Medical Intelligence

Anesthetic Management of the Patient with Hyperthyroidism

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HYPERTHYROIDISM, a complex of physiologic and biochemical aberrations resulting from hypersecretion of the thyroid hormones thyroxine (T4) and/or triiodothyronine (T3), is most frequently associated with diffuse hyperplasia of the thyroid gland. The disease may also result from excess hormonal secretion by one or more functioning thyroid nodules, ectopic thyroid tissue, or more rarely, chorionic tissue of a hydatidiform mole or chorionic carcinoma. Thyrotoxicosis occurs approximately 4.5 times more frequently in females, with a peak incidence in the third and fourth decades of life.30

Synthesis of Thyroid Hormones

Thyroid hormone synthesis and release depend upon a number of interrelated reactions (fig. 1). Extracellular iodine, derived from dietary intake, is actively transported into thyroid follicular cells. The iodine is oxidized, with H₂O₂ presumably serving as the oxidizing agent. Iodination of thyroglobulin follows. Thyroglobulin, a complex glycoprotein whose molecular weight is approximately 660,000, is composed of about 5,650 amino acid residues, 8–10 per cent carbohydrate, and a variable amount of iodine. The structure of the polypeptide is influenced by the extent of iodination, but iodine is seldom present in quantities greater than 1 per cent by weight. Approximately 120 tyrosyl units composed of thyroxine (T4), triiodothyronine (T3), monoiodotyrosine (MIT) and diiodotyrosine (DIT) residues are contained in the thyroglobulin molecule, which is stored in the follicular lumina. After resorption into thyroid cells, thyroglobulin is hydrolyzed, releasing the active hormones T4 and T3 and the iodothyronines. Thyroid-stimulating hormone (TSH) activation of lysosomal enzymes is thought to be responsible for the proteolysis. Iodothyronines are deiodinated and the iodine recycled through the iodine pool while T3 and T4 gain access to the extracellular fluid and thence to the circulation. Of the 120 tyrosyl units, only 10 are eventually converted to iodothyronines.81

Actions of Thyroxine and Manifestations of Hyperthyroidism

Thyroid hormones exert influences upon nuclear, ribosomal, and mitochondrial functions. Hormonal excess produces structural changes in mitochondria and functional alterations in oxidative phosphorylation. The mitochondria are larger than normal and the total number and area of cristae in each mitochondrion is increased five to ten times.64 It is suggested that in hyperthyroidism there is an increase in the content of respiratory enzymes, which have been shown to be associated with the mitochondrial membrane. Only mitochondria from tissues which respond to T4 with increased oxygen consumption (heart, kidney, and liver) have been shown to swell in vitro.13 Mitochondria liberate energy through substrate oxidation. This energy is transformed into the utilizable high-energy phosphate bonds of adenosine-triphosphate (ATP). This transformation of the energy liberated by oxidation

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into phosphoryl-bond energy is termed coupling. Thyroxine induces uncoupling both in vitro and in vitro.25 This uncoupling of oxidative phosphorylation by the thyroid hormones appears to be dose-related. Low concentrations increase the oxidative process without markedly influencing energy transfer. High concentrations so alter the functional organization of the mitochondria that, though hyperoxidation continues, energy cannot be trapped by the phosphorylating system.

In hyperthyroidism, heat production is increased by the hyperoxidative state and the decreased efficiency of energy conversion.26 Many of the clinical manifestations of thyrotoxicosis are related to compensatory mechanisms for dissipation of this excess heat. These include profuse sweating, vasodilation, and tachycardia.

Patients without evidence of other forms of heart disease may develop clinical, radiologic, and electrophysiologic evidence of cardiac dysfunction, including cardiomegaly, pulmonary edema, peripheral edema, and arrhythmias. Refractoriness to the cardiac glycosides is the rule.25 Hyperthyroid patients have been shown to have lower serum digoxin levels than euthyroid patients, regardless of route of administration.27 Graves' disease is commonly classified as a disorder of high-output cardiac failure, but is more appropriately termed a "hyperkinetic circulatory state." The cardiac output is raised in excess of requirements imposed by the hypermetabolic state. Thyroid hormones probably produce this high output by inducing vasodilation of the circulatory beds supplying muscle and skin. The heart responds to the low peripheral resistance with an accelerated rate and augmented stroke volume. In addition, ventricular contractility is enhanced. These cardiovascular responses appear to be a direct effect of the thyroid hormones, perhaps independent of autonomic influences.25,29-30 Thyrotoxic patients without other evidence of cardiovascular disease may have EKG changes consistent with left ventricular hypertrophy.25 Older hyperthyroid patients may have an unimpressive history and minimal physical findings except for the cardiovascular system. Patients with so called "masked" or "apathetic hyperthyroidism" often present with arrhythmias or frank congestive heart failure.

Other manifestations of increased titers of thyroxine are fine tremor, dyspnea on exertion, increased appetite in the face of progressive weight loss, menstrual irregularities, insomnia, and emotional lability.

Hepatosplenomegaly occurs in approximately 2 per cent of patients.44 Jaundice is rare.45 Diminished glycogen synthesis has been shown to occur in thyrotoxicosis.47 Decreased hepatic glycogen content is presumably an etiologic factor in the hepatic pathology. Hyperpigmentation, vitiligo, pretibial myxedema, generalized or localized alopecia, and pruritus are reflections of cutaneous involvement.

Thyroid myopathy is common. Some reduction in the power of muscular contraction probably occurs in every patient with hyperthyroidism.2,64 The complaint of easy fatiguability and weakness of the legs when climbing stairs is frequent.4 The proximal muscles are more extensively involved and atrophy may be evident. Abnormal electromyograms have been found in more than 50 per cent of a limited number of patients investigated.45,46 The abnormal electromyogram is a manifestation of biochemical dysfunction.

Although muscle contraction depends primarily upon ATP as an energy source, phosphocreatine is an important intermediary in the cycle of energy release. In skeletal muscle, the two are in equilibrium, the exchange being catalyzed by the enzyme phosphokinase. Thyroxine has been shown, both in vitro and in vivo, to inhibit creatine phosphokinase.6,37 It may be reasoned that, as a result of uncoupling of oxidative phosphorylation, the muscles of thyrotoxic patients perform with decreased efficiency. In addition, the inability of muscle to synthesize or fix phosphocreatine results in creatinuria and decreased tolerance to exogenous creatine.60 The extent of muscular involvement is roughly proportional to the severity and duration of the thyrotoxicosis and resolves when the euthyroid state is restored.

Ocular pathology occurs in the majority of patients and bears no consistent relationship to metabolic state. At least 50 per cent of patients manifest such mild eye changes as upper lid retraction (Dalrymple's sign), lid lag (von Graefe's sign), and infrequent blinking (Stellwag's sign). Serious changes occur in
approximately 5 per cent of thyrotoxic patients, including proptosis, extraocular muscle, corneal and optic nerve and soft tissue involvement.17

Although ocular pathology may cause him to seek medical attention, most often the patient complains of nervousness, palpitations or fatigue. Frequently the precipitating event appears to be an emotional crisis, often superimposed upon a chronically stressful situation. It has been stated that more than 80 per cent of, and perhaps all, hyperthyroid patients are very seriously disturbed emotionally at the time they become ill. Other investigators have stressed the striking similarities in the developmental dynamics of the personality structure of patients with hyperthyroidism.25

The pathogenesis of Graves' disease appears to involve an autoimmune process as well as alterations in biochemical functions of thyroid hormones at the mitochondrial level. An abnormal thyrotropic-like substance, long-acting thyroid stimulator (LATS) can be identified in the sera of at least 80 per cent of patients with Graves' disease.7,26-28 LATS is a 7s γ-globulin apparently produced by lymphoid tissues.23 The action of LATS on the thyroid appears to be identical to that of TSH except for its half-life.1 However, while TSH appears to localize in thyroid cell nuclei, LATS has an affinity for the cytoplasm.7 The nature of the thyroid component reacting with LATS is unknown. It is tempting to speculate that this component is a thyroid chalone, that is, one of the tissue-specific inhibitors of mitosis which are thought to be active in regulating organ size and growth. Thus, when LATS reacts with the mitotic inhibitor it could induce the thyroid hyperplasia and overactivity characteristic of Graves' disease. Although the exact role of LATS is not defined, it might be related to an autoimmune phenomenon, with its likelihood of occurrence being genetically determined. There are increased prevalences of goiter, Graves' disease, and thyroid antibodies in the relatives of patients with Graves' disease. In addition, the incidences of other organ-specific autoimmune diseases such as idiopathic Addison's disease and pernicious anemia are significantly higher in patients who have Graves' disease.29

Relation of Thyroid and Adrenal Hormones

The relation of the adrenal cortex to the thyroid has been the subject of extensive speculation and investigation.52 Glucocorticoid administration usually has a clinically insignificant effect on thyroid function; however, euthyroid patients receiving large doses of cortisone for several weeks have shown
marked suppression of thyroid function.23,31 Prednisone administered to hyperthyroid patients has depressed radioactive iodine uptake and serum PBI, altered the pattern of thyroxine binding in serum,36 and induced the disappearance of high serum LATS titers.37,53 In addition, a clinically euthyroid state has been produced in some patients receiving only steroid therapy for hyperthyroidism.84 Current data indicate that the corticosteroids diminish thyroid function by suppressing secretion of TSH.41,85 On the other hand, thyrotoxicosis in patients on prolonged high doses of steroids has been reported.11

Hyperplasia of the adrenal cortex in hyperthyroid patients has been described. It has been suggested that excess thyroid hormone may increase metabolic destruction of the corticosteroids, creating a relative adrenal cortical insufficiency, thereby promoting hypersecretion.27

More interest and far more confusion exists concerning the relationship of the adrenal medulla to the thyroid.12,27,30 There is a striking similarity between adrenergic hyperactivity and hyperthyroidism. An early report of increased urinary excretion of epinephrine and norepinephrine by hyperthyroid patients has not been confirmed by subsequent investigators.31,88 Significant elevation of plasma epinephrine has been demonstrated in a few hyperthyroid patients,23 but most investigators have found serum levels of catecholamines and their metabolites to be normal.30 Enhanced sensitivity to endogenous and exogenous catecholamines, generally an accepted feature of the hyperthyroid state, has recently been questioned.31 The beneficial effects of alpha- and beta-adrenergic blocking agents will be dealt with in the subsequent section on anesthetic management of the patient with thyrotoxicosis.

Preparation for Anesthesia and Operation

Medical control of hyperthyroidism is usually simple and complete. However, unreliable patients will not take antithyroid drugs; hypersensitive patients who develop agranulocytosis or severe cutaneous disorders cannot take them. The pregnant woman and the patient with respiratory obstruction secondary to a large gland may require surgery without the benefit of being rendered euthyroid. In addition, the anesthesiologist may be called upon to manage a hyperthyroid patient requiring emergency surgery for an unrelated condition.

When time and individual tolerance permit, the patient should receive antithyroid drugs (fig. 2). Propylthiouracil (Propaceil) or Methimazole (Tapazol) may be used. Mean time of onset of action of the thioureylenes is 8 days, and 6–7 weeks are required before the euthyroid state is achieved.24 The thioureylenes interfere with each step of hormone synthesis, the coupling reaction being somewhat more sensitive than iodination of thyroglobulin.

Iodine should be administered for 7–10 days prior to operation. Acute administration of excess iodine causes a transient inhibition of thyroid hormone formation, the WolffChaikoff effect.29 However, iodine often produces amelioration of symptoms and a decrease in circulating hormonal iodine which occurs too rapidly to be due to inhibition of hormonal secretion. There is evidence that this is a direct effect on the gland rather than antagonism of TSH. In addition to inhibition of hormonal synthesis and secretion, iodine also decreases vascularity and hyperplasia of the overactive gland. Patients treated with iodides may, after therapy in excess of two weeks, compensate to partially overcome the induced decrease in hormone secretion with gradual return toward pretreatment status, the phenomenon of "iodide escape."

Lithium has been shown to act similarly to iodide in preventing iodine release from the thyroid. Theoretically, lithium may be preferable to iodine when combined with the thioureylenes because of the role of iodine as a thyroid hormone substrate and uncertainty regarding complete block of iodination by propylthiouracil or methimazole block. Preliminary investigations appear compatible with this concept.24

If antithyroid drugs are not used, or cannot be administered for a period of time sufficient to achieve a euthyroid state, adrenergic blocking agents and corticosteroids may be used with iodine to render the patient fit for anesthesia and operation.

Although the exact role of the adrenergic nervous system in thyrotoxicosis is uncer-
tain, studies of catecholamine blockade in hyperthyroid states suggest that it may be highly significant. Definite clinical improvement occurs in most patients receiving reserpine. This is presumably secondary to depletion of catecholamine stores. Tremor, muscular weakness, palpitation, dyspnea, and eye signs are usually attenuated. A decreased pulse rate is most often the first objective evidence of drug effect, while blood pressure is little affected. The gland does not change in size, and laboratory evidence of hyperthyroidism persists. Carcinoid syndrome following intramuscular reserpine therapy has been reported in thyroxic patients, all of whom became subjectively and objectively worse. However, most patients are benefited by oral administration of 0.75-1 mg or, in more serious cases, intramuscular administration of 7.5-15 mg per day, in divided doses.

The effect of guanethidine (Ismelin) on cardiovascular dynamics has been studied somewhat more extensively. Elevated heart rate, blood pressure, cardiac output, and cardiac work of the thyroxic patient tend to return toward normal. Oxygen consumption is not significantly lowered. The mechanism of action of guanethidine is similar to that of reserpine. Present evidence indicates that the sympathetic nervous system is not responsible for the elevated oxygen consumption in hyperthyroidism. Significant improvement in the signs and symptoms of thyrotoxicosis may be noted as early as the third day of therapy. Blepharoptosis and/or postural hypotension are the end-points of therapy. The average daily dose is 80 mg.

Alpha-methyl dopa (Aldomet) forms a false neurotransmitter and interferes with the actions of catechols in tissues, as well as exerting a centrally mediated hypotensive effect. Its acute administration slows the pulse rate of the patient with hyperthyroidism but does not significantly change the clinical manifestation of the disease or reduce the elevated cardiac output or oxygen consumption.

Beta-adrenergic receptor blockade with propranolol (Inderal) has been shown to control the peripheral manifestations of thyrotoxicosis successfully. Cardiac output is reduced; oxygen consumption is unchanged. Heart rate is usually slowed; however, it does not decrease to normal levels, suggesting that the circulatory changes of hyperthyroidism are mediated only in part through the beta-sympathetic receptors, and that thyroxine has an independent action on the myocardium. Occasionally, patients refractory to other therapeutic agents will respond to propranolol. The usual dosage is 40 mg four times a day, administered orally. This potent myocardial depressant should be used with extreme caution in managing patients with cardiac failure. Opinion regarding discontinuing propranolol prior to anesthesia is divided, but some anesthesiologists prefer that the patient not receive the drug for one or more days before operation. The half-life of propranolol has been shown to be 3.4 to 6 hours. Therefore, discontinuing
the drug 24 to 48 hours prior to operation should allow sufficient time for complete recovery from the parent drug and any possibly active metabolites. It must be recognized that withdrawal of propranolol in a patient dependent on beta-adrenergic blockade for control of his cardiovascular symptoms may precipitate a thyroid crisis or cardiac decompensation.

Combined alpha- and beta-blockade using phenoxybenzamine (Dibenzyline) and propranolol has been shown to produce rapid alleviation of thyrotoxic symptoms and to reduce oxygen consumption by 12 per cent while leaving results of laboratory tests unaltered.27

The suppressive effect of adrenocortical steroids on thyroid function has been discussed. Therapy for one week prior to operation with adequate daily doses of corticosteroids is recommended in patients who are not clinically euthyroid. Prednisolone, 25 mg, dexamethasone (Decadron), 4 mg, or hydrocortisone, 100 mg, may be used. An additional 200 mg of hydrocortisone or its equivalent is administered intravenously during operation.

All antithyroid drugs, alpha- and beta-adrenergic blocking agents, steroids, and iodine are abruptly discontinued postoperatively.

Anesthetic Management of the Patient with Hyperthyroidism

Adequate premedication is essential. Although morphine is regarded by some as the agent of choice, it may be contraindicated, at least on a theoretical basis, because narcotics can stimulate the sympathoadrenal axis.24,25 The author prefers the short-acting barbiturates and/or diazepam (Valium) as premedicants. An optimal and individualized dose can be arrived at by "trial premedication," a few days prior to operation. In this way dosage can be adjusted to obtain the desired state of sedation and tranquility. Anticholinergic agents are often omitted because they interfere with the sweating mechanism. However, atropine has been utilized to confirm the adequacy of antithyroid treatment. Patients may be considered euthyroid if 0.6 mg atropine does not cause the pulse to increase more than 30 beats/min or exceed 120/min.27

Chlorpromazine (Thorazine) has been reported to cause reduction of the autonomic manifestations of thyrotoxicosis and decrease the basal metabolic rate. It has been shown to lower oxygen consumption and inhibit the calorigenic effects of parenterally administered thyroxine.26 However, it may be relatively contraindicated in the presence of deranged liver function, which occurs in a large percentage of thyrotoxic patients.

The patient should be heavily sedated the evening prior to operation and an intravenous infusion begun. Operation should be scheduled for the early morning and the patient brought to the operating room in his own bed rather than aransed for transfer to a stretcher. He is anesthetized in a quiet room prior to being moved to the operating table. Thiopental (Pentothal) is the induction agent of choice. The thiobarbitalates possess antithyroid activity, presumably related to their thiocarbanate structure (N-C=S). In animals given a single large dose of thiopental (40 mg/kg) impairment of thyroid activity is virtually instantaneous and persists 6-7 days. It may be that thiourea or substituted thioureas formed by the metabolism of thiopental are ultimately responsible for the prolonged effect.34

Inhalation agents that stimulate the sympathoadrenal axis, e.g., diethyl ether and cyclopropane, are contraindicated, as is hypercarbia for the same reason. Methoxyflurane (Penthane) has a depressant effect on the sympathoadrenal system, as evidenced by the decreased content of epinephrine and norepinephrine in adrenal venous blood of animals anesthetized with this agent,33 and has been recommended as the anesthetic of choice.44 Methoxyflurane, however, like other potent halogenated anesthetics, has been shown to exert a direct negative inotropic effect upon the myocardium,34 and must therefore be used with caution should beta-blocking agents be required. The combination may precipitate congestive heart failure. If it is anticipated that the surgical procedure will be prolonged, another agent may be preferable, as nephrotoxicity induced by methoxyflurane has been shown to be time- and dose-related.56,62 In addition, the increase in protein synthesis together with the hypermetabolic state induced by the thyroid hormones might conceivably result in an increased rate of biotransformation
of the anesthetic, thereby enhancing its potential for toxicity.

Halothane (Fluothane), with or without nitrous oxide, may be used as the primary anesthetic. Hyperthyroidism enhances the hepatotoxicity of carbon tetrachloride in experimental animals.\textsuperscript{13} Although the mechanism of halothane-related hepatotoxicity (if indeed such an entity exists) appears to be different from that of carbon tetrachloride,\textsuperscript{20} the incidence and severity of hepatic pathology associated with thyrotoxicosis could perhaps be modified by halogenated hydrocarbon anesthetics. Like diethyl ether, halothane increases serum thyroxine levels, while methoxyflurane and thiopental do not alter the hormone titer.\textsuperscript{59}

A balanced technique using nitrous oxide, thiopental, and a muscle relaxant may be used. The patient should be anesthetized deeply enough so that surgical stimuli do not provoke an outpouring of catecholamines in the event that preoperative adrenergic blockade is incomplete. Thus intravenous supplementation may be necessary.

Spinal anesthesia was used as a therapeutic modality for severe thyrotoxicosis in the 1940’s.\textsuperscript{46} The spinal anesthetic was not administered to provide anesthesia for the surgical procedure. Rather, its purpose was blockade of the sympathetic innervation of the adrenal medulla, thereby reducing the secretion of epinephrine. Following establishment of a T4–5 sensory block, general anesthesia was induced. It has been demonstrated that TSH levels in human plasma do not change appreciably during spinal, diethyl ether, halothane, or methoxyflurane anesthesia.\textsuperscript{59}

Whatever the anesthetic sequence, the patient must be carefully monitored. In addition to precordial or esophageal stethoscope and blood pressure cuff, an electrocardiogram and temperature monitor are mandatory. A cooling blanket, refrigerated intravenous solutions, and ice should be readily accessible. Steroids, injectable iodine, alpha- and beta-adrenergic blocking agents, and chlorpromazine should be at hand.

Thyroid Storm

Monitoring must not cease with termination of the operation. Thyroid storm, when related to surgery, usually develops within 6–18 hours postoperatively. It is characterized by hyperpyrexia, marked tachycardia, and susceptibility to severe hypotension. Before the introduction of antithyroid drugs and adrenergic blocking agents in the preparation of patients for thyroidectomy, storm occurring in the early postoperative period (surgical storm) was the most common thyrotoxic crisis. The so-called “medical storm” may be initiated by infection, trauma, vigorous palpation of a toxic goiter,\textsuperscript{43} or iodine withdrawal.\textsuperscript{53} Respiratory tract infection, particularly bronchopneumonia, is a frequent precipitating factor.\textsuperscript{53,54} The reported incidence of storm in patients hospitalized for thyrotoxicosis is 2–8 per cent.\textsuperscript{53} It is felt by some that thyroid storm is not a complication of the disease, but is rather an intrinsic feature of hyperthyroidism in its most severe form. Thus, thyroid storm may also be called “decompensated thyrotoxicosis.”\textsuperscript{53}

Untreated or inadequately treated thyroid storm is associated with a remarkably high death rate. Aggressive treatment with antithyroid drugs, iodine, steroids, and adrenergic blocking agents must be instituted immediately. It has been suggested that the level of consciousness and state of mentation of the patient in crisis are reliable prognostic signs. Clearing of mental aberrations may be a guide to therapeutic progress. For this reason, guanethidine, which does not pass the blood–brain barrier and thus does not alter consciousness or behavior, may be preferable to reserpine.\textsuperscript{53} Supportive therapy includes increased inspired oxygen concentration, measures to lower body temperature, adequate fluid replacement, and ample caloric intake.

The duration of thyroid storm varies from one to several days, averaging 3 days.\textsuperscript{53} Typically, improvement begins within 12 hours of institution of therapy. It is difficult to obtain an accurate estimate of expected mortality in thyroid storm. With iodine as the only specific therapy, mortality approaches 60–70 per cent. The death rate is probably about 25 per cent when steroids and alpha-adrenergic blocking agents are added to the therapeutic regime.\textsuperscript{40,53} Propranolol may produce dramatic improvement in some patients and will undoubtedly markedly alter the mortality rate. Plasmapheresis has been used success-
fully in patients who failed to respond to aggressive therapy with conventional agents. The thyrotoxic patient undergoing emergency surgery requires expert management if thyroid storm is to be averted. Recognition of the endocrinopathy may be difficult, as acute pathology or trauma may mask hyperthyroidism. The patient may be unaware of its existence or incapable of transmitting the information. Since it is not possible to deplete catecholamine stores acutely, adrenergic blocking agents should be employed. Propranolol is used to bring the heart rate into the near-normal range. Phentolamine (Regitine) may be used to reduce peripheral resistance if hypertension is a significant problem. Sodium iodide and large doses of steroids should be given intravenously. Principles of anesthetic management and monitoring are the same as discussed previously.

Complications of Surgery in the Hyperthyroid Patient

The complications of surgery in the hyperthyroid patient include all of the potential complications of thyroid surgery in the euthyroid patient. Severe hypotension with marked bradycardia or asystole may occur with manipulation of the carotid sinus. Infiltration of the area around the bifurcation of the common carotid artery with a local anesthetic may successfully block the carotid sinus reflex. Intravenous administration of atropine is also useful.

Venous air embolism is uncommon, but if a large vein is opened, it is possible for as much as 200 ml of air to be aspirated during a deep inspiration. Injury to the phrenic or cervical sympathetic nerves, pneumomediastinum, pneumothorax, or tracheal laceration may occur. Postoperatively the airway may be compromised by hematoma, tracheal malacia, or laryngeal edema. Unilateral recurrent laryngeal nerve injury is usually tolerated; however, damage to both nerves may necessitate tracheotomy. Injury of the superior laryngeal nerve results in loss of sensation in the larynx and piniform sinus. Aspiration is often associated with this loss of sensory innervation.

Permanent hypoparathyroidism is not common when surgical dissection is meticulous. Transient hypoparathyroidism is more common and is usually manifest 24–72 hours after operation. The intrinsic muscles of the larynx are exquisitely sensitive to calcium deficiency. Inspiratory stridor occurs long before overt tetany and results in partial or complete laryngospasm. All manifestations of hypoparathyroidism, including upper airway obstruction, will usually abate following intravenous administration of calcium chloride or calcium gluconate.

It is desirable to observe the postthyroidectomy patient in an intensive care facility for 24–72 hours postoperatively.

Summary

Anesthetic management of the hyperthyroid patient who cannot be rendered euthyroid prior to operation is based on an understanding of the pathophysiology of the disease, including thyro-adrenal relationships. Antithyroid drugs, adrenergic blocking agents, iodine, and corticosteroids are essential pharmacotherapeutic agents. The patient must be carefully monitored and mechanical devices and drugs for treating thyroid storm must be readily available before anesthesia for any such patient is undertaken. With careful attention to all these details, the anesthesiologist should be able to provide safe and pleasant anesthesia for the hyperthyroid patient.

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