Lesson 313: Preanesthetic Assessment of the Patient With Hyperthyroidism

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Read this article, reflect on the information presented, then go online and complete the lesson post-test and course evaluation before the termination date below. (CME credit is not valid past this date.) You must achieve a score of 80% or better to earn CME credit.

TIME TO COMPLETE ACTIVITY: 2 hours
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Professional Gaps

Although hyperthyroidism is not uncommon, patients who come to the operating room often are medicated and euthyroid. Thus, the anesthetic care provider may not be aware of potential complications that can be caused by the disease.

Learning Objectives

At the completion of the activity, the reader will be able to:

1. Define hyperthyroidism and Graves’ disease.
2. Describe the special circumstances associated with the administration of anesthesia to hyperthyroid patients.
3. List the manifestations of hyperthyroidism and Graves’ disease.
4. Explain the pathophysiology of hyperthyroidism and Graves’ disease.
5. Determine appropriate preoperative assessment for a patient with hyperthyroidism and Graves’ disease.
6. Interpret the results of laboratory tests.
7. Formulate a plan for anesthesia and postoperative analgesia in a patient with hyperthyroidism and Graves’ disease.
8. Review treatment options for the hyperthyroid patient.
9. State the incidence and prevalence of hyperthyroidism and Graves’ disease.
10. Predict, recognize, and respond to common perioperative complications of hyperthyroid patients.
Case

A 36-year-old woman (height 157 cm, weight 55 kg), 4 weeks into treatment for hyperthyroidism, presented to the emergency room with acute appendicitis. She had a history of claustrophobia and a fear of needles. Her vital signs were within the normal range except her temperature was 99.1°F. On physical examination, she had exophthalmos but there was no evidence of enlarged goiter and her airway appeared normal (Mallampati I). White blood cell count was 12.1 billion/L and hemoglobin was 10.2 g/dL.

The Thyroid Gland

Hyperthyroidism affects about 1.3% of the population and the female-to-male ratio is 5:1. The incidence of Graves’ disease, the most common form of hyperthyroidism, is 4.6 per 1,000 in women. The thyroid gland is found in the anterior neck below the thyroid cartilage and around the trachea. It consists of 2 lateral lobes connected by a thin isthmus. In normal physiology, the thyroid gland manufactures the hormones triiodothyronine (T3) and thyroxine (T4) from a thyroglobulin precursor in the thyroid follicular cells. Both hormones are reversibly bound to circulating plasma proteins for transport to peripheral tissues. T3 binds to receptors in target cells with 10-fold greater affinity than T4, and is proportionately more active. Both hormones function to increase carbohydrate and fat metabolism; increase metabolic rate, minute ventilation, heart rate, and contractility; affect water and electrolyte balance; and potentiate normal activity of the central nervous system (CNS).

Pathophysiology

Many different disorders can cause hyperthyroidism. Risk factors include female sex, smoking, advanced age, and radiation. Genetic factors also play a role. The general clinical manifestations of hyperthyroidism do not specify the underlying cause, but unique problems unrelated to high levels of hormone can be seen in some diseases. The prevalent causes of hyperthyroidism with key clinical features are listed in Table 1. Other major signs and symptoms of hyperthyroidism/thyrotoxicosis that are found in different organ systems are discussed below.

Skin

The skin tends to be soft, warm, and flushed due to increased blood flow. Excessive sweating is common due to increased calorigenesis, and is associated with heat intolerance. Thinning of the hair and skin is also common, occasionally with pruritus and hives. Thyroid dermopathy occurs in less than 5%, most frequently over the anterior and lateral aspects of the lower leg; hence the term pretibial myxedema. Typical lesions are raised, hyperpigmented papules on the skin overlying the shins and are seen only in Graves’ disease.

Eyes

Sympathetic overstimulation, mediated by increased adrenergic receptors in some tissues, results in a wide, staring gaze and lid lag in patients with hyperthyroidism. Only Graves’ disease results in ophthalmopathy and exophthalmos, which is the result of the accumulation of hydrophilic glycosaminoglycans and inflammation of the extraocular muscles, orbital fat, and connective tissue.
Cardiovascular

The cardiovascular effects of hyperthyroidism often are profound and clinically relevant to anesthesiologists. Over time, thyroid hormone increases cardiac output, contractility, blood pressure, systemic vascular resistance, and myocardial oxygen consumption and causes rhythm disturbances. Initially, resistance in peripheral arterioles is decreased through interaction of thyroid hormone with vascular smooth muscle, resulting in decreased mean arterial pressure. As a result, with mean arterial blood pressure lowered, the renin–angiotensin–aldosterone system is activated and renal sodium absorption increases. Ultimately, blood volume and preload increases. These changes can increase cardiac output 50% to 300%. Increased cardiac output, heart rate, and contractility contribute to systolic hypertension. Sinus tachycardia, supraventricular tachycardia, and atrial fibrillation are common rhythm disturbances. Atrial fibrillation occurs in 10% to 20% of patients and is most frequent in patients older than 50 years. Tachycardia may unmask previously asymptomatic coronary artery disease by increasing oxygen demand.8,9

Metabolic/Endocrine

Patients with hyperthyroidism often have low serum total high-density lipoprotein and low-density lipoprotein, as well as lower total serum cortisol concentrations.10,11 This does not affect monitored anesthesia care requirements. Thyroid hormone also can interfere with glucose metabolism by acting as an insulin antagonist in the periphery, resulting in hyperglycemia.12

Respiratory

From the mass effect of a large goiter obstructing the trachea to increased oxygen consumption, hyperthyroidism can inflict many problems on respiratory function. Dyspnea and dyspnea on exertion
are common, with the underlying cause ranging from those listed above as well as increased pulmonary arterial pressure, respiratory muscle weakness and decreased lung volume, and exacerbation of asthma, among others.\textsuperscript{13,14}

\textit{Gastrointestinal}

Gut stimulation can result in hypermotility, malabsorption, diarrhea, and subsequent weight loss despite an increased appetite. Other problems that may be seen later include dysphagia due to goiter, vomiting, and abdominal pain.\textsuperscript{15}

\textit{Hematologic}

Plasma volume is increased with an associated, but not equal, increase in red blood cell mass that culminates in a normochromic, normocytic anemia.\textsuperscript{16}

\textit{Genitourinary}

Urinary frequency and nocturia are common complaints related to hyperthyroidism-mediated voiding dysfunction.\textsuperscript{17} Women experience low serum free estradiol as a consequence of high sex hormone-binding globulin (SHBG) concentrations, as well as high luteinizing hormone. These changes can produce oligomenorrhea, anovulatory infertility, and amenorrhea.\textsuperscript{18} Increased SHBG results in low serum free testosterone in men, but increased conversion of testosterone to estradiol. As a result, men may experience gynecomastia, erectile dysfunction, and reduced libido.\textsuperscript{19}

\textit{Bone}

Bone density is decreased by stimulating bone resorption. Elevated alkaline phosphatase and osteocalcin concentrations suggest increased bone turnover. Patients with chronic hyperthyroidism thus experience osteoporosis with an increased risk for fracture.\textsuperscript{20}

\textit{Neurologic}

Behavioral and personality changes such as nervousness, agitation, insomnia, and depression are frequently experienced. Cognitive impairments such as deficits in concentration, confusion, and amnesia are frequently reported. Tremor and proximal muscle weakness (thyroid myopathy) develop in nearly 50% of patients.\textsuperscript{21}

\textit{Diagnosis}

Combinations of the symptoms cited here may indicate hyperthyroid disease on physical examination. Hyperactivity, rapid speech, and staring gaze combined with tachycardia and tremor, for example, are highly suggestive of hyperthyroidism.\textsuperscript{22} Findings specific to Graves’ disease include exophthalmos, periorbital and conjunctival edema, limitation of eye movement, and infiltrative dermopathy (pretibial myxedema).\textsuperscript{5,7}

Thyroid enlargement (goiter) may or may not be readily apparent, depending on the cause of disease. Patients with Graves’ disease or toxic multinodular goiter experience thyroid enlargement ranging in size from minimal to massive. However, patients with painless thyroiditis may experience no thyroid
enlargement whatsoever, which should alert the physician to the possibility of exogenous hyperthyroidism or struma ovarii. A solitary nodule is characteristic of a toxic thyroid adenoma. Lastly, the thyroid is painful and tender to palpation in subacute thyroiditis.

The next step in the diagnosis of hyperthyroidism is to obtain thyroid function tests (TFTs). All patients with primary hyperthyroidism display a low thyroid-stimulating hormone (TSH), and a TSH assay remains the best method of detecting thyroid dysfunction at the cellular level. Minute changes in thyroid function status can induce drastic changes in TSH levels. Normal TSH levels range from 0.4 to 5.0 mU/L; a TSH level of 0.1 to 0.4 mU/L with normal free T3 (FT3) and free T4 (FT4) signifies subclinical hyperthyroidism, and a TSH level less than 0.03 mU/L is defined as overt hyperthyroidism. Furthermore, patients with overt hyperthyroidism usually demonstrate high FT4 and T3 concentrations. It is not uncommon, however, for a patient to present with only T3 or FT4 elevated. Subclinical hyperthyroidism is notable for a below-normal TSH accompanied by normal serum FT4, T3, and FT3.

Based on a physical examination positive for conspicuous symptoms as well as abnormal TFTs, a differential diagnosis can be established. Primary hyperthyroidism is suspected in all patients with low serum TSH and high FT4 and T3. Graves’ disease and nodular goiter patients typically have increases in T3 greater than T4 due to increased peripheral conversion of T4 to T3. Conversely, patients with amiodarone-induced thyroiditis present with high serum T4 and normal T3 as this drug inhibits the peripheral conversion of T4 to T3. A patient with a TSH-secreting pituitary adenoma, a very rare cause of hyperthyroidism, has normal to high serum TSH despite high FT4 and T3. Table 2 summarizes the presumptive diagnoses based on TSH and TFT status.

**Table 2. Thyroid Assessment Based on Functional Status Tests**

<table>
<thead>
<tr>
<th>Serum TSH</th>
<th>Serum Free T4</th>
<th>Serum T3</th>
<th>Assessment</th>
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<tr>
<td>Unremarkable hypothalamic-pituitary function</td>
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<td>Low</td>
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<td>Euthyroid: T3 therapy</td>
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<tr>
<td>Normal</td>
<td>Low normal or low</td>
<td>Normal or high</td>
<td>Euthyroid: thyroid extract therapy</td>
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<td>Low</td>
<td>Normal</td>
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<td>Hyperthyroidism</td>
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<tr>
<td>Low</td>
<td>Normal</td>
<td>Normal</td>
<td>Subclinical hypothyroidism</td>
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| Abnormal hypothalamic-pituitary function |
| Normal or high | High | High | TSH-mediated hyperthyroidism |
| Normal or low | Low or low-normal | Low or normal | Central hypothyroidism |

T3, triiodothyronine; TSH, thyroid-stimulating hormone; FT3, free T3; FT4, free T4. 

*In central hypothyroidism, serum TSH may be low, normal, or slightly high.
Table modified from reference 40.
Pregnant women also may experience hyperthyroidism. The diagnosis of hyperthyroidism in pregnancy parallels that of other hyperthyroid cases. Complications to the pregnancy itself are of concern, and may include the potential for spontaneous abortion, low birthweight, stillbirth, pre-eclampsia, and, in rare cases, heart failure. (The most common causes of hyperthyroidism in pregnancy are human chorionic gonadotropin [hCG]-mediated hyperthyroidism [1%-3% of pregnancies] and Graves’ disease [0.1%-1% of pregnancies]). The levels of serum hCG in a normal pregnancy rise soon after fertilization. TSH and hCG share significant homology, as the $\alpha$-subunit of the former is almost identical to hCG. Prolonged high concentrations of hCG can cause a (usually) transient gestational hyperthyroidism. It is estimated that up to 20% of pregnant women develop subclinical hyperthyroidism not requiring therapy.29,30

After diagnosis of hyperthyroidism has been made, the next step is to determine the underlying cause. The etiology of the disease can be distinguished using 24-hour radioiodine uptake31 and divided into 2 mechanisms: hyperthyroidism with normal to increased radioiodine uptake suggests overproduction of hormone, whereas a near-absent radioiodine uptake indicates either inflammation and destruction of the thyroid with subsequent release of hormone systemically or an extrathyroidal source of thyroid hormone.32 The causes of hyperthyroidism are listed in Table 3 by radioiodine uptake status.

### Treatment

The treatment of primary hyperthyroidism involves symptomatic relief (Beta-blockers are the mainstay) and therapy to decrease the synthesis of new thyroid hormone,6,33 which requires thionamide therapy (methimazole or propylthiouracil [PTU]), radio-iodine ablation, or surgery.34

The following scenarios are indications for surgery:

- Very large goiters that cause upper airway obstruction or dysphagia, or that would require radioiodine therapy.
- Nonfunctional thyroid nodule suspected as malignant.
- Patients with hyperparathyroidism in addition to hyperthyroidism.
- Pregnant women with allergies to antithyroid drugs and severe hyperthyroidism.
- When other modalities have failed.

The most feared complication associated with thyroid surgery is a rare, life-threatening condition called thyroid storm. Other potential complications include bleeding, nerve
damage, and airway obstruction. Although thyroid storm can develop in patients with chronic hyperthyroid, it is most often precipitated by an acute event such as thyroid or nonthyroidal surgery, trauma, or infection. The incidence has been shown to be 0.20 per 100,000 in hospitalized patients per year. Mortality is substantial, ranging from 10% to 30%. Findings, although rarely identified under general anesthesia, classically involve an exaggeration of the typical symptoms of hyperthyroidism, including tachycardia, hyperpyrexia, CNS dysfunction, and gastrointestinal distress, as well as tremor, warm and diaphoretic skin, and lid lag. Treatment of thyroid storm involves high-dose Beta-blockers, thionamides, iodine solution to block the thyroid hormone release, and glucocorticoids to reduce peripheral conversion of T4 to T3 and maintain vasomotor stability.35-37

Anesthetic Considerations

Preoperative testing relevant to anesthetic care depends in part on the presentation but should include a complete blood count (looking for anemia), blood sugar levels, and radiologic studies of the neck. If there is a large goiter and bleeding may be an issue, a type and screen is indicated.

With the advent of effective drugs such as methimazole and PTU, patients with hyperthyroidism undergoing elective surgery are nearly always euthyroid before the procedure.36 A 6- to 8-week course of either PTU or methimazole, if dosed correctly, will stabilize and normalize the T4 levels sufficiently.37 The surgeon should allow enough time preoperatively for these drugs to become effective. Both medications stall the synthesis of T4 and T3 within the thyroid gland itself. PTU has the added benefit of inhibiting the conversion of T4 to T3 in peripheral tissues by specifically inhibiting deiodinases within various cells. PTU is able to decrease the levels of the inactive metabolite, T4, as well as the active hormone, T3.36

If the patient is unable to undergo the full preoperative regimen of PTU or methimazole, protocols are designed to stabilize them more rapidly in emergent cases. An IV Beta-blocker, most commonly esmolol, in a bolus dose of 10 to 50 mg (250-500 mcg/kg undiluted) or in an infusion dose of 50 to 300 mcg/kg/min (2.5 g in 250 mL 0.9% NaCl or 10 mg/mL), is administered along with ipodate, glucocorticoids and oral or rectal PTU. To date, PTU does not have a form compatible with IV delivery. Esmolol will not affect the underlying pathology of hyperthyroidism but rather will act to normalize the heart rate and relieve many symptoms such as sweating and general anxiety. The β-blocker propranolol has tertiary effects similar to those of PTU and is able to obstruct the deiodinases converting T4 to T3. Dexamethasone, administered at a dose of 2 mg every 6 hours, is generally the glucocorticoid of choice, both inhibiting the peripheral conversion of T4 to T3 as well as decreasing thyroid hormone release directly. Lastly, ipodate acts to bind thyroid hormone receptors and can block these receptors from activation by free circulating thyroid hormones. This antagonistic effect can be substantial in an emergency setting; long-term use is associated with increased thyroid hormone stores and acts to possibly exacerbate the hyperthyroid state.

Commonly, patients with hyperthyroidism present with a substantial goiter that compresses and/or deviates the underlying trachea. Any patient demonstrating substantial goiter should receive a computed tomography scan as part of the preoperative exam to determine the tracheal significance. Flow volume loops are rarely helpful in a routine setting; however, they may help with identifying airway compromise in patients with tracheal compression. Once the extent of the compression is evaluated, the intubation should proceed analogous to any other patient with a compromised airway, including the use of advanced airway devices. A reinforced endotracheal tube should be passed
beyond the point of compression, and preoperative sedation should be minimized. Awake intubation may be the preferred technique.\textsuperscript{38} It should be noted that a surgical airway may be extremely challenging in these patients.

Induction of anesthesia was typically accomplished with the use of thiopental, which, owing to its thioureylene nucleus, decreases the peripheral conversion of T4 to T3 as an added benefit. Because thiopental is currently not available, propofol is the induction agent of choice. Likewise, drugs that have properties that stimulate the sympathetic nervous system should be avoided. These include but are not limited to ketamine, pancuronium, atropine and epinephrine.\textsuperscript{37} Because these patients have a general increase in sympathetic activity as well as sympathetic sensitivity, it is best to avoid any medication that could trigger these receptors. In this regard, dexmedetomidine may be useful.

As of now, virtually all anesthesia maintenance drugs and techniques have demonstrated safety and efficacy in the patient with hyperthyroidism. As stated earlier, some investigators propose to avoid anticholinergic drugs and their sympathomimetic effects (tachycardia, interfering with sweating). In fact, despite increased metabolic demands, studies on hyperthyroid animals did not validate a need for an increased anesthetic dose.\textsuperscript{37} By the same token, the medically induced euthyroid patient has no change in monitored anesthesia care requirement. Studies have shown a significant increase in the hepatotoxic side effects of isoflurane when used on hyperthyroid rats, arguably due to increased metabolic abilities.\textsuperscript{37} However, the dearth of data concerning these outcomes on human patients has left these claims unsubstantiated. At present, there are no studies that demonstrate any adverse effects from desflurane or sevoflurane. It should be noted, however, that all inhalational agents can cause sympathetic hyperactivity.

As noted here, the complication most feared during surgery on a patient with hyperthyroidism is thyroid storm. Currently, the mortality rate of patients experiencing thyroid storm is estimated to be as high as 20%.\textsuperscript{37} What makes the diagnosis and treatment so difficult is that the syndrome manifests much like malignant hyperthermia. Clinicians may test the T4 levels in hopes of differentiating the 2 syndromes without realizing that patients with thyroid storm may actually not have significantly elevated levels of thyroid hormones compared with their usual hyperthyroid state. In fact, there is no laboratory test currently available capable of definitively diagnosing thyroid storm.

Treatment is similar to a patient with hyperthyroidism in the emergent setting.\textsuperscript{39} The most common drug regimen is the use of IV Beta-blockers to ideally decrease the heart rate to 90 bpm, IV dexamethasone at 2 mg every 6 hours, and oral PTU of 200 to 400 mg every 8 hours.\textsuperscript{37} Additionally, cooling blankets and ice packs may be used to combat hyperthermia, and IV fluids should be used as needed to combat inevitable dehydration and electrolyte imbalances.\textsuperscript{39}

**Management of Case Presentation**

The patient’s endocrinologist was in the emergency room when the anesthesia team arrived. After discussion of anesthetic options, the patient agreed to general anesthesia. PTU 400 mg was given immediately and continued every 8 hours throughout her hospital stay. EMLA cream (lidocaine 2.5% and prilocaine 2.5%) was used before venous cannulation. In the operating room, routine monitors were placed. Rapid sequence induction included propofol, lidocaine, fentanyl, and succinylcholine. Particular attention was paid to ensure that lubrication was placed in her eyes and the lids were closed with paper tape. Her heart rate was 96 bpm and esmolol, in 10 mg doses, was titrated to maintain a rate under 90 bpm. Dexamethasone, 2 mg IV, was given before induction and every 6 hours
postprocedure. Cooling blankets were placed intraoperatively. She remained afebrile. Laparoscopic surgery was uneventful. The patient was discharged home on day 3 without issues.

**Conclusion**

Hyperthyroidism and Graves’ disease are disorders that anesthesiologists may encounter. Knowledge of the challenges—such as thyroid storm and cardiovascular or respiratory dysfunction—and how to manage them are crucial for proper care. If at all possible, patients with hyperthyroidism should be made euthyroid preoperatively via PTU or methimazole. Perioperatively, anticholinergic and sympathomimetic drugs such as atropine and epinephrine should be avoided. Induction of anesthesia may be implemented via propofol, and maintenance can be achieved with any of the potent inhalational agents.

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REFERENCES


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**Post-test**

1. **All of the following are manifestations of Graves’ disease, except _____**.
   - a. pretibial myxedema
   - b. exophthalmos
   - c. goiter
   - d. anorexia

2. **_____ is the least appropriate agent for induction of anesthesia in the hyperthyroid patient**.
   - a. Dexmedetomidine
   - b. Ketamine
   - c. Etomidate
   - d. Propofol

3. **What is the most common age and sex associated with hyperthyroidism?**
   - a. Elderly man
   - b. Adult woman
   - c. Adolescent boy
   - d. Adolescent girl

4. **Overt hyperthyroidism can be diagnosed with a thyroid-stimulating hormone level of _____**.
   - a. >5.0 mU/L
   - b. 0.4-5.0 mU/L
   - c. 0.1-0.4 mU/L
   - d. <0.03 mU/L

5. **The incidence of hyperthyroidism in women is _____**.
   - a. 53/100
   - b. 4.6/100
   - c. 1.8/10,000
   - d. 10.3/5,000
6. The euthyroid state can be achieved in hyperthyroid patients by 6 to 8 weeks of therapy with _____.
   a. propylthiouracil  
   b. methimazole  
   c. glucocorticoids  
   d. A or B

7. The most feared perioperative complication with hyperthyroid patients is _____.
   a. renal failure  
   b. hepatotoxicity  
   c. thyroid storm  
   d. aneurysm

8. Which agent(s) should be avoided with respect to the hyperthyroid patient?
   a. Iodine  
   b. Atropine  
   c. Epinephrine  
   d. B and C

9. Treatment of thyroid storm should begin with _____.
   a. glucocorticoids  
   b. propylthiouracil  
   c. high-dose IV Beta-blockers  
   d. opioids

10. Hyperthyroid patients commonly experience _____.
    a. osteoporosis  
    b. constipation  
    c. autosplenectomy  
    d. cold intolerance