An intimate knowledge of the anatomy, physiology, pathophysiology, pharmacology, and specific issues related to anesthesia case management for thyroidectomy is essential to provide high-quality care. Airway management may be difficult despite a normal airway examination due to impingement of a thyroid mass on the laryngeal and tracheal structures. Anesthetists must be prepared to use emergency airway adjuncts in case a patient cannot be ventilated or intubated. Because sympathetic nervous system hyperactivity is associated with increased amounts of thyroid hormone, it is essential that all patients having an elective thyroidectomy be in a euthyroid state before surgery. There are multiple preoperative antithyroid medication regimens that effectively treat thyroid hormone hypersecretion. However, although a rare event, thyroid storm can still occur during the perioperative period. Anesthetic considerations and surgical complications are presented.

Keywords: Antithyroid medications, nerve integrity monitor endotracheal tube, recurrent laryngeal nerve injury, thyroidectomy, thyroid storm.

Objectives
At the completion of this course the reader should be able to:
1. Explain the signs and symptoms that result from recurrent laryngeal nerve injury and the emergency interventions necessitated by such injury.
2. Discuss the proper placement of a nerve integrity monitor endotracheal tube.
3. List the most common causes of hyperthyroidism.
4. Identify a preoperative antithyroid drug regimen for patients with hyperthyroidism.
5. Describe the signs, symptoms, and treatment of thyroid storm.

Introduction
The anatomic and physiologic effects associated with hyperthyroidism can cause end-organ damage, hyperdynamic effects causing extreme cardiovascular lability, and tracheal distortion causing difficult airway management. Creating a euthyroid state by administering a preoperative antithyroid drug regimen is essential. A thorough knowledge of the pathophysiology and potential complications associated with thyroidectomy is necessary for competent patient care. A nerve integrity monitor (NIM) endotracheal tube (Medtronic Xomed, Jacksonville, Florida) may be inserted to help the surgeon assess the potential for recurrent laryngeal nerve (RLN) injury.

Anatomy and Physiology Associated With Thyroid Gland Structure and Function
The thyroid gland is butterfly-shaped and composed of 2 lobes that are connected by a median tissue mass named the thyroid isthmus. It is located on the anterior and anterolateral aspect of the trachea immediately inferior to the larynx. The thyroid gland is the largest endocrine gland in the body, weighing 20 g in a healthy adult. Its major blood supply arises from the superior thyroid arteries, which originate from the external carotid artery and inferior thyroid arteries, a branch of the thyrocervical trunk. Blood flow is approximately 5 times the weight of the gland, and, therefore, the thyroid receives...
one of the greatest blood supplies per gram of tissue in the body. Venous drainage from the thyroid gland passes into the superior thyroid veins to the internal jugular veins and the inferior thyroid veins into the left brachiocephalic vein.

The primary motor function associated with movement of the intrinsic muscles of the larynx and vocal cords is supplied by the RLN. The RLN controls abduction and adduction of the vocal cords via the posterior cricoarytenoid muscles and the lateral cricoarytenoid muscles, respectively. The external branch of the superior laryngeal nerve innervates the pharyngeal constrictors and the cricothyroid muscle, which enhances vocal cord tension. These nerves lie in proximity to the lateral lobes of the thyroid gland. During surgical resection, if these nerves are temporarily or permanently damaged, vocal cord movement can be adversely affected, which may result in airway compromise after extubation. Anatomic representation of the thyroid gland and its associated structures is shown in Figure 1. A more thorough explanation of the results associated with RLN damage is given later in this course.

The thyroid gland is composed of spherical structures called follicles. The walls of the follicles are formed by cuboidal epithelial cells that produce the glycoprotein thyroglobulin, which is essential for thyroid hormone synthesis. Colloid is stored in the central cavity of the follicle, which is where the thyroglobulin is created (Figure 2). The creation of thyroid hormone is a complex process.

- **Thyroid Hormone.** Thyroid hormone, which has a dramatic effect on increasing the basal metabolic rate, consists of thyroxine (T₄) and triiodothyronine (T₃). The major hormone secreted by the thyroid gland is T₄, whereas most of the T₃ is formed by the conversion of T₄ to T₃ at the target tissues. Despite the fact that the amounts of T₄ and T₃ in circulation are 90% and 10%, respectively, the high potency of T₃ results in production of 80% of the metabolic activity.
• **Synthesis.** After iodide is ingested and absorbed by the gastrointestinal tract into the blood, thyroid hormone synthesis begins when iodide is actively transported from the plasma into the colloid of the follicle. Once inside the follicle, iodide is converted to iodine by thyroid peroxidase. This process is known as **iodide trapping**.

The organification of thyroglobulin occurs when iodine molecules attach to the amino acid tyrosine on the thyroglobulin molecule. Thyroxine is formed when 2 diiodotyrosine compounds are linked, and T₃ is composed of diiodotyrosine and monoiodotyrosine (Figure 3).

• **Transport.** When released, the majority of T₄ and T₃ are immediately bound to thyroxine-binding globulins and albumin that are produced by the liver. Both hormones bind to target tissue receptors; however, T₃ has a higher affinity for binding and is 10 times more metabolically active than T₄. Despite the high degree of protein binding, it is the unbound or free T₃ that provides the majority of the metabolic effects. Most peripheral tissues contain the enzymes needed to convert T₄ to T₃; however, the majority of deiodination occurs in the liver and kidneys.

• **Regulation.** Thyrotropin-releasing hormone is secreted by the hypothalamus, which stimulates the production of thyroid-stimulating hormone (TSH) by the anterior pituitary gland. Thyroid-stimulating hormone is transported to the thyroid gland, which stimulates the production of T₄ and T₃ by the thyroid gland. The homeostatic regulation of thyroid hormone production is maintained by a negative feedback loop. When normal or increased amounts of thyroid hormone are present, T₄ and T₃ primarily inhibit further TSH secretion from the anterior pituitary and secondarily by decreasing thyrotropin-releasing hormone formation and release from the hypothalamus (Figure 4).

• **Physiologic Effects.** It is likely that all cells in the body are targets for thyroid hormones. Although not strictly necessary for life, thyroid hormones have profound effects on many physiologic processes such as development, growth, and metabolism.

**Pathophysiology**
Thyroid dysfunction can be treated medically or surgically. A thyroidectomy is performed as definitive treatment for thyrotoxicosis and for malignancy. Thyrotoxicosis is a condition in which excessive amounts of thyroid hormone are present in a patient's system; hyperthyroidism (the most
common cause of thyrotoxicosis) describes states in which the excessive thyroid levels are due to a hyperactive thyroid gland.\(^8,9\) When hyperthyroidism occurs, the follicular cells of the thyroid produce 5 to 15 times the normal amount of thyroid hormone.\(^2\) Hyperthyroidism occurs in 1% to 2% of women and in approximately 0.1% to 0.2% of men.\(^9\) Hyperthyroidism is most frequently caused by 1 of 3 pathologic processes: Graves disease, toxic multinodular goiter, and toxic adenoma.

Thyroid hormone has an essential role in metabolism; therefore, the signs and symptoms of thyrotoxicosis reflect a generalized hypermetabolism (Table 1).\(^9\) A hyperfunctioning thyroid gland may enlarge to 2 to 3 times the normal size and is termed a toxic goiter.\(^2\) Another type of goiter is commonly due to deficient thyroid hormone synthesis or iodine deficiency. Nontoxic (or simple) goiters are usually treated medically, and surgical resection is reserved for cases in which the goiter produces compression of the trachea or major vessels or is unre sponsive to medical treatment.\(^10\)

Toxic multinodular goiter is a condition in which multiple nodules within the thyroid gland assume functional autonomy and secrete thyroid hormones independent of the action of TSH. The cause of autonomy in toxic multinodular goiter is not well understood.\(^9\)

Toxic adenoma (also called Plummer disease and hyperfunctioning solitary nodule) is the result of a single thyroid nodule assuming functional autonomy. Toxic adenoma does not usually manifest with clinical features typical of other causes of thyrotoxicosis. The autonomy of a toxic adenoma has been linked to a mutation that leads to altered function of the TSH receptor pathway.\(^11\)

Graves disease accounts for 68% to 80% of cases and is the most common cause of thyrotoxicosis.\(^11\) This pathologic process is most common among women between the ages of 20 and 40 years. Graves disease is the result of an autoimmune process in which autoantibodies stimulate TSH receptors to produce excessive amounts of thyroid hormones.\(^10\)

An unusual exacerbation of thyrotoxicosis, thyrotoxic crisis (thyroid storm), manifests as severe hypermetabolism with signs such as fever, tachycardia, ectopy, and delirium.\(^11\) Thyrotoxicosis can be precipitated by infection, surgery, and trauma and is more often associated with Graves disease than with toxic multinodular goiter or toxic adenoma.\(^9\)

Malignancies of the thyroid gland are the most common malignancies of the endocrine system and have been associated with exposure to radiation.\(^9,11\) Thyrotoxicosis is a rare occurrence (2%) in thyroid malignancies. Surgery is the principal treatment used to treat thyroid malignancies and is used for excision of the tumor and for staging. Most thyroid malignancies (>90%) are considered well differentiated and categorized as papillary thyroid carcinoma or follicular thyroid carcinoma. Papillary thyroid carcinoma accounts for 70% to 80% of thyroid malignancies and usually is discovered at an early stage and has an excellent prognosis.
Table 2. Oral Drugs Used to Treat Hyperthyroidism

(Adapted from Treatment guidelines from the Medical Letter: drugs for thyroid disorders. Med Lett Drugs Ther. 2009;84(7):57-64.)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Daily oral adult dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thionamides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methimazole</td>
<td>Starting: 10-40 mg once or in divided doses Maintenance: 5-15 mg once or in divided doses</td>
<td>Methimazole preferred due to ease of dosing and a better side effect profile; continue up to the morning of surgery</td>
</tr>
<tr>
<td>Propylthiouracil</td>
<td>Starting: 100-450 mg divided 2 or 3 times per day; maintenance: 100-150 mg divided 2 or 3 times per day</td>
<td></td>
</tr>
<tr>
<td>Iodide</td>
<td>1-3 drops 3 times per day</td>
<td>Iodide therapy added 1 wk before surgery and continued through the day of surgery; decreases production and release of thyroid hormone and reduces thyroid vascularity</td>
</tr>
<tr>
<td>Saturated solution of potassium iodide; Lugol solution</td>
<td>5 drops 3 times per day (dissolve in a full glass of water)</td>
<td></td>
</tr>
<tr>
<td>β-Blockers</td>
<td>20-40 mg 4 times per day</td>
<td>β-Blockers without intrinsic sympathomimetic activity are preferred; also used in emergency thyroid surgery for adrenergic suppression</td>
</tr>
<tr>
<td>Propranolol</td>
<td>80-160 mg once daily</td>
<td></td>
</tr>
<tr>
<td>(Inderal long acting)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atenolol (Tenormin)</td>
<td>25-100 mg once or twice daily</td>
<td></td>
</tr>
<tr>
<td>Metoprolol (Lopressor)</td>
<td>50-200 mg divided 2 or 3 times per day</td>
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Preoperative Medication Regimens

The preoperative use of antithyroid drugs has greatly decreased the morbidity from thyroid surgery and made thyroid storm a rare event. The aim of preoperative management is to restore a normal metabolic state before surgical intervention. Patients with hyperthyroidism have increased T₃ and T₄ values and decreased or normal TSH levels. The primary agents used to treat hyperthyroidism are the thionamide class of drugs, including methimazole and propylthiouracil. Both drugs inhibit the synthesis of thyroid hormone by preventing the oxidation and organic binding of thyroid iodide, preventing the organification of thyroglobulin. An intrathyroidal iodine deficiency is created that further increases the ratio of T₃ to T₄. Because T₃ is more potent than T₄, a high T₃ to T₄ ratio results in a greater metabolic rate. Propylthiouracil also further inhibits the conversion of T₄ to T₃.¹³

Methimazole is preferred to propylthiouracil because it can be given once per day and is effective at low dosages. Major side effects are rare; however, agranulocytosis and hepatitis may occur. Minor side effects occur in 5% of patients and usually include nausea, rash, arthralgias, and gastric intolerance. The onset of action is approximately 2 weeks and is noted by decreased nervousness, fewer palpitations, and less tachycardia. A euthyroid state is usually achieved within 4 to 6 weeks. At that time, the dosage may be substantially reduced to maintain a normal metabolic state. At 7 to 10 days before surgery, after the metabolic state has returned to normal, iodine therapy is generally started with saturated solution of potassium iodide or Lugol solution. Therapy with iodine should not be started until a euthyroid state has been achieved. Antithyroid drugs should not be discontinued when iodine therapy is started but are continued until the morning of surgery.¹⁴

Many of the effects of hyperthyroidism are mediated through adrenergic receptors, specifically, β receptors. The β-blocking drugs are widely used as an adjunct to thionamides for symptomatic control. They help decrease the palpitations, anxiety, heat intolerance, tremors, profuse sweating, and tachycardia produced by high thyroid hormone levels. Propranolol has been traditionally used; however, atenolol and metoprolol are both β₁ selective and may be substituted for propranolol. For patients with a contraindication to β-blockers, diltiazem has been successfully substituted. The β-blockers are generally continued throughout the surgical period and may be incrementally withdrawn postoperatively.¹⁵ Drugs that are used to treat hyperthyroidism are listed in Table 2.

Preoperative Anesthetic Management

The primary goals of the preoperative assessment are ensuring that the patient is euthyroid, assessing the degree of end-organ complications, and determining the extent of airway involvement.¹³,¹⁶ Patients who have not been medically managed for hyperthyroidism are at increased risk for severe perioperative hemodynamic complications. In addition, thyroid tumors and large goiters can...
impinge on the tracheal cartilage and esophageal tissues, resulting in tracheomalacia and airway compromise. Preoperatively, the patient will be taking a combination of antithyroid medications to decrease the synthesis and release of thyroid hormone and to treat the hyperdynamic state associated with hyperthyroidism. The patient may have taken or be currently taking glucocorticoids and, thus, require the administration of a steroid stress dose preoperatively or intraoperatively. In addition, a single dose of steroids can be given to reduce the incidence of postoperative nausea and vomiting. Patients should continue their regimen of antithyroid medications and β-blockade through the morning of surgery.

A euthyroid state is associated with a normal temperature, a normal resting heart rate, and a normal blood pressure. Lack of patient fatigue, intolerance to heat, and weight loss will further aid in determining the efficacy of the preoperative antithyroid medication regimen.

A thorough airway assessment is vital for patients with hyperthyroidism, including visualization and palpation of the patient’s neck to determine if a thyroid goiter exists. Thyroid gland enlargement can cause tracheal and esophageal deviation and compression. Therefore, it is imperative to assess the patient’s ability to breathe while in the supine position. Tracheomalacia can occur from chronic pressure on the tracheal cartilage, indicating the possibility for prolonged intubation. Signs and symptoms of tracheoesophageal compression include stridor, hoarseness, sore throat, a feeling of pressure in the neck, coughing, dysphagia, and/or dyspnea. The anesthetist should consider an awake fiberoptic intubation under topical anesthesia for any patient who is perceived as having a difficult airway, has a large goiter, or has altered airway anatomy.

Preoperative testing should be guided by the patient’s health status and coexisting disease processes. Table 3 lists suggested preoperative tests for patients having a thyroid resection. If there is any indication for the potential for airway compromise, a chest radiograph and a computed tomography scan of the neck and chest should be performed. Patients with hyperthyroidism commonly experience palpitations, increased heart rate, congestive heart failure, and/or atrial fibrillation. If any of these complications exist, an echocardiogram and a consultation with a cardiologist should be obtained to evaluate myocardial function. Patients with hyperthyroidism have a higher incidence of myasthenia gravis and may have skeletal muscle weakness. Care should be exercised because patients with myasthenia gravis may have an increased sensitivity to muscle relaxants. If emergency surgery is necessary for a patient with hyperthyroidism, the usual treatment modality will be ineffective because achieving a euthyroid state usually takes 6 to 7 weeks. Therefore, surgery should proceed only if a life-threatening situation exists. If time permits, oral potassium iodide can be given 6 hours preoperatively to block hormone synthesis and release. A β-blocker such as metoprolol or esmolol should be titrated to decrease the heart rate, blood pressure, and sympathetic nervous system activation.

Intraoperative Anesthetic Management

Intraoperative anesthetic management should focus on the prevention of sympathetic nervous system stimulation, proper positioning, and adequate hemodynamic monitoring. An adequate depth of anesthesia should be established and maintained to prevent sympathetic nervous system stimulation.

General endotracheal anesthesia is the anesthetic technique of choice for thyroidectomy. Infrequently, local anesthesia such as a cervical plexus block can be considered; however, this technique is reserved for patients in poor health in whom a general anesthetic is not indicated. Thiopental administered for induction has been shown to have antithyroid activity; however, it is doubtful that significant antithyroid activity would result after a single dose. Muscle relaxation can be provided to assist with tracheal intubation as long as the airway is not compromised by a tumor or goiter. Paralysis is not routinely maintained because it will inhibit the surgeon’s ability to assess the integrity of the RLN.

The Medtronic NIM electromyographic (EMG) endotracheal tube (Medtronic Xomed) is constructed of a flexible silicone elastomer and has a distal inflatable cuff. The tube is fitted with 4 stainless steel wire electrodes (2 pairs) that are embedded in the silicone of the main shaft of the endotracheal tube and exposed only for a short period.
distance, slightly superior to the cuff. The electrodes are designed to make contact with the patient's vocal cords to facilitate EMG monitoring of the RLN when connected to a multichannel EMG monitoring device. If monitoring correctly, the EMG monitor should show a consistent sound signal and an action potential tracing. The red wire pair of the NIM tube should contact the anterior and posterior portions of the right true vocal cord, and the blue wire pair should contact the anterior and posterior portions of the left true vocal cord (Figure 5). Paralysis and laryngeal tracheal anesthesia administration with lidocaine inhibit accurate EMG readings. Research is ongoing regarding the efficacy of EMG; however, some studies have shown no statistically significant difference in outcome between a standard endotracheal tube and an NIM tube.19,20

At various institutions, the NIM tube is inserted by using a GlideScope (Verathon, Bothell, Washington), that allows the surgeon and the anesthetist to visualize the vocal cords for correct placement.

Maintenance of anesthesia can be provided by inhalational anesthetics such as sevoflurane and isoflurane with or without nitrous oxide. The inhalation agents have an inhibitory effect on the sympathetic nervous system, which is especially important for patients with hyperthyroidism.1,16 Care should be taken when providing muscle relaxation for patients with underlying myasthenia gravis. If muscle relaxation is used, cautious titration and frequent monitoring of neuromuscular function with a nerve stimulator are prudent.3 A combined deep and superficial cervical plexus block can be considered for intraoperative and postoperative pain management.21

The patient should be constantly monitored for an increase in core body temperature and a hyperdynamic response.1,3 If hypotension occurs, it is best treated with a direct acting vasopressor (phenylephrine) rather than an indirect acting vasopressor (ephedrine) that stimulates the release of catecholamines.3 Hypercarbia and hypoxia are potent stimulators of the sympathoadrenal axis; therefore, adequate inspired oxygen concentrations and minute ventilations should be maintained.1

The patient is positioned supine with the head elevated 30° and the neck extended by using a roll behind the neck and shoulders (Rose position).16 The arms are tucked at patient's sides with the ulnar nerves padded and protected. Hyperextension of the neck should be avoided in patients with atlantoaxial joint instability and in patients with limited range of motion. Due to the limited access to the face, special care should be taken to protect the eyes from injury, especially in patients with exophthalmos.

Figure 5. Nerve Integrity Monitor (NIM) Endotracheal Tube
A. Refers to proper placement of the NIM endotracheal tube in relation to the thyroid gland.
B. Depicts the 4 electrode contact points in relation to the vocal cords.
LU indicates left upper; LL, left lower; RU, right upper; and RL, right lower.
Endoscopic thyroidectomy can be performed by assisted thyroidectomy and total endoscopic thyroidectomy; or excision of the affected lobe of the gland (lobar). During surgical excision, hemostasis is crucial; the 2 anterior jugular veins must be avoided, and ligation of the middle thyroid arteries is crucial. In addition, the right and left RLNs, which lie along the tracheal esophageal grooves, need to be identified to avoid injury. The estimated blood loss is approximately 50 to 75 mL.16

In an effort to improve cosmetic results by minimizing scarring of the neck, minimally invasive techniques for thyroid removal have been developed, including video-assisted thyroidectomy and total endoscopic thyroidectomy. Total endoscopic thyroidectomy can be performed by accessing the neck, chest wall (subclavicular or breast), or axilla. Insufflation of carbon dioxide is used to aid surgical visualization and excision.22 Minimally invasive techniques for removal of the thyroid gland remain controversial because the gland must be removed intact for adequate histologic analysis.16

- **Thyroid Storm.** Any patient undergoing a thyroidectomy is at risk for thyroid storm throughout the perioperative period, even if the patient is in a euthyroid state preoperatively. Thyroid storm is most frequently caused by physiologic stress.22 Thyroid storm is a life-threatening emergency, and its manifestations include tachycardia, hyperpyrexia, hypertension, tremor, sweating, widened pulse pressure, agitation, confusion, dysrhythmias, myocardial ischemia, and congestive heart failure. Prompt recognition and diagnosis are essential to decrease morbidity and mortality because similarities exist in the symptoms of thyroid storm, pheochromocytoma, malignant hyperthermia, neuroleptic malignant syndrome, sepsis, anaphylaxis, and light anesthesia.1,23

Treatment of thyroid storm involves restoring intravascular volume, providing hemodynamic support, and controlling hyperthermia. Table 4 lists the interventions used to manage thyroid storm. Invasive hemodynamic monitoring and vasodilating medications may be necessary to treat hypertension and cardiovascular instability.1,23

Excessively forceful coughing during emergence should be avoided to prevent complications such as a hematoma at the surgical site. The surgeon should be encouraged to inject local anesthesia at the surgical incision to decrease postoperative pain. Vocal cord function may need to be visually assessed before extubation due to the possibility of RLN damage, tracheomalacia, or hematoma.1,16 The patient should be monitored closely for postoperative respiratory distress.

**Postoperative Management**

The most common postoperative complications include hypocalcemia, RLN damage, and hematoma at the surgical site. A comprehensive list of the potential postoperative complications is given in Table 5.

Postoperative hypocalcemia can result from hypoparathyroidism. The 4 parathyroid glands (2 superior and 2 inferior) are located on the posterior aspect of the thyroid gland and produce parathyroid hormone, which increases the serum calcium level.1,3,16,24 Inadequate release of parathyroid hormone is due to the inadvertent removal of the parathyroid glands during a total thyroidectomy. It can also occur secondary to parathyroid gland devascularization, injury, or “stunning” from section.23,25

Hypocalcemia causes neuronal excitability in sensory and motor nerves. Patients most commonly experience signs and symptoms associated with hypocalcemia 24 to 96 hours postoperatively.3 The degree of hypocalcemia coincides with the severity of the symptoms, which include perioral numbness and tingling, abdominal pain, paresthesias of the extremities, carpopedal spasm, tetany, laryngospasm, mental status changes, seizures, Q-T prolongation on the electrocardiogram, and cardiac arrest.1,3,24,26

Neuromuscular irritability can also be confirmed by assessing for the Chvostek sign (facial contractions elicited by tapping the facial nerve in the periauricular area) and the Trousseau sign (carpal spasm after inflation of a blood pressure cuff).24,26 In addition, monitoring postoperative ionized calcium levels is recommended because these values are reflec-

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**Table 4. Management of Thyroid Storm (Adapted from Schwartz et al.16)**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ensure adequate oxygenation</td>
<td></td>
</tr>
<tr>
<td>Administer glucose-containing intravenous fluids</td>
<td></td>
</tr>
<tr>
<td>Administer β-adrenergic blockers</td>
<td></td>
</tr>
<tr>
<td>Administer sodium iodide</td>
<td></td>
</tr>
<tr>
<td>Administer propylthiouracil or methimazole</td>
<td></td>
</tr>
<tr>
<td>Administer glucocorticoids</td>
<td></td>
</tr>
<tr>
<td>Correct electrolyte imbalances</td>
<td></td>
</tr>
<tr>
<td>Correct acid-base imbalances</td>
<td></td>
</tr>
<tr>
<td>Administer acetaminophen</td>
<td></td>
</tr>
<tr>
<td>Apply cooling blankets</td>
<td></td>
</tr>
</tbody>
</table>

**Table 5. Complications Associated With Thyroidectomy (Adapted from Koransky et al.16)**

<table>
<thead>
<tr>
<th>Complication</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypocalcemia-hypoparathyroidism</td>
<td></td>
</tr>
<tr>
<td>Recurrent laryngeal nerve injury, unilateral or bilateral</td>
<td></td>
</tr>
<tr>
<td>Neck hematoma</td>
<td></td>
</tr>
<tr>
<td>Thyroid storm</td>
<td></td>
</tr>
<tr>
<td>Superior laryngeal nerve injury</td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td></td>
</tr>
<tr>
<td>Pneumothorax</td>
<td></td>
</tr>
<tr>
<td>Tracheomalacia</td>
<td></td>
</tr>
<tr>
<td>Sympathetic trunk nerve injury (rare) resulting in Horner syndrome</td>
<td></td>
</tr>
<tr>
<td>Thoracic duct injury (rare), chylous fistula</td>
<td></td>
</tr>
</tbody>
</table>
tive of the physiologically active form of calcium.

Treatment for severe symptomatic hypocalcemia includes the administration of calcium gluconate or calcium chloride (10 mL of 10% solution) intravenously during several minutes and followed by a continuous infusion (1-2 mg/kg per hour) until calcium levels normalize.3,26

Damage to the RLN during thyroidectomy is rare and estimated to occur in up to 14% of cases. The surgical identification and preservation of the RLN is essential to avoid injury. A study by Canbaz et al27 supports the practice of identification and exposure of the RLN and its branches during total thyroidectomy, which decreases the rate of RLN injury. Damage to the RLN may be unilateral or bilateral; however, unilateral nerve injury is more common.3

Unilateral RLN damage causes the ipsilateral vocal cord to remain midline during inspiration, resulting in hoarseness. Bilateral RLN injury results in dysfunction of both vocal cords, which remain midline during inspiration. After extubation, biphasic stridor, respiratory distress, and aphony occur due to unopposed adduction of the vocal cords and closure of the glottic aperture. Unlike unilateral nerve injury, bilateral nerve injury necessitates immediate intervention with emergency reintubation or tracheotomy.3,26 The external branch of the superior laryngeal nerve controls motor function to the pharyngeal constrictor and cricothyroid muscles. Although damage to the superior laryngeal nerve is a rare event during thyroidectomy, denervation can result in hoarseness.

Postoperative bleeding of the surgical site results in a hematoma in the neck that causes airway obstruction and asphyxiation. Common symptoms of a hematoma in the neck include neck swelling, neck pain and pressure, dyspnea, and stridor. Initial treatment includes the emergency evacuation of the hematoma followed by airway management.1,3,26

Due to the intricacy of the surgical procedure, the use of the NIM endotracheal tube, and the complex physiologic and pathophysiologic process that are associated with hyperthyroidism, a thorough knowledge of these concepts is necessary to formulate a comprehensive anesthetic plan. Communication between the surgeon and anesthetist is vital for comprehensive and safe intraoperative management. A postoperative assessment is important to ensure that the patient's airway remains patent and hemodynamic stability is maintained.

REFERENCES


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