Understanding the endocrine system

The endocrine system consists of glands, specialized cell clusters, and hormones, which are chemical transmitters secreted by the glands in response to stimulation. Together with the central nervous system (CNS), the endocrine system regulates and integrates the body’s metabolic activities and maintains homeostasis.

Hypothalamus: The heart of the system
The hypothalamus is the integrative center for the endocrine and ANS (involuntary). It helps control some endocrine glands by neural and hormonal pathways.

On the path to the posterior pituitary gland
Neural pathways connect the hypothalamus to the posterior pituitary gland. Neural stimulation of the posterior pituitary gland in turn causes the secretion of two effector hormones—antidiuretic hormone (ADH) and oxytocin. When ADH is secreted, the body retains water. Oxytocin stimulates uterine contractions during labor and milk secretion in lactating women.

Please release me
The hypothalamus also exerts hormonal control at the anterior pituitary gland by releasing or inhibiting hormones. Hypothalamic hormones stimulate the anterior pituitary gland to release four types of trophic (gland-stimulating) hormones:
- adrenocorticotropic hormone (ACTH), also called corticotropin
- thyroid-stimulating hormone (TSH)
- luteinizing hormone (LH)
- follicle-stimulating hormone (FSH).

The secretion of trophic hormones stimulates their respective target glands, such as the adrenal cortex, the thyroid gland, and the gonads. Hypothalamic hormones also control the release of effector hormones from the pituitary gland. Examples are growth hormone (GH) and prolactin.

Getting feedback
A negative feedback system regulates the endocrine system by inhibiting hormone overproduction. This system may be simple or complex. A patient with a possible endocrine disorder needs careful assessment to identify the cause of the dysfunction. Dysfunction may result from defects:
- in the gland
- in the release of trophic or effector hormones
- in hormone transport
- of the target tissue.

How do you end up with an endocrine disorder?
Endocrine disorders may be caused by:
- hypersecretion or hyposecretion of hormones
- hyporesponsiveness of hormone receptors
- inflammation of glands
- gland tumors.

Dysfunctional
Hypersecretion or hyposecretion may originate in the hypothalamus, the pituitary effector glands, or the target gland. Regardless of origin, however, the result is abnormal hormone concentrations in the blood. Hypersecretion leads to elevated levels; hyposecretion leads to deficient levels.

Turn it off! What? Turn it off!!!
In hyporesponsiveness, the cells of the target organ don’t have appropriate receptors for a hormone. This means the effects of the hormone aren’t detected. Because the receptors don’t detect the
hormone, there’s no feedback mechanism to turn the hormone off. Blood levels of the hormone are normal or high. Hyporesponsiveness causes the same clinical symptoms as hyposecretion. 

An inflamed discussion (and tumor talk)
Inflammation is usually chronic, commonly resulting in glandular secretion of hormones. However, it may be acute or subacute, as in thyroiditis. Tumors can occur within a gland, as in thyroid carcinoma. In addition, tumors occurring in other areas of the body can cause abnormal hormone production (ectopic hormone production). For example, certain lung tumors secrete ADH or parathyroid hormone (PTH).

Glands
The endocrine glands release hormones into the circulatory system, which distributes them throughout the body. Glands discussed below include the: • adrenal glands • pancreas • pituitary gland • thyroid gland • parathyroid glands.
Adrenal glands
The adrenal glands produce steroids, amines, epinephrine, and norepinephrine. Hyposecretion or hypersecretion of these substances causes a variety of disorders and complications that range from psychiatric and sexual problems to coma and death. The adrenal cortex secretes three types of steroidal hormones: mineralocorticoids, glucocorticoids and adrenal androgens and estrogens.

**Aldosterone in action**
Aldosterone is a mineralocorticoid. It regulates the reabsorption of sodium and the excretion of potassium by the kidneys. It may play a role in HTN development.

**Cue cortisol** (a glucocorticoid)
- stimulation of gluconeogenesis (formation of glycogen from noncarbohydrate sources)
- suppression of immune response
- assistance with stress response
- assistance with maintenance of blood pressure and cardiovascular function.

**A gland with a lot of nerve**
The adrenal medulla has an aggregate of nerve tissue that produces the catecholamine hormones epinephrine and norepinephrine that cause vasoconstriction. Epinephrine causes the response to physical or emotional stress called the fight-or-flight response. This response produces bronchodilation, increased BP, HR, & blood glucose level.

**Pancreas**
The pancreas produces glucagon and insulin.

**Fasting? You'll need glucose fast . . .** Glucagon is a hormone stimulates the release of stored glucose from the liver (in fasting) to raise blood glucose levels.

**Multiple roles of insulin**
Insulin is a hormone released in the postprandial state. It aids glucose transport into the cells and promotes glucose storage. It also stimulates protein synthesis and enhances free fatty acid uptake and storage. Insulin deficiency or resistance causes diabetes mellitus.

**Pituitary gland**
The posterior pituitary gland secretes two effector hormones:
- oxytocin, stimulates uterine contractions during labor and causes milk let-down reflex in lactating women
- ADH, controls the concentration of body fluid and conserve water.

**The ABCs of ADH**
ADH secretion depends on plasma osmolality (concentration), which is monitored by hypothalamic neurons. Hypovolemia and hypotension are the most powerful stimulators of ADH release. Other stimulators include pain, stress, trauma, nausea, and the use of morphine, tranquilizers, and certain anesthetics.

**It's no secret**
In addition to the trophic hormones (ACTH, TSH, LH, and FSH), the anterior pituitary gland secretes prolactin and GH. Prolactin stimulates milk secretion in lactating females. GH affects most body tissues. It triggers growth by increasing protein production and fat mobilization and decreasing carbohydrate use.

**Thyroid gland**
The thyroid gland, located in the anterior neck, secretes the iodine-containing hormones thyroxine (T4) and triiodothyronine (T3). Thyroid hormones are necessary for normal growth and development. They also act on many tissues by increasing metabolic activity and protein synthesis.

**A good prognosis with treatment:** Diseases of the thyroid are caused by thyroid hormone overproduction or deficiency and gland inflammation and enlargement. Most patients have a good
prognosis with treatment. Untreated, thyroid disease may progress to an emergency (thyroid crisis/storm). It can also cause irreversible disabilities such as vision loss.

**Parathyroid glands**
There are four parathyroid glands located behind the thyroid gland. These glands secrete PTH, which helps regulate calcium levels and control bone formation.

**Disorderly conduct**
Disorders of the parathyroid gland involve hyposecretion of PTH resulting in a reduction of serum calcium that can lead to tetany and seizures, or hypersecretion of PTH, which results in elevated serum calcium levels that can lead to cardiac arrhythmias, muscle and bone weakness, and renal calculi.

**Endocrine disorders** The endocrine disorders discussed in this chapter include: • a pituitary disorder of water metabolism (diabetes insipidus) and a pancreatic disorder (diabetes mellitus) • three thyroid gland disorders (simple goiter, hyperthyroidism, and hypothyroidism).

**Diabetes insipidus (DI)**
DI is a disorder of water metabolism caused by a deficiency of ADH (vasopressin). The absence of ADH allows filtered water to be excreted in the urine instead of reabsorbed. The disease causes excessive urination and excessive thirst and fluid intake. It may first appear in childhood or early adulthood and is more common in men than in women.

**How it happens**
Some drugs as well as injury to the posterior pituitary gland can cause abnormalities in ADH secretion. A less common cause is a failure of the kidneys to respond to ADH. Lesions of the hypothalamus and posterior pituitary gland can also interfere with ADH synthesis, transport, or release. Lesions may be caused by brain tumor, removal of the pituitary gland (hypophysectomy), aneurysm, thrombus, immunologic disorder, or infection.

**When ADH is absent**
Normally, ADH is synthesized in the hypothalamus and then stored by the posterior pituitary gland. When it’s released into the general circulation, ADH increases the water permeability of the distal and collecting tubules of the kidneys, causing water reabsorption. If ADH is absent, the filtered water is excreted in the urine instead of being reabsorbed, and the patient excretes large quantities of dilute urine.

**What to look for:** The patient’s history shows:
• abrupt onset of extreme polyuria (4-16 L/day of dilute urine)
• polydipsia (extreme thirst) and polydipsia
In severe cases, fatigue occurs because sleep is interrupted. Children often have enuresis, sleep disturbances, irritability, anorexia, and decreased weight gain and linear growth. Additional signs and symptoms may include:
• weight loss • dizziness • weakness • constipation • increased serum sodium and osmolality.

**What lies underneath?**
The prognosis is good for uncomplicated DI with adequate water replacement, and patients usually lead normal lives. However, when the disease is complicated by an underlying disorder such as cancer, the prognosis varies.

**One thing leads to another**
Untreated DI can produce hypovolemia, hyperosmolality, circulatory collapse, loss of consciousness, and CNS damage. These complications are most likely to occur if the patient has an impaired or absent thirst mechanism.
A prolonged urine flow may produce chronic complications, such as bladder distention, enlarged calyces, hydrourereter, and hydronephrosis. Complications may also result from underlying conditions, such as metastatic brain lesions, head trauma, and infections.

What tests tell you
These tests distinguish diabetes insipidus from other disorders causing polyuria:
• Urinalysis reveals urine of low osmolality (50-200 mOsm/kg of water).
• Dehydration test differentiates ADH deficiency from other forms of polyuria
• Plasma or urinary ADH evaluation may be performed after fluid restriction or hypertonic saline infusion to determine whether DI originated from damage to the posterior pituitary gland (neurogenic) or failure of the kidneys to respond to ADH (nephrogenic). ADH levels are decreased in neurogenic DI and elevated in the nephrogenic type. If the patient is critically ill, diagnosis may be based on these laboratory values alone:
  • urine osmolality of 200 mOsm/kg
  • urine specific gravity of 1.005
  • serum osmolality of 300 mOsm/kg
  • serum sodium of 147 mEq/L.
Diabetes mellitus
Diabetes mellitus (DM) is a disease in which the body doesn’t produce or properly use insulin, leading to hyperglycemia. The disease occurs in two primary forms:
- type 1 (formerly referred to as insulin-dependent diabetes mellitus)
- type 2 (formerly referred to as non–insulin-dependent diabetes mellitus)
Several secondary forms also exist, caused by such conditions as pancreatic disease, pregnancy (gestational diabetes mellitus), hormonal or genetic problems, and certain drugs or chemicals. The incidence of DM increases with age.

How it happens
Normally, insulin allows glucose to travel into cells. There, it’s used for energy and stored as glycogen. It also stimulates protein synthesis and free fatty acid storage in adipose tissue. Insulin deficiency blocks tissues’ access to essential nutrients for fuel and storage. The pathophysiology behind each type of diabetes differs.

Type 1 diabetes
In type 1 diabetes, the beta cells in the pancreas are destroyed or suppressed. Type 1 diabetes is subdivided into idiopathic and immune-mediated types. With the idiopathic type, patients have a permanent insulin deficiency with no evidence of autoimmunity. In the immune-mediated type, a local or organ-specific deficit may induce an autoimmune attack on beta cells. This attack causes an inflammatory response in the pancreas called insulitis. Islet cell antibodies may be present long before symptoms become apparent. These immune markers also precede evidence of beta cell deficiency. Autoantibodies against insulin have also been noted. By the time the disease becomes apparent, 80% of the beta cells are gone.

Type 2 diabetes
Type 2 diabetes may be caused by:
- resistance to insulin action in target tissues
- abnormal insulin secretion
- inappopriate hepatic gluconeogenesis (overproduction of glucose).
Type 2 diabetes may also develop as a consequence of obesity.

Secondary diabetes
Three common causes of secondary diabetes are:
- physical or emotional stress, which may cause prolonged elevation in levels of the stress hormones cortisol, epinephrine, glucagon, and GH.
- pregnancy, which causes weight gain and high levels of estrogen and placental hormones
- use of adrenal corticosteroids, hormonal contraceptives, and other drugs that antagonize the effects of insulin.
Some viral infections have been implicated, such as CMV, adenovirus, rubella, and mumps.

Acute danger
Two acute metabolic complications of diabetes are diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic non-ketotic syndrome (HHNS). These life-threatening conditions require immediate medical intervention.

Chronic complications
Patients with DM also have a higher risk of various chronic complications affecting virtually all body systems. The most common chronic complications are cardiovascular disease, peripheral vascular disease, eye disease (retinopathy), kidney disease, skin disease (diabetic dermopathy), and peripheral and autonomic neuropathy.

What to look for
Patients with type 1 diabetes usually report rapidly developing symptoms, including muscle wasting and loss of subcutaneous fat.
With type 2 diabetes, symptoms are generally vague and longstanding and develop gradually. Patients generally report a family history of DM, GD, delivery of a baby weighing ≥ 4kg, severe viral infection, another endocrine disease, recent stress or trauma, or use of drugs that increase blood glucose levels. Obesity, especially in the abdominal area, is also common.

**Screening guidelines**

- Adults should be tested for diabetes every 3 years starting at age 45. Those who get a high glucose reading should have the test repeated on another day.
- People at increased risk may need to be tested earlier or more often. Higher-risk groups include anyone who is overweight or has high blood pressure, high cholesterol, or a strong family history of diabetes.
- The cutoff used for declaring someone as diabetic is a fasting plasma glucose level greater than or equal to 126 mg/dl on at least two occasions.
It takes both types

Patients with type 1 or type 2 diabetes may report symptoms related to hyperglycemia, such as:

- excessive urination (polyuria)
- excessive thirst (polydipsia)
- excessive eating (polyphagia)
- weight loss
- frequent skin infections
- fatigue
- weakness
- dry, itchy skin
- vision changes
- vaginal discomfort.

Patients with either type of diabetes may have poor skin turgor, dry mucous membranes related to dehydration, decreased peripheral pulses, cool skin temperature, and decreased reflexes. Patients in crisis with DKA may have a fruity breath odor because of increased acetone production.
What tests tell you In nonpregnant adults, a diagnosis of DM may be confirmed by:
- symptoms of diabetes and a random blood glucose level equal to or above 200 mg/dl
- a fasting plasma glucose level equal to or greater than 126 mg/dl on at least two occasions
- a blood glucose level above 200 mg/dl on the second hour of the glucose tolerance test and on at least one other occasion during a glucose tolerance test.

Three other tests may be done:
- An ophthalmologic examination may show diabetic retinopathy.
- Urinalysis shows the presence of acetone.
- Blood tests for glycosylated hemoglobin monitor the long-term effectiveness of diabetes therapy.

Goiter
A goiter is an enlargement of the thyroid gland. It isn’t caused by inflammation or neoplasm and isn’t initially associated with hyperthyroidism or hypothyroidism. This condition is commonly referred to as nontoxic goiter. It’s classified two ways:
- endemic, caused by lack of iodine in the diet
- sporadic, related to ingestion of certain drugs or food and occurring randomly.

Nontoxic goiter is most common in females, especially during adolescence, pregnancy, and menopause. At these times, the demand for thyroid hormone increases.

A toxic topic
Toxic goiter arises from long-standing nontoxic goiter and occurs in elderly people. The enlarged thyroid gland develops small rounded masses and secretes excess thyroid hormone.

How it happens
Nontoxic goiter occurs when the thyroid gland can’t secrete enough thyroid hormone to meet metabolic needs. As a result, the thyroid mass increases to compensate. This usually overcomes mild to moderate hormonal impairment. TSH levels in nontoxic goiter are generally normal. Enlargement of the gland probably results from impaired hormone production in the thyroid and depleted iodine, which increases the thyroid gland’s reaction to TSH.

Pass the iodine, please
Endemic goiter usually results from inadequate dietary intake of iodine, which leads to inadequate synthesis of thyroid hormone.
Too much of a good thing
Sporadic goiter commonly results from ingestion of large amounts of goitrogenic foods or use of goitrogenic drugs. These foods and drugs contain agents that decrease T4 production. Such foods include rutabagas, cabbage, soybeans, peanuts, peaches, peas, strawberries, spinach, and radishes. Goitrogenic drugs include: • iodides • aminosalicylic acid • lithium (Eskalith).

A closer look
• Depletion of glandular organic iodine along with impaired hormone synthesis increases the thyroid’s responsiveness to normal TSH levels.
• Resulting increases in both thyroid mass and cellular activity overcome mild impairment of hormone synthesis. Although the patient has a goiter, his metabolic function is normal.
• When the underlying disorder is severe, compensatory responses may cause both a goiter and hypothyroidism.

What to look for
A nontoxic goiter causes these signs and symptoms:
• single or multinodular, firm, irregular enlargement of the thyroid gland
• stridor • respiratory distress and dysphagia
• dizziness or syncope

Bigger isn’t always better
The enlarged thyroid gland frequently undergoes exacerbations and remissions. Fibrosis may alternate with hyperplasia, and nodules containing thyroid follicles may develop. Production of excessive amounts of thyroid hormone may lead to thyrotoxicosis.
Complications from a large retrosternal goiter mainly result from the compression and displacement of the trachea or esophagus. Thyroid cysts and hemorrhage into the cysts may increase the pressure on and compression of the surrounding tissues and structures. Large goiters may obstruct venous return, produce venous engorgement and, rarely, cause collateral circulation of the chest.
With treatment, the prognosis is good for patients with either endemic or sporadic goiter.

What tests tell you
These tests are used to diagnose nontoxic goiter and rule out other diseases with similar S&Sx:
• Serum thyroid hormone levels are usually normal. Abnormal T3, T4&TSH levels rule it out.
• Thyroid antibody titers are usually normal. Increases indicate chronic thyroiditis.
• Radioactive iodine (131I) uptake is usually normal.
• Urinalysis may show low urinary excretion of iodine.
• Radioisotope scanning identifies thyroid neoplasms.
Hyperthyroidism
When thyroid hormone is overproduced, it creates a metabolic imbalance called hyperthyroidism or thyrotoxicosis. Excess thyroid hormone can cause various thyroid disorders; Graves’ disease is the most common.

How grave is Graves’ disease?
Graves’ disease is an autoimmune disorder that causes goiter and multiple systemic changes. It occurs mostly in people ages 30 to 60, especially when their family histories include thyroid abnormalities. Only 5% of patients are younger than age 15.

Taken by storm
Thyrotoxic crisis, also known as thyroid storm, is an acute exacerbation of hyperthyroidism. It’s a medical emergency that may lead to life-threatening cardiac, hepatic, or renal failure. Inadequately treated hyperthyroidism and stressful conditions, such as surgery, infection, preeclampsia, and DKA, can lead to thyrotoxic crisis.

How it happens
In Graves’ disease, thyroid-stimulating antibodies bind to and stimulate the TSH receptors of the thyroid gland. The trigger for this autoimmune response is unclear; it may have several causes. Genetic factors may play a part; the disease tends to occur in identical twins. Immunologic factors may also be the culprit; the disease occasionally coexists with other autoimmune endocrine abnormalities, such as type 1 DM, thyroiditis, and hyperparathyroidism.
Graves’ disease is also associated with the production of several auto antibodies formed because of a defect in suppressor T-lymphocyte function.

Hyperthyroidism in hiding
In a person with latent hyperthyroidism, excessive iodine intake and, possibly, stress can cause active hyperthyroidism.

What to look for
The classic features of Graves’ disease are:
• an enlarged thyroid gland • exophthalmos (abnormal protrusion of the eye)
• nervousness • heat intolerance • weight loss despite increased appetite
• excessive sweating • diarrhea • tremors
• palpitations.
• CNS: difficulty concentrating, anxiety, excitability or nervousness.
• Cardiovascular system; arrhythmias,
• Respiratory system—dyspnea on exertion and, possibly, at rest
• GI system—anorexia, nausea, and vomiting
• Musculoskeletal system—muscle weakness, generalized or localized muscle atrophy).
• Reproductive system—menstrual abnormalities, impaired fertility, and gynecomastia.
• eyes—infrequent blinking, strabismus (eye deviation), and exophthalmos.

Recognizing hyperthyroidism
Below illustrates the common signs of an overactive thyroid.
**Touch tells you**

On palpation, the thyroid gland may feel asymmetrical, lobular, and enlarged to three or four times its normal size. The liver may also be enlarged. Hyperreflexia is also present.

**What tests tell you**

These laboratory tests confirm Graves’ disease:

- Radioimmunooassay shows increased serum T3 and T4 concentrations.
- TSH level is low in primary hyperthyroidism and elevated when excessive TSH secretion is the cause.
- Thyroid scan reveals increased uptake of 131I.

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**Treating hyperthyroidism**

In Graves’ disease, the most common hyperthyroid disorder, treatment consists of drug therapy, including radioactive iodine (131I) therapy, and surgery.

**Drug therapy**

Antithyroid drugs, such as propylthiouracil and methimazole, are used for children, young adults, pregnant women, and patients who refuse other treatments.

**Radioactive iodine therapy**

A single oral dose of 131I is the treatment of choice. This form of therapy is contraindicated during pregnancy, therefore, pregnancy should be ruled out before the initiation of treatment. Women should be cautioned to avoid pregnancy for 3 months after treatment. Radioactive iodine should be used cautiously in patients younger than age 20.

During treatment, the thyroid gland picks up the radioactive element as it would regular iodine. Subsequently, the radioactivity destroys some of the cells that normally concentrate iodine and produce thyroxin (T4), thus decreasing thyroid hormone production and normalizing thyroid size and function.

In most patients, hypermetabolic symptoms diminish within 6 to 8 weeks. However, some patients may require a second dose.

**Surgery**

Partial thyroidectomy is indicated for patients younger than age 40 who have a very large goiter and whose hyperthyroidism has repeatedly relapsed after drug therapy, for pregnant patients, and for patients allergic to 131I and other antithyroid drugs. The surgery involves removal of part of the thyroid gland, decreasing its size and capacity for hormone production.

**Preoperative preparations**

Preoperatively, the patient may receive iodide (Lugol’s solution or potassium iodide solution), antithyroid drugs, or high doses of propranolol to help prevent thyroid storm. If normal thyroid function is not achieved, surgery should be delayed and propranolol administered to decrease the risk of cardiac arrhythmias.

**Other treatments**

Therapy for hyperthyroid ophthalmopathy includes local applications of topical drugs but may require high doses of corticosteroids. A patient with severe exophthalmos that causes pressure on the optic nerve may require surgical decompression to reduce pressure on the orbital contents.

Treatment for thyrotoxic crisis includes giving an antithyroid drug, IV propranolol to block sympathetic effects, a corticosteroid to inhibit the conversion of triiodothyronine to T3, and replace depleted cortisol, and an iodide to block the release of thyroid hormones. Supportive measures include the administration of nutrients, vitamins, fluids, and sedatives.

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**Hypothyroidism**

In thyroid hormone deficiency (hypothyroidism) in adults, metabolic processes slow down. That’s because of a deficit in T3 or T4, both of which regulate metabolism. The disorder is most prevalent in women and in people with Down syndrome. Its incidence is increasing in people ages 40 to 50.

**Primary or secondary**

Hypothyroidism is classified as primary or secondary. The primary form stems from a disorder of the thyroid gland itself. The secondary form stems from a failure to stimulate normal thyroid function. This form may progress to myxedema coma, a medical emergency.

**How it happens**

Primary hypothyroidism has several possible causes:

- thyroidectomy
- inflammation from radiation therapy
- other inflammatory conditions, such as amyloidosis and sarcoidosis
• chronic autoimmune thyroiditis (Hashimoto’s disease).
Secondary hypothyroidism is caused by a failure to stimulate normal thyroid function. For example, the pituitary may fail to produce TSH (thyrotropin) or the hypothalamus may fail to produce thyrotropin-releasing hormone.
Secondary hypothyroidism may also be caused by an inability to synthesize thyroid hormones because of iodine deficiency (usually dietary) or the use of antithyroid medications.

Throughout the organization
Because insufficient synthesis of thyroid hormones affects almost every organ system in the body, signs and symptoms vary according to the organs involved as well as the duration and severity of the condition.

What to look for
The signs and symptoms of hypothyroidism may be vague and varied. Early ones include:
• energy loss  • fatigue  • forgetfulness  • sensitivity to cold
• unexplained weight gain  • constipation.
As the disease progresses, the patient may have:
• anorexia  • decreased libido  • menorrhagia (painful menstruation)
• paresthesia (numbness, prickling, or tingling)  • joint stiffness  • muscle cramping.
Other signs and symptoms include:
• CNS—psychiatric disturbances, ataxia (loss of coordination
• integumentary system—dry, flaky, inelastic skin; puffy face, hands, and feet; dry, sparse hair
• cardiovascular system—hypercholesterolemia (high cholesterol)
• reproductive system—impaired fertility
• eyes and ears—conductive or sensorineural deafness and nystagmus
• hematologic system—anemia, which may result in bleeding tendencies

Going to extremes
Severe hypothyroidism, or myxedema, is characterized by thickening of the facial features and induration of the skin, weak pulse, bradycardia, muscle weakness, sacral or peripheral edema, and delayed reflex relaxation time

What tests tell you
Primary hypothyroidism is confirmed by an elevated TSH level and low serum free T4 level.
Additional tests may be performed:
• Serum TSH levels determine whether the disorder is primary or secondary.
• Serum antithyroid antibodies show elevated levels in autoimmune thyroiditis.
• Radioisotope scanning identifies ectopic thyroid tissue.
• Skull X-ray, CT scan, and MRI locate pituitary or hypothalamic lesions that may cause secondary hypothyroidism.

### Endocrine system review

**Understanding the endocrine system**
- Hypothalamus helps control endocrine glands
- Adrenal cortex secretes mineralocorticoids, glucocorticoids, adrenal androgens, and estrogen
- Adrenal medulla produces epinephrine and norepinephrine
- Pancreas produces glucagon and insulin
- Pituitary gland secretes oxytocin and antidiuretic hormone
- Thyroid gland secretes thyroxine and triiodothyronine
- Parathyroid glands secrete parathyroid hormone

**Causes of endocrine disorders**
- Hypersecretion or hyposecretion of hormones
- Hyporesponsiveness of receptors of hormones
- Inflammation of gland
- Tumor of gland

**Endocrine disorders**
- **Addison's disease**—autoimmune disease (primary) that causes massive destruction of both adrenal glands
- **Cushing's syndrome**—typically results from excess corticotropin, which leads to hyperplasia of the adrenal cortex
- **Diabetes insipidus**—caused by deficiency of ADH
- **Diabetes mellitus**—occurs in two primary forms:
  - **type 1**—beta cells in pancreas are destroyed or suppressed; insulin isn't secreted
  - **type 2**—may be insulin resistance, overproduction of glucose, or abnormal insulin secretion
- **Goiter**—enlargement of the thyroid gland; occurs in two forms:
  - **nontoxic goiter**—thyroid gland is enlarged because it's unable to secrete enough thyroid hormone to meet metabolic needs
  - **toxic goiter**—occurs after long-standing nontoxic goiter
- **Hypothyroidism**—autoimmune disorder that overproduces thyroid hormone
- **Hyperthyroidism**—a thyroid deficiency that causes metabolic processes to slow down