats is possible only when they're treated with a combination of T₃ and T₄.^{[30][58]} He then noted a study in which the use of both T₃ and T₄ by human patients was superior to that of T₄ alone. The patients' cognitive function

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was better when they used both hormones.^[392]

“It would appear,” he then stated, “that the treatment of hypothyroidism is about to come full circle.” By this, Toft meant that the use of T₃ and T₄ in combination—as was used throughout most of the 20th century—is the superior therapy.

Then, on August 14, 2002, he contradicted his recent public statements. On that date, he wrote a letter replying to thyroid patient advocate Linda Thipthorp in Cornwall, UK. “I would reiterate,” he wrote, “that the overwhelming majority of patients feel perfectly well taking Thyroxine [sic] alone in a dose that restores serum TSH to normal.”^[393] The absurdity of this view is obvious from the percentages in Table 1.

Rather than being satisfied with T₄-replacement, as Toft claims, thousands of hypothyroid patients are highly dissatisfied with their continuing hypothyroid symptoms. Many are outraged at endocrinologists’ arrogant dismissal of their continuing symptoms as features of mysterious “new diseases” such as fibromyalgia, chronic fatigue syndrome, or myalgic encephalomyelitis (ME). And some patients, although relieved when they promptly recover their health with the thyroid hormone therapy we describe in this chapter, are furious that conventional therapy kept them sick for so long.

TWO MAJOR SOURCES OF UNDER-REGULATION BY THYROID HORMONE

Under-regulation by thyroid hormone results from one or both of two disorders: hypothyroidism and partial cellular resistance to thyroid hormone.

Hypothyroidism

Hypothyroidism is abnormally low production of thyroid hormone by the thyroid gland. The hypothyroid patient, then, has too little thyroid hormone to properly regulate the metabolism of her cells. As a result, the metabolism of her cells is abnormally slow. If severe enough, she’ll have symptoms of hypometabolism.

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A patient may be hypothyroid because her thyroid gland is diseased. The most common disease of the gland that causes hypothyroidism is autoimmune thyroiditis. In this disease, antibodies attack and destroy the follicles of the gland. Thyroid hormone is manufactured in the follicles, and when they are destroyed, the gland isn't capable of producing enough thyroid hormone.

A patient may also be hypothyroid because something is wrong with either the hypothalamus or the pituitary gland in her brain. Each of these structures produces a hormone that helps regulate the thyroid gland. The hypothalamus produces TRH, and the pituitary produces TSH (see the *Glossary* for an explanation of these hormones). Too little TRH or TSH can reduce the thyroid gland's production of thyroid hormone, leaving the patient hypometabolic.

Cellular Resistance to Thyroid Hormone

A patient's body may be under-regulated by thyroid hormone because her cells are partly resistant to the hormone. Patients who are hypometabolic due to resistance to thyroid hormone are classified as having "partial peripheral cellular resistance to thyroid hormone." These patients have "normal" laboratory thyroid hormone levels. In other words, they have normal amounts of thyroid hormone in their blood, but these normal amounts are not enough to overcome the resistance in their cells.

Some patients' thyroid hormone resistance is caused by mutations of the thyroid hormone receptor. Many other causes have been proposed, as I (JCL) detailed in *The Metabolic Treatment of Fibromyalgia* (pages 322-329).^[1] The causes are debatable, but what isn't debatable is that among patients with a diagnosis of fibromyalgia, the disorder is common.^{[161][162]} Our clinical experience has also shown that the disorder is far from rare among other hypometabolic patients.

Deciding Whether a Patient is Hypothyroid or Resistant to Thyroid Hormone

Deciding whether a patient is hypothyroid or possibly thyroid

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hormone resistant is important. The decision should determine what type of thyroid hormone the patient begins treatment with.

Deciding if a Patient is Hypothyroid. When we evaluate a patient, we first consider whether she has symptoms typical of hypothyroidism. If she does, but also has a wholesome diet, takes nutritional supplements, exercises, and doesn't take metabolism-impairing drugs, we consider that she may have hypothyroidism or thyroid hormone resistance.

To learn if she is hypothyroid, we order a particular profile of laboratory thyroid tests for new patients. The profile includes tests for TSH, total T_4 , T_3 -uptake, and free- T_4 (thyroxine) index.

Until recently, if a patient's thyroid profile didn't indicate hypothyroidism, we could order a TRH stimulation test. Results of this test helped us to decide if a patient had hypothyroidism due to a problem with her hypothalamus or pituitary gland. If so, we diagnosed her disorder as "central hypothyroidism." Two of our studies indicated that among fibromyalgia patients, 44% had TRH stimulation test results consistent with central hypothyroidism.^{[2][3]} Most of these patients recovered when treated for hypothyroidism, even though their standard thyroid test profiles hadn't indicated hypothyroidism. At this time, however, the TRH stimulation test isn't available.

An alternate profile contains tests for TSH and free T_4 . We prefer the other profile, however, because it permits us to identify problems with the proteins in the blood that carry thyroid hormone. Your doctor may use either profile. However, if he orders only a TSH, we recommend that you protest and insist upon the other component(s) of one of the profiles.

We also order tests of thyroglobulin and thyroid peroxidase (microsomal) antibodies. Some patients with chronic muscle pain and other symptoms of hypometabolism have elevated antibodies, but their other lab thyroid test results are within the reference ranges. Most of these patients respond well to a thyroid hormone product that contains both T_3 and T_4 .

If the patient has a high TSH, low measures for T_3 or T_4 , or

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high antithyroid antibodies, it's safe to tentatively conclude that she is hypothyroid. Technically, the decision that the patient is hypothyroid is always tentative unless the patient has a medical history that suggests that her thyroid gland is damaged in some way. The history might include surgical removal of part of her thyroid gland (thyroidectomy), previous drug therapy to destroy part of her thyroid gland, or high levels of antithyroid antibodies that might have damaged her gland. But even if she doesn't have such a history, she may still have abnormal thyroid test results and symptoms of hypothyroidism. If so, a trial of therapy using a product that contains both T₃ and T₄ is warranted. (For an explanation of why our patients don't use T₄ alone, see section below titled "Use of T₄ is the Least Effective Approach to Thyroid Hormone Therapy.")

When a patient's thyroid hormone levels are normal, but her TSH is high, the assumption is that her thyroid gland is undergoing failure. Even if she doesn't have hypothyroid symptoms, some doctors would start her on thyroid hormone therapy. The purpose of the therapy is to prevent her from developing hypothyroid symptoms. The diagnosis her doctor gives her is "subclinical hypothyroidism." In general, we agree with this preemptive therapeutic approach.

It's common for a patient with hypothyroid-like symptoms to have borderline thyroid test results.^[205] If the patient doesn't have another condition that plausibly explains her symptoms, it's reasonable for her to begin a trial of thyroid hormone therapy.

The question naturally arises: What is a borderline thyroid test result? The answer shows that the diagnosis of hypothyroidism isn't scientifically precise, but is in fact arbitrary.

When I (JCL) began working with hypothyroid patients in the mid-1980s, the endocrinology specialty argued that a TSH level of around 6.0 was the upper end of normal. According to them, it was proper to diagnosis hypothyroidism only if the TSH was higher than that number. So at that time, if a patient of mine had a TSH of 5.7, I would have considered it borderline for the diagnosis of primary hypothyroidism.

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Recently, the American Association of Clinical Endocrinologists^[16] (AACE) and the National Academy of Clinical Biochemistry^[342] decided that the range of TSH values indicating normal thyroid function is narrower than they formerly believed. The AACE has decided that 0.3-to-3.0 is the new reference range. According to this new range, if a patient's TSH level is above the upper limit, the doctor should diagnose hypothyroidism. As a result, when a patient's TSH is perhaps 2.8 or so, I (GH-L) might consider it borderline. Today, both of us would consider a TSH of 5.7 clearly high, not borderline.

We want to emphasize that decisions based on the results of lab thyroid test results are fallible.^{[1,pp.794-798,824-826][404][405]} The tests aren't especially reliable: test results on one day may be markedly different from those on the next day or the following week.^[404] And, as we just noted, those who continue to attempt to establish reference ranges (what used to be called "ranges of normal") periodically change the ranges. What conventional medicine considers a "normal" test result today, it may consider abnormal tomorrow.

Despite their faults, lab thyroid tests are a useful tool for classifying patients. And classifying patients provides some basis for choosing the type of thyroid hormone they begin treatment with.

When hypothyroid patients use T₄-containing products such as desiccated thyroid, they can expect the therapeutic effects from a newly increased dose from one-to-two weeks later. It's reasonable, then, for them to wait about two weeks before reevaluating the intensity of their symptoms (see *Chapter 3*).

Deciding if a Patient is Resistant to Thyroid Hormone. Resistance to thyroid hormone is a relatively new but well-researched field.^[1,pp.295-338] Researchers first documented a family whose cells are partly resistant to thyroid hormone in 1967.^[221] In 1990, other researchers found one cause of the resistance: a mutation in a gene on chromosome 3.^[220] More than a hundred mutations have now been found in this gene in different patients who are resistant to thyroid hormone.

Mutations in the gene, however, aren't always the cause of

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thyroid hormone resistance. Some of our patients who clearly had resistance and fully recovered by using T_3 (within the context of metabolic rehab) were free from mutations in the gene.^[1,p.277] Resistance may result from several possible mechanisms (for a full description, see Chapter 2.6, *The Metabolic Treatment of Fibromyalgia*, pages 303-313, 319-321, 323-329^[1]). Regardless of the cause of a patient's resistance, our current statistics suggest that 34% of our patients with a diagnosis of fibromyalgia have resistance.^[161]

Unfortunately, we can't determine when we first evaluate a patient whether she has thyroid hormone resistance. We can only suspect that she might. If she has a diagnosis of fibromyalgia, chronic fatigue syndrome, or ME, this means that she has symptoms typical of hypothyroidism or thyroid hormone resistance.

The symptoms, of course, are also typical of other possible conditions. The most common ones are multiple nutritional deficiencies, cortisol deficiency, low physical fitness, or the use of metabolism-slowing drugs. Any of these may cause symptoms remarkably similar to those of thyroid hormone deficiency or resistance.

Commonly, when a patient consults us, she's already gone to great lengths to recover her health; she's adopted a wholesome diet, taken nutritional supplements, exercised to tolerance, and abstained from the use of metabolism-impairing drugs. With this patient, our first aim is to learn whether she has a thyroid hormone deficiency. Often her history and thyroid test results don't suggest a deficiency. In these cases, we next look for lab test results that suggest some other disorder that could account for her symptoms of hypometabolism. If we find none, we then suspect that her symptoms are caused by thyroid hormone resistance.

We wrote above that TRH stimulation tests aren't available right now. Without this test, we can't rule out central hypothyroidism. The only way to determine its likely presence is through a trial of T_4/T_3 therapy. If this treatment doesn't relieve the patient's symptoms, it's possible that she may have thyroid hormone

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resistance. A trial of T_3 therapy is then warranted.

During the trial, the patient carefully progresses through metabolic rehab using plain T_3 . The method of adjusting her dose of T_3 , and the safety monitoring she undergoes, are based on our experiences with hundreds of resistance patients and our scientific studies of those patients.^{[36][38][92][93][94][135][137][188][189][292][403]}

We want to emphasize that our patients use plain T_3 —*not sustained-release or timed-release T_3* . They take their full dose of T_3 on an empty stomach (one hour before a meal, or three hours after) once each day (see Figure 1).

When thyroid hormone resistance patients use T_3 , therapeutic effects from a newly increased dose may occur within several days to a week. It's reasonable for these patients to reevaluate the intensity of their symptoms a week after they increase their T_3 dose (see *Chapter 3*).

We can't give a specific starting dose for patients in general. It varies for different patients, depending on their health status, severity of their symptoms, and other factors. Many patients, however, start with what the clinician calculates to be a full replacement dose—one that provides all the thyroid hormone a normal thyroid gland would produce to regulate metabolism. The patient's doctor should help her calculate an appropriate starting dose.

From the starting dose, the patient gradually increases the thyroid hormone at intervals that may vary from several days to a week or two. She increases her dose based on her doctor's calculations; the amount of each increase is most often small. Gradually, through these increases, she reaches what we call her "therapeutic window"—the dose at which she has no symptoms of overstimulation, and her hypothyroid-like symptoms improve or disappear altogether. In most cases, her therapeutic window involves doses of T_3 that would overstimulate people whose cells respond normally to thyroid hormone.

Let us say emphatically, however, that this doesn't mean that all patients who think they are resistant should take high doses of T_3 ! Treatment with thyroid hormone must always be individualized.

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Bear in mind that many patients who have failed to benefit from T_4 alone or combined T_3 and T_4 , recover from their symptoms on *fairly low doses* of T_3 . Patients and the doctors treating them with T_3 shouldn't forget this. High enough doses of T_3 can be harmful to anyone, and it's crucial that patients not subject themselves to overstimulation from excessive doses.

Used responsibly, however, T_3 has no adverse effects.^[12] In fact, it's far safer than any of the drugs doctors commonly prescribe to control the symptoms from untreated or under-treated hypothyroidism.

If a patient reaches her therapeutic window, and she meets the following four criteria, we conclude that she has partial cellular resistance to thyroid hormone:

- Before beginning to use T_3 , her lab thyroid test results suggested that she didn't have hypothyroidism.
- Her hypothyroid-like symptoms are improved or relieved by her use of a dose of T_3 that would overstimulate most other people.
- After markedly improving or recovering from her symptoms with the use of T_3 , her free T_3 blood level is high.
- *She has no evidence of tissue overstimulation*: she has no symptoms of overstimulation, and repeatedly, her results are normal on EKGs, serum and urine lab tests, and bone densitometry.

We want to emphasize that patients undergoing treatment for possible thyroid hormone resistance must be under the care of a doctor. Not just any doctor, however. The doctor must be educated about thyroid hormone resistance and experienced in guiding patients through proper treatment for the disorder.

We believe it's important to emphasize this for several reasons. First, patients can now obtain different thyroid hormone products, including plain T_3 , without a prescription over the Internet. We feel this is a boon to patients. It means they can buy T_3 for a *far* lower

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price than buying it in most pharmacies in the United States. Also, being able to buy T_3 without a prescription essentially removes the shackles of T_4 -replacement therapy that conventional endocrinology has strapped on patients the world over who need thyroid hormone therapy.

But this new freedom to obtain T_3 without a prescription imposes a responsibility on all those who take advantage of the freedom. To maintain it, patients must not give the fascist factions within conventional medicine justification for enlisting the help of legislators and regulatory agencies to take away the freedom. It matters not that conventional medicine, through T_4 -replacement therapy, has now ruined the lives of scores of millions of people, and brought the lives of others to a premature end. It would only take a patient or two being harmed by self-administered T_3 , and conventional medicine would quickly move to deprive all patients of this freedom.

What we're saying here is that safety must be the foremost concern—not only for doctors who treat patients with T_3 , but for the patients themselves. So, if you suspect that you have thyroid hormone resistance, enlist the guidance of a collaborative doctor educated about the condition and experienced in its treatment.

■ For the most extensive document ever written on thyroid hormone resistance, please see *The Metabolic Treatment of Fibromyalgia*, Chapter 2.6, pages 295-338.^[1]

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Educational Information on Thyroid Hormone Therapy

The information we provide in this section on thyroid hormone therapy is strictly for educational purposes. We believe it's important to help educate patients about what safe and effective thyroid hormone therapy is and isn't.

It's important that patients be well-educated about thyroid hormone therapy. This is so because, in general, doctors don't know how to safely and effectively treat patients with the hormone. If most patients are to get safe and effective treatment, they must be well-informed about what is proper thyroid hormone therapy.

With accurate knowledge, for example, a patient can judge the competence of the doctor whose care she's presently under. If necessary, she should try to educate him. Or, if he isn't open to learning, she can dismiss him and find another doctor she judges to be competent to help her. This is the exemplary assertive approach of Vicky Massey, LMP, who wrote the *Foreword* to this book.

OPTIMAL IMPROVEMENT WITH THYROID HORMONE THERAPY

To get optimal improvement with thyroid hormone, the patient should follow two rules. First, she should *not* permit her doctor to adjust her dosage according to lab thyroid test results. TSH and thyroid hormone levels have nothing whatever to do with making correct decisions about safe and effective doses of thyroid hormone.^{[345][406]} Using the results of thyroid tests to adjust a patient's dose is likely to sabotage the patient's effort to recover from her symptoms of hypothyroidism or thyroid hormone resistance.

Second, the patient should enlist her doctor's assistance in adjusting the hormone dose according to changes in her symptoms due to the last dosage increase. The patient should monitor for changes in her symptoms using the method we describe below and in *Chapter 3*. The patient's doctor can help her by monitoring how she's responding in other ways (see section below titled "Tissue Responses to Thyroid Hormone that Your Therapist or Doctor Can Monitor").

Other Responses to Treatment

the Patient Can Evaluate

Three useful measures of patients' tissue responses to thyroid hormone are the pulse rate, blood pressure, and basal temperature. It's useful to take these measures at least a couple of times each week and record them. Reviewing the list of measures can be a helpful gauge of treatment progress. Graphing the measures is even more useful.

Pulse Rate and Blood Pressure. Many patients with poor metabolic health have low blood pressure and a slow pulse rate. This is especially true of those with a diagnosis of fibromyalgia or chronic fatigue syndrome.

If a patient has low blood pressure and a slow pulse rate, effective thyroid hormone therapy will most likely increase both. Some patients, however, must make lifestyle changes to further increase their blood pressure. For example, a patient who has avoided table salt for health reasons may have to begin ingesting some to help raise her blood pressure.

Unfortunately, your doctor's office probably isn't the best place for your blood pressure or pulse rate to be measured. A rise in blood pressure in a doctor's office is so common that it's called "white coat hypertension." Patients' pulse rates are also usually higher in their doctors' offices.

For the patient to get her true resting blood pressure and pulse rate, she'll most likely have to purchase a device that measures both. These devices are widely available in drug stores and athletic stores. They can be useful not only for taking one's blood pressure and pulse rate, but to help distinguish thyroid hormone overstimulation from other causes of increased blood pressure and pulse rate.

To get a true measure of resting blood pressure and pulse rate, sit in a quiet and comfortable room. With your eyes closed, think of something pleasant for a minute or two. Then, when you feel deeply relaxed, take your blood pressure and pulse rate. If possible, have someone else take the measures while you remain relaxed with your eyes closed.

Basal Body Temperature. The basal temperature is the body

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temperature at rest. Body heat depends on the amount of foodstuff the body oxidizes or burns. Thyroid hormone is the main agent that regulates the rate at which foodstuff is burned. In patients with hypothyroidism and thyroid hormone resistance, the rate is too low. As a result, most have a body temperature lower than normal.

During World War II, Dr. Broda Barnes and Dr. Joseph Ehrlich studied the basal body temperatures of a thousand soldiers. Over the next thirty years, Dr. Barnes studied the temperatures of many thousands of other patients. He established that the normal armpit temperature is between 97.8° and 98.2° Fahrenheit. Most patients with hyperthyroidism or infections had armpit temperatures above 98.2°, and most hypothyroid patients had temperatures below 97.8°. As thyroid hormone normalized the metabolic health of hypothyroid patients, their basal body temperatures increased into the range of 97.8°-to-98.2°. ^[185,pp.42-47]

Men can take their basal body temperature on any day. Women, on the other hand, have temperature variations during their menstrual years. The typical woman's temperature is lowest at the time of ovulation, and it's highest just before the start of her menstrual flow. Dr. Barnes recommended that the woman take her basal temperature on the second and third days of the period after her flow starts.

The patient should shake down a thermometer before she goes to bed and place it on a night stand. When she wakes in the morning, she should place the thermometer deep into her armpit and leave it there for ten minutes by the clock, while remaining still the entire 10 minutes.

Dr. Barnes noted that young children may have trouble lying still for ten minutes. It may be best to take their rectal temperature

Figure 1. How our patients take thyroid hormone

Our patients take their thyroid hormone in a single daily dose when their stomach is empty. This means that they take their thyroid hormone—no matter what form of thyroid hormone they take—at least one hour before meals or taking nutritional supplements, or three hours after.

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for two minutes. The normal range is 98.8°-to-99.2° Fahrenheit.^[185,p.48]

As with the blood pressure and pulse rate, the patient should record her temperature readings and the dates she took them. The readings are most useful when posted to a line graph. (For extensive information on body temperature as a tissue response to thyroid hormone, see *The Metabolic Treatment of Fibromyalgia*, Chapter 4.3, pages 842-849.^[11])

Body Temperature May Not Change. Effective thyroid hormone therapy doesn't always increase some patients' low body temperatures, and not all patients with hypothyroidism or thyroid hormone resistance have low body temperatures. For these types of patients, the basal body temperature isn't a good gauge for monitoring change with thyroid hormone therapy.

Dr. Barnes warned, "The basal temperature is not a perfect test for thyroid function. There are conditions other than hypothyroidism that may produce a low reading—for example, starvation, pituitary gland deficiency, or adrenal gland deficiency. But starvation is certainly not difficult to rule out—and some thyroid is frequently indicated, anyhow, for the other conditions."^[185,p.46]

Tissue Responses to Thyroid Hormone That Your Therapist or Doctor Can Monitor

Health care practitioners can help the patient monitor for tissue changes in response to thyroid hormone therapy. Most useful are changes in the puffiness of skin and muscle and the speed of the Achilles reflex.

Puffiness of Skin and Connective Tissue in Muscles. When a patient is going through metabolic rehab, a practitioner who examines and physically treats her at intervals can look for changes in her skin and muscles. A massage therapist, certified myofascial trigger point therapist, naprapath, or chiropractic physician who works on the patient's muscles can be especially helpful.

Some patients whose hypometabolism is caused by hypothyroidism or thyroid hormone resistance have puffy skin and puffy

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swelling of the connective tissue around muscles. We've observed this for many years in hypometabolic patients, especially those with a diagnosis of fibromyalgia. Substantial evidence shows that the puffiness results from too little thyroid hormone regulation of connective tissue cells.^[1,pp.425-446] Starlanyl and Jeffrey reported the swellings in fibromyalgia patients,^{[18][22]} and in a study of one patient, they found that treatment with T₃ relieved the swellings.^[18]

An attentive therapist or doctor who treated you before and after you began to use thyroid hormone may find that your puffiness has disappeared. Its disappearance is a valuable sign that your current dose of thyroid hormone is benefitting you, at least in this one respect. If they haven't already, other benefits are likely to soon follow.

Achilles Reflex Speed. The relaxation phase of the Achilles reflex is abnormal in about 80% of patients who are hypothyroid or resistant to thyroid hormone. Accordingly, it's abnormal in roughly the same percentage of patients with a diagnosis of fibromyalgia.^[1,pp.879-880]

The Achilles reflex is abnormal in that the relaxation phase is slower than the contraction phase. When the doctor hits a patient's Achilles tendon at the back of the ankle, the calf muscles contract at a normal rate, and the foot dips down. Normally, the foot comes back up at the same speed at which it dipped. But for most patients with too little thyroid hormone regulation, the calf muscles relax too slowly. This causes the foot to come back at a slower speed. The speed is so much slower that it's obvious to most anyone watching.

An occasional hypometabolic patient has a variation of the slow relaxation phase. After the doctor taps the tendon, her foot dips quickly. But the foot stalls briefly, or may jerk slightly, before beginning a slow or normal relaxation phase.

As patients increase their doses of thyroid hormone into the effective range, the relaxation phase of the Achilles reflex becomes more rapid. Eventually it equals the speed of the contraction phase.

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And any jerks or pauses before or during the relaxation phase cease. (Overstimulation with thyroid hormone speeds both the contraction and relaxation phases of the reflex.)

Before beginning to use thyroid hormone, the patient should ask a doctor or therapist to test her Achilles reflex. If the relaxation phase is slow at this baseline measurement, changes in the speed of the relaxation phase can serve as a measure of the patient's tissue response to thyroid hormone. (For extensive information on the Achilles reflex as a tissue response to thyroid hormone, see *The Metabolic Treatment of Fibromyalgia*, Chapter 4.3, pages 850-852.^[1])

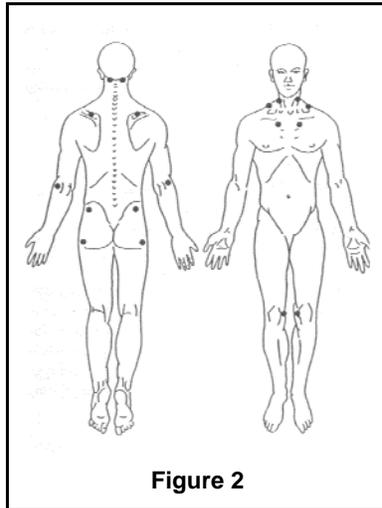


Figure 2

Algometer Exam of Tender Points. If your diagnosis is fibromyalgia, measuring the sensitivity of your tender points can be useful. We examine 18 tender points represented by dots on the body drawing (see Figure 2).

Using a finger or thumb to gauge how sensitive the points are isn't reliable. A doctor or therapist, however, can use a pressure gauge called an "algometer" to measure the sensitivity of the tender points.

After measuring how much

pressure is needed to set off the perception of pain at each tender point, the doctor or therapist should calculate the average amount of pressure that initiated pain at the 18 points. It's most useful to create a line graph for the average tender point sensitivity. Changes in the sensitivity during metabolic rehab are a useful gauge of a patient's tissue responses to thyroid hormone.

Responsiveness of Trigger Points to Treatment. The muscles of many hypometabolic patients contract too easily. This can cause

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trigger points in the muscles to refer pain too readily. Commonly, these patients must be treated physically far more often to keep their trigger points from referring pain.

When the patients undergo metabolic rehab including the use of thyroid hormone, their trigger points respond better to physical treatment. Relief that may have lasted only days may now last for weeks. A practitioner who has treated the patient both before and during her metabolic rehab may note her improved response to physical treatment.

REALISTIC PERSPECTIVE ON THE SAFE USE OF THYROID HORMONE

The potential harm from the use of thyroid hormone has been *grossly* exaggerated by the endocrinology specialty. Despite this, patients and their doctors should exercise caution to avoid thyroid hormone overstimulation. They can best do so by ignoring the endocrinology speciality's exaggerations, and seeing the potential for harm with proper perspective.

Needless Worry Over Potential Harmful Effects of Thyroid Hormone

The three main potential harmful effects from the misuse of thyroid hormone are bone thinning, heart problems, and acute adrenal crises.

Osteoporosis. Through the 1980s and 1990s, doctors forced millions of patients' to keep their thyroid hormone doses too low in order to avoid causing osteoporosis. In the early 1990s, studies began appearing in journals showing that TSH-suppressive doses of thyroid hormone *don't* cause excessive bone thinning, and certainly don't cause osteoporosis or an increased rate of fractures.

For years, I've (JCL) talked with bone density radiologists about the potential for bone thinning from the use of thyroid hormone. Some have chuckled over the absurdity of the endocrinologists' belief that TSH-suppressive doses of thyroid hormone cause significant bone thinning, osteoporosis, or an increase in fractures.

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Bone density tests of our own patients have shown that those taking higher-end thyroid hormone doses usually have higher bone density. The main cause of their higher bone density is that their thyroid hormone doses are high enough to enable them to exercise vigorously enough to thicken their bones. And, of course, their wholesome diets and nutritional supplements also contribute.

It appears that the conventional endocrinologists' scientifically false belief that TSH-suppressive doses of thyroid hormone cause osteoporosis has actually contributed to bone thinning among patients. Those restricted to T₄-replacement therapy usually are too weak and fatigued to exercise vigorously. Their low physical activity level has undoubtedly caused progressive thinning of their bones, as well as deterioration of their health in general.

Table 2. Nutrient complex that favors bone thickening

- Vitamin D
- Calcium (from hydroxyapatite)
- Phosphorus (from hydroxyapatite)
- Magnesium (from magnesium oxide)
- Zinc (as amino acid chelate)
- Copper (as amino acid chelate)
- Manganese (as amino acid chelate)
- Boron (as amino acid chelate)

If a patient already has osteoporosis when she begins using thyroid hormone, caution is prudent. If she hasn't recently had a bone density study, she should ask her doctor to order one. She should ask him to order follow-up studies at intervals, perhaps every six months. She may have to teach the doctor that,

despite the opinion of conventional endocrinologists, we *absolutely cannot* accurately infer from a TSH level the effect of a dose of thyroid hormone on bone density. If the doctor wants to know the effect, he should measure it directly by ordering a bone density study.

When the patient's dose of thyroid hormone is high enough to enable her to exercise, she should do "bone-jerking" types of exercises to tolerance each day. She should also make sure she gets enough nutrient complex and protein each day to help thicken her

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bones. The complex we take includes the nutrients listed in Table 2.

Heart Problems. “You’re going to have a heart attack and die!” is the warning many doctors give their patients when they ask to be treated with desiccated thyroid—and especially if they ask for T₃. (If you haven’t read the *Foreword* to this book by Vicky Massey, LMP, you may find interesting her experience with a doctor who bellowed this warning at her.) Where these doctors get this idea is a mystery, but they certainly don’t get it from the scientific literature.

The belief of these doctors that patients using desiccated thyroid or T₃ run a high risk of heart attacks is clearly ignorance. Still, though, it’s prudent for each patient to take reasonable precautions when using thyroid hormone. We recommend that each patient at least get a baseline EKG before starting to use thyroid hormone. If the patient’s doctor has any doubts about the health of her heart, a consultation with a cardiologist is wise. We’ve referred many patients for cardiac consults before they began taking thyroid hormone. Rarely, a cardiologist has recommended that the patient only cautiously increase her thyroid hormone dose. Never has a cardiologist reported to us that a patient should not use thyroid hormone.

Most patients with heart disease can safely use thyroid hormone. In fact, using the hormone is likely to improve the patients’ heart disease. These patients, however, should use thyroid hormone cautiously. And they—just like every other person who takes thyroid hormone—should have a wholesome diet, take a full array of nutritional supplements, and get regular aerobic exercise to tolerance.

Figure 3. Atrial fibrillation

The atria are the two small upper chambers of the heart. They receive blood from the veins and in turn force it into the large chambers of the heart called the “ventricles.” Fibrillation is uncontrolled twitching or quivering of muscle fibrils. Normally, the muscles of the atria contract rhythmically. In atrial fibrillation, however, the atrial muscles twitch rapidly and irregularly. The irregular twitching of the atria bombards the ventricles, potentially causing them, too, to contract irregularly.

If a patient has compromised heart function, her initial dosage should be low enough to avoid aggravating the heart condition. If her doctor finds that she tolerates the dose well, he should guide her through small dosage increases until she reaches her optimal dose. He must monitor her each step of the way using all appropriate procedures to ensure her safety.

Recently, endocrinologists have warned that TSH-suppressive doses of thyroid hormone increase the risk of atrial fibrillation by 33%. A number of studies showed that a certain set of people who had low TSH levels had a higher incidence of atrial fibrillation (see Figure 3).

But don't conclude from this finding that if you take a dose of thyroid hormone that suppresses your TSH level, you'll have atrial fibrillation. What endocrinologists—the main doctors who warn of this risk—don't bother to tell you is that these studies were done on elderly, sedentary individuals. In fact, in some of the studies, the patients were bedridden in nursing homes. In none of the studies did the researchers control for a heart-protective diet, nutritional supplements, or cardiovascular exercise to tolerance. The patients appear to have been in such poor health that they may have developed atrial fibrillation if they drank too much coffee each day. It's ludicrous and outrageously wrong to conclude that the results of these studies apply to healthier people using TSH-suppressive doses of thyroid hormone.

We require all patients under our care to adopt a wholesome

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diet, take nutritional supplements, and exercise to tolerance. These practices are what we call (as in the previous paragraph) “heart-protective,” meaning that they encourage healthier hearts that are resistant to disease and other problems such as atrial fibrillation. Because we require our patients to engage in heart-protective lifestyle practices, we haven’t had a single patient under our care ever have a heart problem from the use of thyroid hormone—despite the fact that many of them take enough of the hormone to suppress their TSH levels.

Acute Adrenal Crisis. The risk of an acute adrenal crisis—collapse of the patient from too little cortisol—is a risk from the use of thyroid hormone that many endocrinologists unrealistically exaggerate. The fact is that the published medical literature contains only a few reports of acute adrenal crisis. These reports were of patients in extraordinary circumstances.^{[120][121][122][123]}

It is simply irrational to conclude from these few exceptional reports that most patients using thyroid hormone should be concerned about having an acute adrenal crisis. We’ve worked with many patients who developed symptoms of adrenal insufficiency after they began using thyroid hormone. Not a single one of these patients, however, has experienced acute adrenal crisis.

Relieving Thyroid Hormone Overstimulation

Occasionally a patient inadvertently increases her thyroid hormone dose too high. The symptoms of overstimulation she experiences are usually rapid heart rate and tremors. We’ve listed other symptoms of overstimulation in the form titled “Are You Overstimulated?” (See *Forms*.)

Tremors can be detected more easily if the patient will sit with the balls of her feet on the floor, and raise both heels up. If her ankles quiver, this suggests thyroid hormone overstimulation. If the Achilles reflex speed is extremely rapid in both phases, this too indicates overstimulation.

Before concluding that thyroid hormone is causing these results, however, it’s important to rule out other possible causes. The

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patient should make sure she hasn't taken stimulants such as ephedra, antihistamines, decongestants, or too much caffeine. Emotional upset or acute stress can also cause symptoms that resemble those of thyroid hormone overstimulation.

The patient truly overstimulated by thyroid hormone should decrease her daily dose. If the patient's thyroid hormone product is T_3 , reducing her next scheduled dose can stop the stimulation within 24-to-48 hours. If her thyroid hormone product contains T_4 , the overstimulation might not stop for a week or so despite her reduced dose. In this case, she should ask her doctor about using a small dose of propranolol several times each day until the overstimulation stops. In general, results are best when the doctor prescribes 20 mg plain (not sustained-release) propranolol tablets. Propranolol is highly effective at relieving symptoms such as tremors. Many public speakers, trial lawyers, and musicians and other entertainers use propranolol before their performances to prevent symptoms of anxiety or nervousness from interfering with the performances.

For most people, 20-to-40 mg of plain propranolol stops the symptoms of overstimulation within 30-to-45 minutes. Usually, the symptoms don't reappear until several hours have passed. Some patients get relief from the symptoms for 6-to-8 hours.

The 20-to-40 mg dose of propranolol is harmless for most patients except those with asthma. *In general, patients with asthma should not take propranolol or any other beta-blocker.*

In general, it's not necessary to completely stop taking thyroid hormone just because mild overstimulation has occurred. Suddenly stopping the hormone altogether may produce rebound hypometabolism. The rebound symptoms of hypometabolism can be more unpleasant than the overstimulation. Reducing a patient's dose by an amount calculated by her doctor will relieve the overstimulation.

IMPORTANT POINTS TO BEAR IN MIND

**A Low TSH Level Does *Not*
Mean the Patient is Hyperthyroid**

An unfortunate convention in orthodox endocrinology is to define “hyperthyroidism” as a suppressed TSH level. Distinct thyroid hormone excess (as in Graves’ disease) does suppress the TSH level. If the hormone excess is severe enough, it will harm the affected patient.

But to describe a patient taking a TSH-suppressive dose of thyroid hormone as “hyperthyroid” is to imply that the patient will suffer effects similar to those of untreated Graves’ patients. Certainly, if the TSH-suppressive dose of thyroid hormone is too large, it will harm the patient. But many patients’ TSH-suppressive doses merely induce normal metabolism in them and have no harmful effects. Depriving these patients of TSH-suppressive doses of thyroid hormone, however, *is* likely to harm them; they’ll have chronic symptoms of hypothyroidism and may develop advanced cardiovascular disease.

To define hyperthyroidism by a suppressed TSH level is, therefore, misleading. It fails to distinguish between the blood levels of thyroid hormone and TSH, and the effect of thyroid hormone on the metabolism of body tissues. A suppressed TSH and hyperthyroidism are *not* one and the same.

**Use of T₄ Is the Least Effective
Approach to Thyroid Hormone Therapy**

Our patients today don’t use T₄ (thyroxine) alone. As we were developing metabolic rehab, many of our hypothyroid patients used T₄ alone. Some of them fully recovered, some improved but didn’t fully recover no matter how high their dose, and many didn’t benefit at all no matter how much they used.

Most every one of our hypothyroid patients who had a poor response to T₄ alone fully recovered when they switched to a product containing both T₃ and T₄, or T₃ alone. Because of our patients’

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comparatively poor responses to T_4 alone, and their excellent responses to combined T_3 and T_4 or T_3 alone, we decided that it was inhumane to restrict them to the use of T_4 alone. Using our method of treatment, it took months for us to learn that a particular patient wasn't responding well to T_4 alone. We couldn't justify the wait, just to learn eventually that the patient had to switch to a generally more effective product.

If your doctor insists on treating you with T_4 alone, your protest will be a reasonable one. If he won't cooperate, you'll be smart to find another doctor who will.

Potential Harm From Too Low a Dose of Thyroid Hormone

For some patients, taking a low dose of thyroid hormone is worse than taking none at all. This is especially true if the patient is taking low-dose T_4 .

Low doses of T_4 may actually slow metabolism further. This can happen when the T_4 the patient is taking lowers her pituitary gland's secretion of TSH. The lowered TSH in turn reduces her thyroid gland's secretion of T_3 and T_4 . If her T_4 dose is too low to compensate for the thyroid gland's reduced release of T_3 and T_4 , her metabolism will slow further. To her surprise, her symptoms of hypothyroidism will worsen after she begins the use of the low dose of T_4 .

For a more detailed explanation of the harmful effects of low dose T_4 , see <www.drloewe.com/QandA/askdrloewe/t4therapy.htm>.