

Treatment Guidelines for Patients With Hyperthyroidism and Hypothyroidism

Peter A. Singer, MD; David S. Cooper, MD; Elliot G. Levy, MD; Paul W. Ladenson, MD; Lewis E. Braverman, MD; Gilbert Daniels, MD; Francis S. Greenspan, MD; I. Ross McDougall, MB, ChB, PhD; Thomas F. Nikolai, MD

Objective.--To develop a set of minimum clinical guidelines for use by primary care physicians in the evaluation and management of patients with hyperthyroidism and hypothyroidism.

Participants.--Guidelines were developed by a nine-member ad hoc Standards of Care Committee of the American Thyroid Association (the authors of this article). The participants were selected by the committee chair and the president of the American Thyroid Association on the basis of their clinical experience. The committee members represented different geographic areas within the United States, in order to take into account different practice styles.

Evidence.--Guidelines were developed on the basis of expert opinion of the participants, as well as on available published information.

Consensus Process.--Input was obtained from all of the participants, each of whom wrote an initial section of the document. A complete draft document was then written by three participants (P.A.S., D.S.C., and E.G.L.) and resubmitted to the entire committee for revision. The revised document was then submitted to the entire membership of the American Thyroid Association for written comments, which were then reviewed (mainly by P.A.S., D.S.C., and E.G.L.). Many of the suggestions of the American Thyroid Association members were incorporated into the final draft, which was then approved by the Executive Council of the American Thyroid Association. The entire process, from initial drafts to final approval, took approximately 18 months.

Conclusions.--A set of minimum clinical guidelines for the diagnosis and treatment of hyperthyroidism and hypothyroidism were developed by consensus of a group of experienced thyroidologists. The guidelines are intended to be used by physicians in their care of patients with thyroid disorders, with the expectation that more effective care can be provided, and at a cost savings.

(*JAMA*. 1995;273:808-812)

HYPERTHYROIDISM and hypothyroidism are highly prevalent conditions that usually come to the attention of the primary care physician first. In order to provide physicians practice guidelines for patients with thyroid disorders, the American Thyroid Association created an ad hoc Standards of Care Committee, charged with developing such guidelines, following the procedure outlined in the abstract. These guidelines were felt by the leadership of the American Thyroid Association to be an important step, hopefully leading to more cost-effective, as well as clinically appropriate, care. The clinical guidelines were designed to provide latitude in decision making, taking into consideration varying practice styles, as well as differences in patient presentations.

TREATMENT GUIDELINES FOR PATIENTS WITH HYPERTHYROIDISM

The term "hyperthyroidism" encompasses a heterogeneous group of disorders, all characterized by elevated levels of thyroid hormones in the blood. Since Graves' disease is the most common cause of hyperthyroidism, the following discussion will concentrate on that disorder.

Initial Visit

Medical History.--A detailed medical history will usually provide the clinician with sufficient clues to suggest the diagnosis of hyperthyroidism. Patients should be asked about nervousness, fatigue, palpitations, exertional dyspnea, weight loss, heat intolerance, irritability, tremor, muscle weakness, decreased menstrual flow in women, sleep disturbance, increased perspiration, increased frequency of

bowel movements, change in appetite, and thyroid enlargement. Patients should also be asked about photophobia, eye irritation, diplopia, or a change in visual acuity.

In individuals in whom Graves' disease is not obvious, questions regarding recent iodine exposure, prior or current thyroid hormone use, anterior neck pain, pregnancy, or history of goiter should be included. A family history of thyroid disease should be sought.

Physical Examination.--An appropriately thorough physical examination should be performed during the initial evaluation. Aspects of the examination to be stressed include weight and height, pulse rate and regularity, blood pressure, cardiac examination, thyroid enlargement (diffuse or nodular), proximal muscle weakness, tremor, an eye examination (for evidence of ophthalmopathy), and a skin examination (for pretibial myxedema).

Older individuals may have few if any symptoms and signs of hyperthyroidism except for weight loss and cardiac abnormalities, in particular atrial fibrillation and/or congestive heart failure.

Laboratory Evaluation.[\[ref. 1\]](#)--True hyperthyroidism must be distinguished from "euthyroid hyperthyroxinemia," which may be caused by certain drugs, nonthyroidal illness, and a variety of other, less common factors. Specific tests to establish the diagnosis of hyperthyroidism include an estimate or direct measurement of free thyroxine (T_4) (which is elevated in hyperthyroidism), as well as a serum thyroid-stimulating hormone (TSH) measurement (which is suppressed in hyperthyroidism). The TSH level should be measured in an assay that is sensitive enough to clearly discriminate euthyroid from hyperthyroid individuals. When the free T_4 level (estimate) is elevated in a clinically hyperthyroid patient, a serum TSH level that is not suppressed should alert the clinician to the possibility of hyperthyroidism due to a TSH-producing pituitary adenoma.

If hyperthyroidism is confirmed, other tests may be performed according to the clinical situation. These may include total triiodothyronine (T_3), thyroid autoantibodies, and a radioactive iodine uptake test. The latter test should be obtained if the diagnosis of Graves' disease is not secure; this may be the case in patients with "painless," postpartum, or subacute thyroiditis who will have low, rather than elevated, radioactive iodine uptake values.[\[ref. 1\]](#) Specific treatment should generally be withheld until the biochemical diagnosis and cause of hyperthyroidism are confirmed. In most instances, symptomatic relief can be obtained with beta-adrenergic-blocking drugs while the patient is undergoing additional diagnostic testing.

Treatment Plan

The treatment of Graves' hyperthyroidism is directed toward lowering the serum concentrations of thyroid hormones to reestablish a eumetabolic state. There are currently three available modalities of treatment, all of which are effective. These include antithyroid drugs (ATDs), radioactive iodine (^{131}I), and thyroid surgery.

The patient should have a clear understanding of the indications and implications of all forms of therapy, including risks, benefits, and side effects, and should be an active participant in the decision-making process regarding type of therapy. Because therapy is frequently ablative, the participation of an endocrinologist in the patient's treatment may be beneficial in those cases in which the primary care physician does not have experience with the disorder.

In patients with hyperthyroidism and a low radioactive iodine uptake, none of these therapies are indicated, since low-uptake hyperthyroidism usually implies thyroiditis, which generally resolves spontaneously. Therapy with beta-blocking agents is usually sufficient to control the symptoms of hyperthyroidism in these individuals.

Antithyroid Drugs.[\[ref. 2\]](#)--The ATDs, methimazole and propylthiouracil, inhibit thyroid hormone biosynthesis. They are useful either as a primary form of therapy or to lower thyroid hormone levels before (and in some cases after) radioactive iodine therapy or surgery. Long-term ATD therapy may lead to remission in some patients with Graves' disease. Initial daily doses of methimazole generally range from 10 to 40 mg, and for propylthiouracil, 100 to 600 mg. There is no clear-cut standard for duration of therapy with ATDs, but when used as primary therapy, they are usually given for 6 months to 2 years,

although a longer period of administration is acceptable. Some physicians prefer a regimen of combined ATD and thyroid hormone to avoid frequent adjustments of ATD doses.

Adverse reactions to both methimazole and propylthiouracil occur, including rash, itching, and less commonly, arthralgias or hepatic abnormalities. Hepatic necrosis caused by propylthiouracil and cholestatic jaundice caused by methimazole are sufficiently rare enough that routine monitoring of liver function tests is unnecessary. The most serious reaction to either drug is agranulocytosis, which occurs in about 0.3% of patients. Patients should be cautioned about the side effects of ATD prior to the initiation of therapy. Some clinicians obtain white blood cell (WBC) counts prior to initiating ATD, since mild leukopenia is common in Graves' disease. A baseline WBC may therefore be useful for comparison if subsequent WBC counts are obtained.

Patients developing fever, rash, jaundice, arthralgia, or oropharyngitis should promptly discontinue their medication, contact their physician, and have appropriate laboratory studies including a complete blood cell count with WBC differential.

Lithium carbonate or stable iodine has been used to block release of thyroid hormone from the thyroid gland in patients who are intolerant to ATDs, although their use is infrequent.

Radioactive Iodine Therapy. [ref. 3]--Radioactive iodine (^{131}I) is the most commonly used form of treatment in the United States. It is safe, the principal side effect being the early or late development of hypothyroidism, necessitating life-long thyroid hormone replacement following ^{131}I treatment. Treatment with ^{131}I does not cause a reduction in fertility and does not cause cancer, nor has it been shown to produce ill effects in offspring of those so treated prior to pregnancy. It is contraindicated during pregnancy. Its use in individuals under the age 20 years, while controversial, is common. Pregnancy needs to be excluded before ^{131}I is administered to young women and should be deferred for a few months following therapy. Therapy with ^{131}I is also contraindicated in women who are breast-feeding. Elderly patients or individuals at risk for developing cardiac complications may be pretreated with ATDs prior to ^{131}I therapy, especially if hyperthyroidism is severe, to deplete the gland of stored hormone, thereby minimizing the risk of exacerbation of hyperthyroidism due to ^{131}I -induced thyroiditis. In some patients, ATDs may be required for control for several months following radioiodine therapy. A radioactive iodine uptake test is usually performed just prior to the administration of ^{131}I to determine the appropriate dose.

Surgery. [ref. 4]--Thyroidectomy is infrequently recommended for patients with Graves' disease. Specific indications include patients with very large goiters who may be relatively resistant to ^{131}I , those who have coincidental thyroid nodules, pregnant patients allergic to ATDs, and patients who are allergic to ATDs and/or do not wish ^{131}I therapy. The procedure should be performed only by an experienced surgeon and only after careful medical preparation. Patients must be cautioned about potential complications of surgery, including hypoparathyroidism and injury to the recurrent laryngeal nerve. Hyperthyroidism may persist or recur if insufficient thyroid tissue is removed, whereas hypothyroidism usually develops after near-total thyroidectomy.

Adjunctive Therapy.--The most useful adjuncts are beta-adrenergic blockers such as propranolol or nadolol, which can provide symptomatic improvement until the euthyroid state has been achieved. Patients who cannot tolerate beta-blockers may be treated with calcium channel blockers such as diltiazem.

Continuing Care

Since the treatment of hyperthyroidism may last for a few years, a follow-up plan must be established.

Antithyroid Drugs.--Patients treated with ATDs should generally be seen initially at 4- to 12-week intervals, depending on the severity of the illness, until euthyroidism is achieved. At this time, the ATD dose can often be reduced. Patients are then monitored every 3 to 4 months thereafter while continuing to take ATDs. An interval examination should include weight, pulse, blood pressure, thyroid, and an eye examination. Thyroid function tests should include an estimation of free T_4 , and if clinical symptoms and signs of hyperthyroidism are present, a T_3 determination may also be indicated. The serum TSH level may remain suppressed for several months even after T_4 and T_3 levels normalize, yielding potentially misleading laboratory results.

Once ATDs are discontinued, patients should be seen at 4- to 6-week intervals for the first 3 to 4 months after the medication is stopped, and then at increasing intervals for the duration of the first year. If clinical and biochemical euthyroid status persists, patients should be evaluated yearly for the next 2 to 3 years and at increasing intervals thereafter.

Radioactive Iodine.--Patients should be seen at 4- to 6-week intervals for the first 3 months following radioactive iodine therapy, and then at intervals as the clinical situation dictates. Hypothyroidism generally ensues following treatment within the first 6 to 12 months following therapy, but may occur at any time. Therefore, at least annual follow-up is necessary for those individuals who continue to be euthyroid. Levothyroxine sodium should be administered when sustained hypothyroidism develops, the end point of replacement therapy being a normal free T₄ estimate and TSH level. Once patients are on a stable dose of levothyroxine, they may be followed at yearly intervals. At subsequent visits, a serum TSH measurement is probably sufficient to assure the adequacy of therapy.

Surgery.--After thyroidectomy, the patient should be followed as warranted for postoperative care, and at approximately 2 months after surgery, to assess thyroid status. Recurrent hyperthyroidism can occur after surgery, but hypothyroidism is far more common, and depends primarily on the size of the thyroid remnant. If levothyroxine therapy is necessary, patients can be followed at yearly intervals after establishing clinical and biochemical euthyroidism. Patients who are euthyroid following surgery should also be followed yearly, using the serum TSH level to document euthyroidism.

Special Problems

Hyperthyroidism and Pregnancy.[\[ref. 5\]](#)--Pregnancy may be adversely affected by poorly controlled hyperthyroidism, with an increased rate of fetal loss. The goal of treatment during pregnancy is to maintain euthyroidism, using the smallest doses of ATDs possible. Propylthiouracil is preferred in pregnancy because it crosses the placenta less than methimazole, but methimazole is not contraindicated, and is used successfully by some clinicians. Since pregnancy itself has an ameliorative effect on Graves' disease, low doses or even discontinuation of ATDs may be possible in the third trimester.

Hyperthyroid pregnant patients should be seen at 4- to 6-week intervals (or more frequently as the situation dictates), with a collaborative effort between the treating physician and the obstetrician.

Thyroid-stimulating immunoglobulin titers, obtained in the last trimester, may predict the likelihood of neonatal hyperthyroidism, but any newborn from a mother who has a history of hyperthyroidism should be observed for this possibility. Patients treated for hyperthyroidism during pregnancy should be reevaluated 6 weeks post partum, since there can be postpartum worsening of the disease.

If surgery is felt to be necessary because of inability to adequately control hyperthyroidism with ATDs, it should preferably be performed when the chance for fetal survival is likely in the event of early delivery.

Graves' Ophthalmopathy.[\[ref. 6\]](#)--The minority of patients with Graves' disease have clinical eye involvement, which may even develop after the diagnosis and treatment of hyperthyroidism. Milder eye symptoms include excess tearing, photophobia, and a feeling of grittiness. More severe symptoms include proptosis, diplopia, eye pain, and a decrease in visual acuity. Physical findings may include eyelid retraction, conjunctival injection and suffusion (chemosis), proptosis (either unilateral or bilateral), periorbital edema, and ophthalmoplegia.

Exposure keratitis may occur when the patient is unable to close the eyelids completely. When eye

disease occurs in patients with known hyperthyroidism, no specific laboratory tests are required to confirm the diagnosis. When ophthalmopathy occurs in patients who are biochemically euthyroid, autoimmune thyroid disease should be suspected, and the diagnosis can be confirmed by the finding of antimicrosomal (antithyroperoxidase [anti-TPO]) antibodies or thyroid-stimulating antibodies in the serum. In euthyroid patients, orbital computed tomography or magnetic resonance imaging may be indicated to exclude the diagnosis of other orbital diseases that can mimic thyroid ophthalmopathy.

Therapy of Graves' eye disease is directed toward restoring thyroid function to normal, as well as treating the eye symptoms. Sunglasses (to decrease photophobia) and artificial tears (for lubrication) may be helpful. For periorbital edema, elevation of the head of the bed while sleeping, as well as the judicious use of diuretics, may be useful. Systemic glucocorticoids have been used by some physicians in patients with

active ophthalmopathy, in an effort to prevent its progression, particularly after ^{131}I therapy, but their efficacy is not fully established. Management of patients with more than mild symptoms and signs should be carried out in conjunction with an ophthalmologist.

Toxic Nodular Goiter.[\[ref. 7\]](#)--Toxic nodular goiter (TNG), or Plummer's disease, is more common than Graves' disease in elderly patients. The hyperthyroidism may be caused by multiple hyperfunctioning nodules or, less frequently, a single hyperfunctioning nodule. The disorder should be differentiated from Graves' disease. Ophthalmopathy is not present in patients with TNG.

Diagnostic approaches in a patient with suspected TNG include the thyroid function tests mentioned previously in the section on Graves' hyperthyroidism. The absence of thyroid autoantibodies may help to differentiate TNG from Graves' disease. The radioiodine uptake and thyroid scan may be useful in patients with TNG to determine whether a dominant nodule is hypofunctioning, suggesting the need for needle aspiration to rule out thyroid carcinoma. Although ^{131}I is usually recommended for the treatment of TNG, surgery is appropriate for certain individuals who prefer surgery and are good operative risks, as well as for children, adolescents and young adults, and in those patients with large goiters, or if there is concern about thyroid malignancy.

As is the case with Graves' disease, elderly patients with TNG may be treated first with ATDs until they become euthyroid, followed by ^{131}I therapy. Surgery may be indicated if there is a very large goiter or if symptoms of tracheal or esophageal compression are present. Patients with solitary hyperfunctioning thyroid nodules are usually treated with radioiodine, but surgery is equally appropriate for children and adolescents.

Thyroid Storm.[\[ref. 8\]](#)--Thyroid storm is a life-threatening, clinical syndrome characterized by exaggerated signs and symptoms of hyperthyroidism, fever, and altered mental status. While it usually occurs in individuals with Graves' disease, it has also been reported in patients with other causes of hyperthyroidism. Thyroid storm is usually precipitated by a concurrent illness or injury, but has been reported to occur spontaneously following withdrawal of ATDs or following radioactive iodine therapy for hyperthyroidism.

There are no specific laboratory findings distinguishing thyroid storm from uncomplicated hyperthyroidism. Thus, when the diagnosis is suspected clinically, therapy must be initiated immediately. Treatment should be initiated in the intensive care unit, and consists of providing supportive measures, treating the precipitating cause, and administering specific pharmacologic agents such as (1) drugs that inhibit thyroid hormone biosynthesis (propylthiouracil or methimazole); (2) drugs that inhibit release of thyroid hormone from the thyroid gland (eg, potassium iodide, lithium carbonate, ipodate); and (3) agents that decrease the peripheral effects of thyroid hormone (eg, propylthiouracil, corticosteroids, ipodate, iopanoic acid). The selection of drugs depends on the specific clinical situation. Because of the complexity of thyroid storm, it is recommended that an endocrinologist participate in the evaluation and management of such patients.

TREATMENT GUIDELINES FOR PATIENTS WITH HYPOTHYROIDISM

Initial Visit

Hypothyroidism is a disorder of diverse causes in which the thyroid gland fails to secrete adequate amounts of thyroid hormone. The overwhelming majority of cases are due to primary thyroid gland failure because of chronic autoimmune (Hashimoto's) thyroiditis, radioactive iodine therapy, or surgery. Therefore, the following discussion will emphasize primary hypothyroidism.

Medical History.--A comprehensive medical history can uncover symptoms that will help establish the diagnosis in the patient with previously undiagnosed hypothyroidism. If the diagnosis has already been made, it is important to confirm it by history and to document pretreatment thyroid function abnormalities whenever possible. In the past, patients frequently were treated with thyroid hormone for reasons that would not be acceptable by current standards. In addition, many patients previously treated with thyroid hormone have forgotten the reasons for this therapy, as well as the adequacy of their clinical response. Patients should be asked about symptoms of tiredness, weakness, fatigue, sleepiness, cold intolerance, dry

skin, hoarseness, constipation, joint pains, muscle cramps, mental impairment, depression, menstrual disturbances in women and especially menorrhagia, infertility, and weight gain.

Physical Examination.--A comprehensive physical examination should be performed during the initial evaluation. Findings from the physical examination that may indicate hypothyroidism include goiter or a nonpalpable thyroid gland, bradycardia, edema, hoarseness, delayed relaxation of deep tendon reflexes, slow speech, and cool, dry skin.

Laboratory Evaluation.[ref. 1]--To establish the diagnosis of hypothyroidism, a serum TSH measurement and a free T₄ estimate (or direct measurement) should be performed. When autoimmune thyroiditis is the suspected underlying cause, it is helpful to confirm antithyroid antibody titers, either antimicrosomal antibody (anti-TPO antibody) or antithyroglobulin antibody. The antimicrosomal antibody test is more sensitive and specific. If the TSH level is low, inappropriately normal, or insufficiently elevated in the presence of low T₄ values, central hypothyroidism caused by hypothalamic or pituitary disease should be excluded before starting thyroid replacement therapy. Also, thyroid function tests obtained from ill hospitalized patients must be interpreted with caution, since serum T₄ and/or TSH levels may suggest hypothyroidism.

Treatment Plan.[ref. 9]--Levothyroxine sodium is the treatment of choice for the routine management of hypothyroidism. Levothyroxine preparations are manufactured in many different dosages and allow precise titration of an individual patient's requirements. Adults with hypothyroidism require approximately 1.7 microg/kg of body weight per day for full replacement. Children may require higher doses (up to 4 microg/kg of body weight per day). Older patients may need less than 1 microg/kg per day. Therapy is usually initiated in patients under the age of 50 years with full replacement. For those patients who are older than 50 years, or in younger patients with a history of cardiac disease, a lower initial dosage is indicated, starting with 0.025 to 0.05 mg of levothyroxine daily, with clinical and biochemical reevaluations at 6- to 8-week intervals until the serum TSH concentration is normalized. Some individuals older than 50 years, such as those recently treated for hyperthyroidism or those known to have had hypothyroidism for only a short time, such as a few months, may be treated with full replacement doses of levothyroxine. Certain drugs, eg, cholestyramine, ferrous sulfate, sucralfate, and aluminum hydroxide antacids, may interfere with levothyroxine absorption from the gut. Levothyroxine administration should be spaced at least 4 hours apart from these medications. Other drugs, especially the anticonvulsants phenytoin and carbamazepine and the antituberculous agent rifampin, may accelerate levothyroxine metabolism, necessitating higher levothyroxine doses.

Continuing Care

Periodic monitoring is essential in the management of patients with hypothyroidism to judge the clinical response to treatment, patient compliance in taking the medication, and development of drug interactions, and to adjust the replacement dosage because of changes in body weight or advancing age. Patients should be evaluated initially about every 6 to 8 weeks to monitor the response to the dose of levothyroxine. Once the TSH concentration has been normalized, the need for frequent visits is reduced. Visit frequency of every 6 to 12 months is then sufficient, depending on the clinical situation. Should it be necessary to adjust a patient's dosage, he or she should return in 2 to 3 months to assess the therapeutic response and to remeasure the TSH concentration.

An interim history should assess response to therapy with thyroid hormone, evaluating clinical improvement in symptoms, as well as possible side effects of the medication. A physical examination relevant to the thyroid status should be performed annually. A TSH concentration should be measured at least annually. For patients who have recently started receiving levothyroxine or who have had their dosage, type, or brand of thyroid preparation changed, the TSH concentration should be measured after 8 to 12 weeks.

Special Considerations

Elderly Persons.[ref. 10]--In many cases, hypothyroidism in elderly patients is characterized by a paucity of specific signs and symptoms. The symptoms may be subtle and include hoarseness, deafness,

confusion, dementia, ataxia, depression, dry skin, or hair loss. Because of high prevalence of hypothyroidism in women past 60 years of age, it is recommended that such individuals be screened with a serum TSH measurement. All patients with a prior history of any medically or surgically treated thyroid disease should be screened with a serum TSH measurement yearly. In addition, patients with other autoimmune diseases and those with unexplained depression, cognitive dysfunction, or hypercholesterolemia should be screened with TSH measurements. Therapy should be directed at using the dose of levothyroxine required to maintain normal TSH concentrations.

Pregnancy.[ref. 11]--During pregnancy, many hypothyroid patients have an increase in levothyroxine requirement, which can be detected with a TSH measurement. The patient should be checked during each trimester to make sure that the TSH concentration is still normal, with further adjustments as indicated by the appropriate testing. The levothyroxine dose should return to the prepregnancy dose immediately after delivery and a serum TSH level should be obtained 6 to 8 weeks post partum.

Iatrogenic Hyperthyroidism.[ref. 9][ref. 12]--Some patients, especially elderly patients, tolerate the effects of excess T_4 poorly. If symptoms of palpitations, tremor, difficulty in concentrating, or chest pain develop, the patient should be evaluated with appropriate tests, and if hyperthyroidism is confirmed, the current dose of levothyroxine should be withheld for 1 week and restarted at a lower dose. Other patients remain asymptomatic despite elevations of free T_4 and/or suppression of TSH concentrations. Since levothyroxine overreplacement has been associated with reduced bone mineral content, particularly in postmenopausal women, it is recommended that these patients have their dose reduced until the TSH concentration is normalized, unless TSH suppression is the objective, as in patients with a history of well-differentiated thyroid cancer.

Subclinical Hypothyroidism.[ref. 9, 10]--As many as 15% of patients older than 65 years, as well as many other adults, have a normal free T_4 estimate (or normal direct free T_4 measurement) and an elevated TSH concentration, but few, if any, hypothyroid symptoms. This state is referred to as "subclinical hypothyroidism." Some patients with this mild disorder feel better when treated with levothyroxine. Therapy for subclinical hypothyroidism is probably advisable, especially if thyroid autoantibodies are positive, because overt hypothyroidism develops with high frequency in such patients. If the physician decides not to treat these patients, they should be evaluated at yearly intervals for evidence of more severe clinical and biochemical loss of thyroid function.

Myxedema Coma.[ref. 8]--Coma caused by myxedema is a rare, life-threatening state in which severe, usually long-standing hypothyroidism markedly worsens. In general, it occurs in elderly individuals and is usually precipitated by an intercurrent medical illness. The clinical manifestations, in addition to obtundation or coma, may include hypothermia, bradycardia, respiratory failure, and even cardiovascular collapse.

Therapy of myxedema coma includes intravenous administration of levothyroxine and/or liothyronine sodium as well as pharmacologic doses of glucocorticoids. Also, precipitating or associated disorders must be aggressively treated.

Patients with myxedema coma should be treated in the intensive care unit with appropriate monitoring and with the participation of an endocrinologist.

Use of Other Thyroid Hormone Preparations[ref. 9]

There are few indications for thyroid hormone preparations other than levothyroxine. Liothyronine may be useful prior to treatment of thyroid cancer with radioactive iodine, as patients can be withdrawn from liothyronine for shorter periods of time than levothyroxine. Chronic liothyronine therapy for hypothyroidism is not recommended, since its use is associated with a greater degree of iatrogenic hyperthyroidism. Some individuals, especially elderly individuals, are very sensitive to the deleterious effects of T_3 . Biological and synthetic thyroid hormone preparations containing both T_4 and T_3 are also not currently recommended for therapy since they produce fluctuating and often elevated T_3 concentrations, although their use is not necessarily contraindicated.

Use of Thyroid Hormone for Other Situations[\[ref. 13\]](#)

Thyroid hormone therapy has been used for nonthyroidal problems, including obesity, infertility, menstrual irregularity, short stature, and chronic fatigue. There is no scientific proof that such conditions respond to thyroid hormone therapy and its use is not felt to be appropriate. Some psychiatrists, however, report the benefit of adding thyroid hormone medication to tricyclic antidepressants in selected patients with depression, and clinical improvements have been noted.

From the Division of Endocrinology, Diabetes, and Hypertension, University of Southern California School of Medicine, Los Angeles (Dr Singer); Division of Endocrinology, Johns Hopkins University School of Medicine, Baltimore, Md (Drs Cooper and Ladenson); Division of Endocrinology, Mt Sinai Hospital, Baltimore, Md (Dr Cooper); Division of Endocrinology, University of Miami (Fla) School of Medicine (Dr Levy); Division of Endocrinology, University of Massachusetts Medical School, Worcester (Dr Braverman); Thyroid Unit, Massachusetts General Hospital, Boston (Dr Daniels); Division of Endocrinology, University of California, San Francisco School of Medicine (Dr Greenspan); Division of Nuclear Medicine, Stanford (Calif) University School of Medicine (Dr McDougall); and Marshfield (Wis) Clinic (Dr Nikolai).

The authors gratefully acknowledge the expert secretarial assistance of Elsa C. Ahumada.

Reprint requests to University of Southern California, 1355 S San Pablo St, Room 120, Los Angeles, CA 90033 (Dr Singer).

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