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Thyroid imbalances:

Dealing with disorderly conduct

By Ann Crawford, PhD, RN, and Helene Harris, MSN, RN

THYROID HORMONES AFFECT overall metabolism and electrolyte balance. Disorders in thyroid hormone function can cause widespread and potentially life-threatening effects. This article reviews hyperthyroidism and hypothyroidism and what nurses need to know about each condition. For details about this endocrine gland, see *All about thyroid function* and *PTH and calcitonin: Opposing actions maintain balance*.

HYPERTHYROIDISM

A clinical syndrome, hyperthyroidism results when tissues are exposed to high levels of circulating thyroid hormone. In most cases, hyperthyroidism is due to hyperactivity of the thyroid gland.¹ This common endocrine disorder can occur at any age, although most patients are diagnosed between ages 20 and 40. Women are much more likely to be diagnosed with a form of hyperthyroidism than men.^{2,3} The high levels of circulating thyroid hormones increase sympathetic nervous system (SNS) activity and metabolic rate, causing many of the clinical manifestations of hyperthyroidism.¹ (See *Clinical manifestations of hyperthyroidism and hypothyroidism*.) Hyperthyroidism may be acute or chronic, depending on its underlying etiology.

Thyroid hormone overstimulation of the cardiovascular system causes “fight-or-flight” responses including an increase in heart rate, stroke volume, myocardial contractility, and BP.^{1,2} No matter what the cause, manifestations of overproduction of thyroid hormones are termed *thyrotoxicosis*. However, thyrotoxicosis isn’t synonymous with hyperthyroidism, and thyrotoxicosis can occur without hyperthyroidism.

Elevated thyroid hormone levels increase the body’s metabolic rate and affect the metabolism of proteins, fats, and carbohydrates. Because of this, although the patient has an increased appetite and food intake, energy needs exceed the supply and the patient loses weight. With the high metabolic rate, protein degradation exceeds protein synthesis, causing a

negative nitrogen balance. Fat metabolism is increased, reducing fat stores. Sustained hyperthyroidism leads to more chronic nutritional deficits.^{1,2}

Hypersecretion of thyroid hormones can also affect the secretion of hypothalamic and anterior pituitary hormones, including sex hormone production.²

All about thyroid function^{1,2,4}

The thyroid is an endocrine gland located in the anterior portion of the neck, just below the cricoid cartilage. Its two lobes are positioned on opposite sides of the trachea, and are joined by a narrow band of tissue (isthmus) that passes across the trachea anteriorly, giving the gland a butterfly-like shape. The tissue is composed of two types of cells: follicular cells, which produce thyroxine (T₄) and triiodothyronine (T₃), and parafollicular cells, which produce and secrete thyrocalcitonin (also called calcitonin).

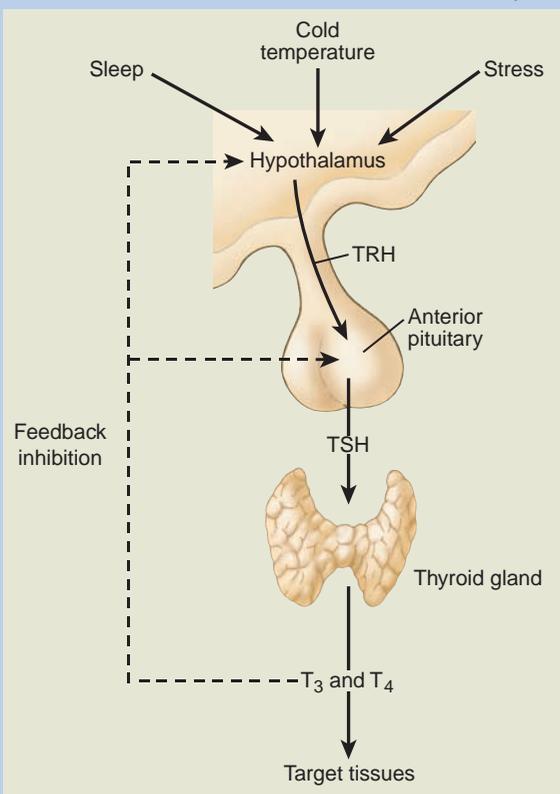
The manufacture of thyroid hormones requires dietary ingestion of protein and iodine. An iodine deficiency leads to decreased production of T₃ and T₄, which may lead to goiter.

T₃ and T₄ increase metabolism, which in turn increases oxygen utilization and heat production by the body tissues. Most circulating T₃ and T₄ are bound to plasma proteins; a smaller portion circulates as free hormone that can easily enter cells and attach to a receptor site within the cell's nucleus. When T₃ or T₄ binds to this receptor, it turns on genes important to metabolism.

T₃ is a more active thyroid hormone, so following entrance into the cell, T₄ is converted into T₃. Several factors may impede the conversion of T₄ to T₃, including stress, starvation, beta-blockers, amiodarone, corticosteroids, iodinated contrast media, and PTU. Conversely, cold temperatures may increase the conversion.

T₃ and T₄ are secreted through a negative feedback mechanism involving the hypothalamus, the anterior pituitary gland, and the thyroid gland. In response to certain stimuli such as cold temperatures and stress, the hypothalamus secretes thyrotropin-releasing hormone (TRH), which prompts the anterior pituitary gland to release TSH. TSH then stimulates the thyroid gland to produce and release the thyroid hormones. If circulating levels of T₃ and T₄ are low, release of TSH is increased. Conversely, if circulating levels of the thyroid hormones are elevated, release of TSH is inhibited.

Calcitonin, the other hormone produced in the thyroid gland, decreases serum calcium by reducing bone resorption (break-down). Calcitonin also reduces the renal tubular reabsorption of calcium and phosphate. The level of calcium in the blood dictates calcitonin secretion. Low calcium levels inhibit secretion of calcitonin, and high calcium levels stimulate its secretion. In addition, pregnancy, a high-calcium diet, and increased secretion of gastrin also increase calcitonin secretion.²



Looking for causes

Patients taking the antiarrhythmic drug amiodarone, a heavily iodinated compound, can develop hyperthyroidism or hypothyroidism. Thyrotoxicosis can be caused by radiation exposure, ingestion of excess thyroid hormone, thyroiditis (usually temporary), interferon-alpha therapy, pituitary tumors, and metastatic thyroid cancer.³

Graves disease is an autoimmune disease characterized by abnormal stimulation of the thyroid gland, in which the body makes antibodies that bind to the thyroid-stimulating hormone (TSH) receptor sites in the thyroid. Thyroid-stimulating immunoglobulins attach to thyroid tissue, causing gland hypertrophy and thyroid hormone overproduction. Patients with Graves disease demonstrate the key features of thyrotoxicosis.^{1,3}

Toxic multinodular goiter is hyperthyroidism caused by multiple thyroid nodules, usually composed of enlarged thyroid tissue or benign tumors. The overproduction of thyroid hormones is usually less severe than in Graves disease. Clinical presentation is similar, though without the manifestation of exophthalmos or pretibial edema.²

Diagnostic testing and management

Hyperthyroidism is diagnosed based on the patient's health history, clinical presentation, and blood work results, including T₃, T₄, TSH, and T₃ resin uptake. Measurement of TSH receptor antibodies may be used to diagnose Graves disease.²

A radioiodine uptake and thyroid scan may be performed to evaluate the thyroid gland's size, position, and function. A thyroid ultrasound may also be used to determine the size of the thyroid gland and presence of masses or nodules.^{2,4}

Treatment for hyperthyroidism includes antithyroid drug therapy, radioiodine, and surgery (subtotal or near-total thyroidectomy).⁵ Medications such as methimazole and

PTH and calcitonin: Opposing actions maintain balance

Actions of parathyroid hormone (PTH) are opposite to those of calcitonin. PTH secretion stimulates increased bone resorption, with resultant elevations in serum calcium and phosphorus levels. Through coordinated management of calcitonin and PTH release and inhibition, the endocrine system in healthy people maintains appropriate serum levels of calcium and phosphorus.

Actions	Parathyroid hormone	Calcitonin
Intestinal absorption of calcium	Increases indirectly through increased activation of vitamin D	Probably not affected
Intestinal absorption of phosphate	Increases	Probably not affected
Renal excretion of calcium	Decreases	Minor effect
Renal excretion of phosphate	Increases	Minor effect
Bone resorption	Increases	Decreases
Bone formation	Decreases	Uncertain
Serum calcium levels	Produces a prompt increase	Decreases with pharmacologic doses
Serum phosphate levels	Prevents an increase	Decreases with pharmacologic doses.

Source: Porth CM. *Essentials of Pathophysiology*. 3rd ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams and Wilkins; 2011:1090.

propylthiouracil (PTU) inhibit the synthesis of thyroid hormones, but don't inactivate circulating thyroid hormones or those stored in the thyroid. These medications are used for long-term management. Potassium iodide and iodine solutions inhibit the synthesis and release of the thyroid hormone, and generally are used for short-term therapy, such as before thyroid surgery.⁶

Beta-blockers can be used to help control adrenergic signs and symptoms, especially in the early stages of treatment before the antithyroid drugs take effect. These drugs block the effects of thyroid hormone on SNS receptors in the heart, decreasing myocardial oxygen demands.⁶

Radioiodine (radioactive iodine) causes progressive destruction of thyroid cells. Potential adverse reactions include pharyngitis or neck tenderness for a few days, and complications include hypothyroidism.⁶ Radioiodine may exacerbate ophthalmopathy for patients with Graves disease (more on this later).⁶ Because fewer than 50% of patients treated with antithyroid medications remain in long-term remission, radioactive iodine is used increasingly as a permanent treatment.⁵

Surgery, once a primary method of treating hyperthyroidism, is reserved today for special circumstances, such as a patient's inability to take antithyroid medication, pregnancy, a large goiter, or ophthalmopathy.^{2,3} Most of

the thyroid gland is removed, permanently reducing thyroid hormone production. Following the surgery, most patients must take thyroid replacement medication daily to prevent hypothyroidism.^{5,7} Major complications of surgery include bleeding, laryngeal edema, hypoparathyroidism, and recurrent laryngeal nerve damage.⁸

Nursing considerations and patient teaching

Monitor the patient's vital signs closely; an alteration in metabolic rate can lead to significant increases in heart rate, BP, and temperature. Assess all body systems and monitor food intake and appetite. Also assess muscle strength and watch for tremors, heat intolerance, emotional changes, and irritability, which could indicate thyrotoxicosis.

Monitor the patient's thyroid hormone levels to determine if antithyroid medications are effective. Some antithyroid drugs can cause leukopenia, agranulocytosis, and hepatotoxicity, so monitor the patient's complete blood cell count and liver panel results, including bilirubin and transaminases. Closely monitor serum glucose and electrolyte levels, especially in patients exhibiting manifestations of thyrotoxic crisis.⁶ (See *Responding to life-threatening crises*.)

Iodine toxicity can occur if the patient is taking too much iodine or the dose is too high. Signs of iodine

toxicity include a burning sensation in the mouth and throat, metallic taste, sore teeth and gums, increased salivation, nasal discharge, gastrointestinal upset, and swollen eyelids. Teach patients to report these signs and symptoms to their healthcare provider immediately.

Because antithyroid medications work to decrease thyroid function, be alert for manifestations of hypothyroidism.⁶ Consult the full prescribing information for details about drugs prescribed for your patient.

Encourage patients to have regular follow-up appointments, which include monitoring their thyroid hormone levels. Teach them to avoid foods high in iodine, such as seafood, tofu, soy, cheddar cheese, mayonnaise, and iodized salt, and to wear a medical-alert bracelet or medal.

Patients with Graves disease are at risk for Graves ophthalmopathy, an autoimmune disease in which inflammation increases the volume of the extraocular muscles and retro-orbital tissues. Tell patients to notify their healthcare provider if they develop periorbital edema, eye irritation, blurred vision, and excessive tearing worsened by exposure to cold, wind, or bright lights.⁹

HYPOTHYROIDISM

A deficiency of thyroid hormones, hypothyroidism decreases metabolic rate. Primary hypothyroidism

(99% of diagnosed cases) is caused by an autoimmune disease, or iatrogenic destruction of the thyroid gland. Iatrogenic causes include surgical removal or irradiation of thyroid tissue, or medications that suppress thyroid function, such as antithyroid drugs and lithium. Other causes include postpartum thyroiditis, subacute granulomatous thyroiditis, and inadequate ingestion of necessary ingredients for thyroid hormone formation (such as iodine or tyrosine).^{2,10} Amiodarone, as mentioned earlier, can cause hyperthyroidism or hypothyroidism.³

Congenital hypothyroidism, which occurs in about 1 of every

5,000 births, is a preventable cause of mental retardation. With proper neonatal screening and treatment, this problem is easily resolved.¹ In adults with adequate iodine intake, the most common cause of hypothyroidism is chronic autoimmune thyroiditis, or Hashimoto disease.^{7,10}

Less commonly, hypothyroidism is caused by decreased secretion of TRH from the hypothalamus or decreased secretion of TSH from the anterior pituitary gland.¹¹ Insufficient stimulation of the thyroid gland by TSH (central hypothyroidism) is caused by either pituitary disease (secondary hypothyroidism) or

hypothalamic disease (tertiary hypothyroidism).¹¹

Hypothyroidism occurs most often in women, who are usually diagnosed between ages 30 and 60.^{2,7} Overall, hypothyroidism is more common than hyperthyroidism.¹²

Low levels slow things down

In hypothyroidism, low circulating levels of thyroid hormone cause a generalized slowing of metabolic processes. Low thyroid hormone levels stimulate the anterior pituitary gland to release TSH in an attempt to trigger the production of more thyroid hormones. TSH binds to the thyroid tissue and over time causes goiter, an abnormal enlargement of the thyroid gland, although it doesn't necessarily promote increased thyroid hormone production.^{1,2,10}

The decreased metabolic rate affects most tissues and organs. As cellular energy diminishes, metabolites (called glycosaminoglycans) build up within the cells, forming a cellular edema of water mixed with mucus, known as *myxedema*. This nonpitting edema develops throughout the body, most conspicuously around the eyes, in the hands and feet, and between the scapulae. The tongue appears enlarged and the patient's voice may sound husky or hoarse due to laryngeal edema.²

Clinical manifestations of hypothyroidism are caused by the decreased metabolic rate. Because hypothyroidism often occurs slowly, the onset of signs and symptoms is insidious, developing over months to years.² Because many of the clinical manifestations are vague, nonspecific, and slow to develop, they may be ignored or unnoticed by patients and their families.^{1,2,10}

Diagnostic testing and management

As with hyperthyroidism, diagnosis for hypothyroidism is based on the patient's health history, clinical manifestations, and serum thyroid

Clinical manifestations of hyperthyroidism and hypothyroidism

	Hyperthyroidism	Hypothyroidism
Basal metabolic rate	Increased	Decreased
Sensitivity to catecholamines	Increased	Decreased
General features	<ul style="list-style-type: none"> • Exophthalmos (in Graves disease) • Lid lag • Accelerated growth (child) 	<ul style="list-style-type: none"> • Myxedematous features • Deep voice • Impaired growth (child)
Blood cholesterol levels	Decreased	Increased
General behavior	<ul style="list-style-type: none"> • Restlessness, irritability, anxiety • Hyperkinesia • Wakefulness 	<ul style="list-style-type: none"> • Mental retardation (infant) • Mental and physical sluggishness • Somnolence
Cardiovascular function	<ul style="list-style-type: none"> • Increased cardiac output • Tachycardia and palpitations 	<ul style="list-style-type: none"> • Decreased cardiac output • Bradycardia
Gastrointestinal function	<ul style="list-style-type: none"> • Diarrhea • Increased appetite 	<ul style="list-style-type: none"> • Constipation • Decreased appetite
Respiratory function	Dyspnea	Hypoventilation
Muscle tone and reflexes	Increased, with tremor and twitching	Decreased
Temperature tolerance	Heat intolerance	Cold intolerance
Skin and hair	<ul style="list-style-type: none"> • Increased sweating • Thin and silky skin and hair 	<ul style="list-style-type: none"> • Decreased sweating • Coarse and dry skin and hair
Weight	Loss	Gain

Source: Porth CM. *Essentials of Pathophysiology*. 3rd ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams and Wilkins; 2011:786.

hormone levels and TSH. In overt primary hypothyroidism, serum TSH is elevated and T3 and T4 levels are decreased. In subclinical primary hypothyroidism, serum TSH levels are high and T3 and T4 levels are normal.^{4,10,11}

The treatment of choice for hypothyroidism is thyroid hormone replacement with levothyroxine, a synthetic form of T4. Levothyroxine has the same effects as the body's own thyroid hormone, increasing the metabolic rate, which in turn increases the heart rate; respiratory rate; BP; and fat, protein, and carbohydrate metabolism.⁶ Adverse reactions to levothyroxine are potentially serious and may mimic the manifestations of hyperthyroidism.⁶

Use levothyroxine cautiously in older adults and in patients with cardiovascular disease, hypertension, or diabetes. Patients with adrenal insufficiency or severe cardiovascular disease shouldn't take levothyroxine.¹³

Other medications for treating hypothyroidism include desiccated thyroid extract (pork or beef), liothyronine (T3), and liotrix (4:1 mixture of T4 and T3).¹⁴

Nursing interventions

Obtain a health history and perform medication reconciliation, including all prescription and over-the-counter medications, and herbal and nutritional supplements. Monitor vital signs regularly and thoroughly assess patients' physiologic status, including cardiovascular, neurologic, and pulmonary status, and renal function. To help determine nutritional status, ask patients what they've weighed in the past. Monitor serum thyroid hormone levels, blood urea nitrogen, and creatinine.

Because levothyroxine may potentiate the effects of anticoagulants, monitor coagulation lab values (prothrombin time and international normalized ratio), and watch for signs and symptoms of bleeding. Also watch for signs of hyperglycemia in all patients taking levothyroxine, but especially in those with diabetes who

Responding to life-threatening crises

Also known as thyroid storm, **thyrotoxic crisis** is a life-threatening exacerbation of hyperthyroidism that occurs in patients with an undiagnosed or inadequately treated hyperthyroid state.⁸ Most commonly associated with Graves disease, thyrotoxic crisis is caused by a heightened response in the catecholamine receptor sites that intensifies SNS effects on the body. Clinical manifestations of hyperthyroidism are exaggerated in this crisis state and include fever, cardiovascular changes (chest pain, tachycardia, hypertension, heart failure), and neurologic issues (agitation, confusion, delirium). Without prompt treatment, patients can die from heart failure.^{1,3,4}

For patients who develop thyrotoxic crisis, rapid diagnosis and treatment is key to survival. Clinicians must also identify and treat (whenever possible) the acute event that often precipitates it, such as infection. Apply cooling blankets and ice packs to aggressively treat hyperpyrexia. As prescribed, administer acetaminophen rather than aspirin, which can increase serum free T3 and T4 levels by interfering with their protein binding.¹⁵ Also administer replacement fluids, electrolytes, and glucose as prescribed to maintain normal serum levels.

The therapeutic regimen typically includes:

- beta-blockers to control signs and symptoms of increased adrenergic tone and reduce myocardial oxygen demands.
- a thionamide to block thyroid hormone synthesis. PTU, the most effective drug to block thyroid synthesis, is normally administered orally. If the patient can't take anything orally, PTU may be administered rectally.
- an iodine solution to block thyroid hormone release.
- glucocorticoids or an iodinated radiocontrast agent to reduce the conversion of T4 to T3, and to promote vasomotor stability.^{6,15}

Myxedema coma is a rare, life-threatening emergency that occurs due to unrecognized, untreated, or undertreated hypothyroidism, and is often seen in older adults (usually women) in the winter months. The patient will exhibit a worsening of hypothyroidism signs and symptoms (hypothermia, bradycardia, hypotension, hypoventilation, hyponatremia, hypoglycemia, and metabolic acidosis), deteriorating neurologically to a comatose state.¹⁶ Diminished metabolism causes respiratory depression, cardiovascular collapse, and shock.^{2,4,7}

Treatment of myxedema coma includes mechanical ventilation, thyroid replacement therapy, fluid and vasopressor administration, passive rewarming, and I.V. dextrose. However, even with aggressive treatment, mortality is high.^{2,4,7}

take insulin or oral antidiabetic drugs. Levothyroxine opposes the effects of insulin in the body, increasing the risk of hyperglycemia.⁶

Monitor for continued signs and symptoms of hypothyroidism, which may indicate a need to increase the medication dosage, and for signs and symptoms of hyperthyroidism, which may indicate a need to decrease the dosage. Use appropriate patient safety measures and assist with ambulation and activities of daily living as needed.⁶

Patient education

Teach patients that thyroid replacement therapy must be taken for life, and warn them not to discontinue medication or change brands or dosages without contacting their healthcare provider. Because thyroid

medications can cause insomnia if taken at night, they're usually administered in the morning on an empty stomach. Because iron and calcium supplements and antacids can decrease absorption of levothyroxine, patients shouldn't take these supplements or antacids within 4 hours of taking levothyroxine. Patients should follow up with their healthcare provider regularly, including assessment of serum thyroid hormone levels.

Teach patients to change positions slowly and to notify their healthcare provider immediately if they develop an increased or irregular pulse, palpitations, nervousness, heat intolerance, diarrhea, sweating, or irritability. Women who are pregnant, breastfeeding, or planning to get pregnant should notify their healthcare provider.⁶

Encourage patients to wear a medical-alert bracelet or medal. Tell them not to consume large amounts of the following foods, which can prevent thyroid hormone secretion: strawberries, peaches, pears, cabbage, spinach, turnips, peas, cauliflower, and radishes.⁶

Restoring order

Thyroid disorders are complex and can affect many body systems. By knowing how to recognize hyperthyroidism and hypothyroidism and their complications, you can help patients get appropriate care and avoid life-threatening complications. ■

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The authors and planners have disclosed that they have no financial relationships related to this article.

DOI-10.1097/01.NURSE.0000421372.90370.3e

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