CHAPTER 19 Nursing Care of Clients with Endocrine Disorders

LEARNING OUTCOMES

- Apply knowledge of normal anatomy, physiology, and assessments of the thyroid, parathyroid, adrenal, and pituitary glands when providing nursing care for clients with endocrine disorders.
- Compare and contrast the manifestations of disorders that result from hyperfunction and hypofunction of the thyroid, parathyroid, adrenal, and pituitary glands.
- Explain the nursing implications for medications prescribed to treat disorders of the thyroid and adrenal glands.
- Provide appropriate nursing care for the client before and after a subtotal thyroidectomy and an adrenalectomy.
- Use the nursing process as a framework for providing individualized care to clients with disorders of the thyroid, parathyroid, adrenal, and pituitary glands.

CLINICAL COMPETENCIES

- Assess functional health status of clients with endocrine disorders and monitor, document, and report abnormal manifestations.
- Use evidence-based research to provide appropriate teaching for self-medicating with thyroid hormone.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualized nursing interventions for clients with endocrine disorders.
- Teach clients that hormone replacement is lifelong, and how to take medications efficiently and effectively.
- Monitor respiratory function after thyroidectomy.
- Monitor for latent tetany following parathyroid removal, planned or inadvertent.
- Anticipate and recognize the effects of adrenal hormones.
- Revise plan of care as needed to provide effective interventions to promote, maintain, or restore functional health status to clients with endocrine disorders.

MEDIALINK

Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD-ROM accompanying this textbook, and on the Companion Website at http://www.prenhall.com/lemone

KEY TERMS

acromegaly, *558*
Addisonian crisis, *553*
Addison's disease, *553*
Cushing's syndrome, *548*
diabetes insipidus, *559*
euthyroid, *537*
exophthalmos, *536*
gigantism, *558*
goiter, *536*
Graves' disease, *534*
Hashimoto's thyroiditis, *543*
hyperparathyroidism, *547*
hyperthyroidism, *534*
hypoparathyroidism, *548*
hypothyroidism, *541*
myxedema, *541*
myxedema coma, *543*
pheochromocytoma, *557*
pretibial myxedema, *536*
proptosis, *536*
syndrome of inappropriate ADH secretion (SIADH), *558*
tetany, *548*
thyroid storm or crisis, *537*
thyroidectomy, *537*
thyroiditis, *536*
thyrotoxicosis, *534*
toxic multinodular goiter, *536*

The thyroid, parathyroid, adrenal, and pituitary glands are part of the endocrine system. Disorders of the structure and function of these glands alter normal hormone levels and the way body tissues use those hormones. When hormone production increases or decreases, people experience alterations in health.

Clients with disorders of the glands discussed in this chapter require nursing care for multiple problems. They often face exhausting diagnostic tests, changes in physical appearance and emotional responses, and permanent alterations in lifestyle. Nursing care is directed toward meeting physiologic needs, providing education, and ensuring psychologic support for the client and family. A holistic approach to the complex needs of clients with these endocrine disorders is an essential component of nursing care.

DISORDERS OF THE THYROID GLAND

Altered thyroid hormone (TH) production or use affects all major organ systems. In the adult, TH changes primarily affect metabolism, cardiovascular function, gastrointestinal function, and neuromuscular function. Thyroid disorders—both hyperthyroidism and hypothyroidism—are among the most common endocrine disorders.

THE CLIENT WITH HYPERTHYROIDISM

Hyperthyroidism (also called **thyrotoxicosis**) is a disorder caused by excessive delivery of TH to the peripheral tissues. Because the primary effect of TH is to increase metabolism and protein synthesis, hyperthyroidism affects all major organ systems of the body. The increase in metabolic rate and the alterations in cardiac output, peripheral blood flow, oxygen consumption, and body temperature are similar to those found in increased sympathetic nervous system activity (Porth, 2005).

The effects of hyperthyroidism are the result of increased circulating levels of TH. This hormonal excess increases the metabolic rate and heightens the sympathetic nervous system's physiologic response to stimulation. The sensitizing effect of abnormally elevated TH levels increases the cardiac rate and stroke volume. As a result, cardiac output and peripheral blood flow increase. Elevated TH levels also increase carbohydrate, protein, and lipid metabolism. Lipids are depleted, and glucose tolerance decreases. Protein degradation increases, resulting in a negative nitrogen balance. Over time, the hypermetabolic effects of excess TH result in caloric and nutritional deficiencies.

Pathophysiology and Manifestations

Hyperthyroidism results from many different factors, including autoimmune stimulation (as in Graves' disease), excess secretion of thyroid-stimulating hormone (TSH) by the pituitary gland, thyroiditis, neoplasms (such as toxic multinodular goiter), and an excessive intake of thyroid medications. The most common etiologies of hyperthyroidism are Graves' disease and toxic multinodular goiter.

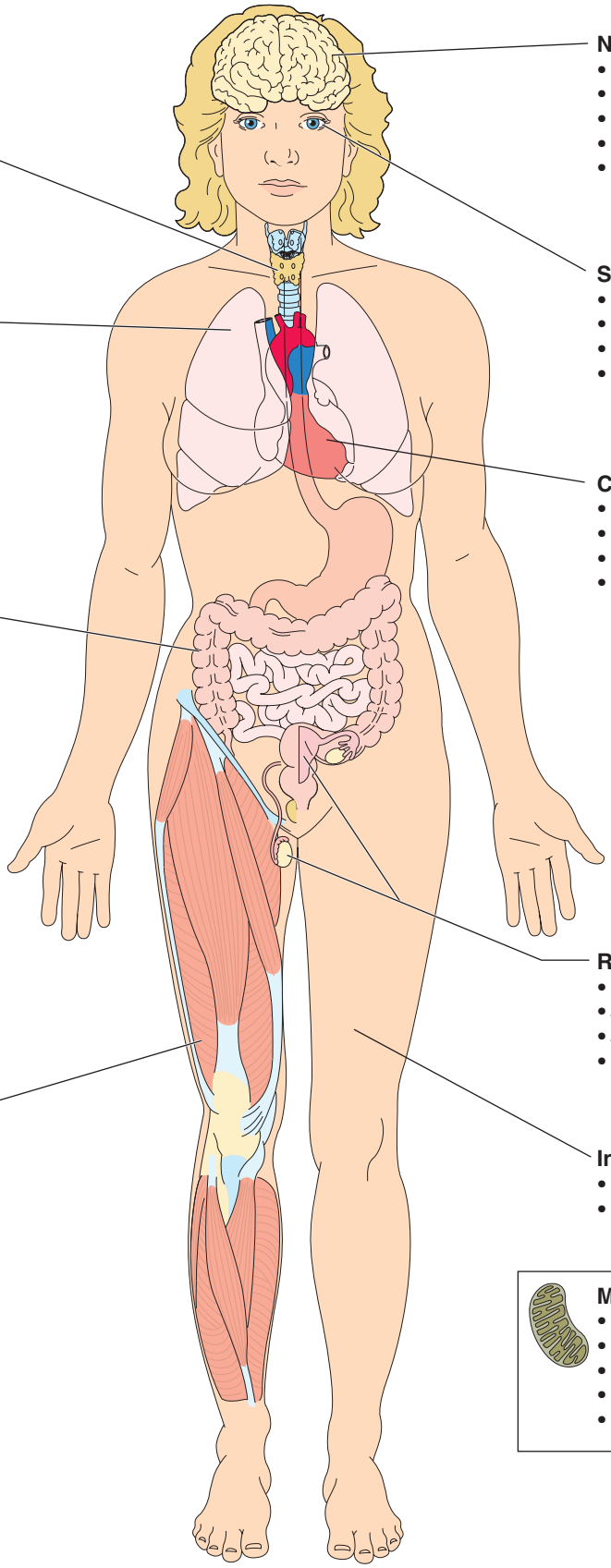
The client with hyperthyroidism typically has an increased appetite, yet loses weight and may have hypermotile bowels and diarrhea. Additional manifestations related to hypermetabolism include heat intolerance, insomnia, palpitations, and increased sweating. The skin is smooth and warm, hair may become fine, and hair loss in the scalp, eyebrow, axillary, or pubic areas of the body is common. Emotional lability is common. The *Multisystem Effects of Hyperthyroidism* are shown on the following page.

Graves' Disease

Graves' disease, the most common cause of hyperthyroidism, is an autoimmune disorder, sometimes associated with the presence of other autoimmune disorders such as myasthenia gravis and pernicious anemia (Porth, 2005). The serum of more than 80% of clients with Graves' disease has an antibody that binds to thyroid-stimulating hormone (TSH) receptors in the thyroid follicles and causes the thyroid cells to hyperfunction (Tierney et al., 2005). When this antibody binds to the TSH receptors on the thyroid gland, it stimulates hormone synthesis and secretion, enlarging the gland. The cause is unknown, but there is a hereditary link.

Graves' disease is seen five times more often in women than in men and occurs most frequently between the ages of 20 and 40. It is seen worldwide, with the incidence often correlated with the amount of iodine in the diet. Increased iodine intake (such as from radiocontrast dyes used in diagnostic tests or from ingestion of kelp tablets) has been associated with an increased frequency of hyperthyroidism.

MULTISYSTEM EFFECTS OF **Hyperthyroidism**



Endocrine
- Goiter

Respiratory
- Dyspnea

Gastrointestinal
- Nausea
- Vomiting
- Diarrhea
- Abdominal pain

Musculoskeletal
- Muscle wasting
- Weakness
- Fatigue

Neurologic
- Hand and eye tremors
- Nervousness
- Insomnia
- Emotional lability
- ↑ reflexes

Sensory
- Blurred vision
- Photophobia
- Lacrimation
- Exophthalmos (Graves' disease)

Cardiovascular
- Hypertension
- Tachycardia
- Dysrhythmias
- Palpitations

Reproductive
- Amenorrhea (female)
- ↓ fertility (female)
- ↓ libido (male)
- Impotence (male)

Integumentary
- Fine, thin hair
- Flushed, moist skin

Metabolic Processes
- Hyperthermia
- Diaphoresis
- Hunger
- Weight loss
- Fluid volume deficit

TABLE 19–1 Laboratory Findings in Hyperthyroidism

TEST	NORMAL VALUES	FINDINGS
Serum TA	Negative to 1:20	Increased
Serum TSH (sensitive assay)	2–10 mU/ml mU = microunits	Decreased in primary hyperthyroidism
Serum T_4	5 to 12 mcg/dL mcg = microgram	Increased
Serum T_3	80 to 200 ng/dL	Increased
T_3 uptake (T_3RU)	25 to 35 relative percentage	Increased
Thyroid suppression		Increased RAI uptake and T_4 levels

Clients with Graves' disease have an enlarged thyroid gland (**goiter**) and manifestations of hyperthyroidism (as shown in Table 19–1). The goiter can result from excess TSH stimulation (when the amount of circulating TH is deficient), abnormal growth-stimulating immunoglobulins, or substances that inhibit TH synthesis. A goiter may be present in hyperthyroidism or hypothyroidism.

The ophthalmopathy of Graves' disease is manifested as proptosis and visual dysfunction. **Proptosis** (forward displacement) of the eye occurs in about one-third of cases (Porth, 2005). The forward protrusion of the eyeballs (**exophthalmos**) results from an accumulation of inflammation by-products in the retroorbital tissues. Often the sclera is visible above the iris. The upper lids are often retracted, and the person has a characteristic unblinking stare (Figure 19–1 ■). Proptosis is usually bilateral, but it may involve only one eye. The client may experience blurred vision, diplopia, eye pain, lacrimation, and photophobia. The inability to close the eyelids completely over the protruding eyeballs increases the risk of corneal dryness, irritation, infection, and ulceration. Infiltration of the muscles that move the eye and of the optic nerve lead to paralysis and vision loss. The treatment of Graves' disease may stabilize these symptoms but generally does not reverse these changes in the eyes.

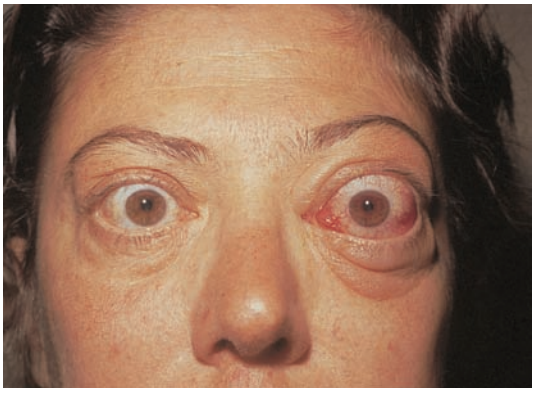


Figure 19–1 ■ Exophthalmos in a client with Graves' disease. The disease causes edema of fat deposits behind the eyes and inflammation of the extraocular muscles. The accumulating pressure forces the eyes outward from their orbits.

Source: University of Illinois, Custom Medical Stock Photo, Inc.

MEDIALINK Case Study: Hyperthyroidism

A rare, characteristic dermopathy of Graves' disease is **pretibial myxedema**. Plaques and nodules develop bilaterally over the shins and dorsal surface of the feet. These plaques are edematous, erythematous, and sometimes hyperpigmented. Like the ophthalmopathy, the skin changes often persist despite successful treatment of Graves' disease (Levin & Greer, 2001).

Other manifestations include fatigue, difficulty sleeping, hand tremors, and changes in menstruation ranging from decreased flow to amenorrhea. Older clients may present with atrial fibrillation, angina, or congestive heart failure.

Toxic Multinodular Goiter

Toxic multinodular goiter (Figure 19–2 ■) is a tumor characterized by small, discrete, independently functioning nodules in the thyroid gland tissue that secrete excessive amounts of TH. It is not known how these nodules grow or become independent, but a genetic mutation of follicle cells is suspected. Elevated TH levels result in manifestations of hyperthyroidism; however, they are slower to develop and neither ophthalmopathy nor dermopathy develop (McCance & Huether, 2002). The client with this type of hyperthyroidism is usually a woman in her 60s or 70s who has had a goiter for a number of years.

Excess TSH Stimulation

Overproduction of TSH by the pituitary usually stimulates the thyroid gland to produce excess TH. The elevation in TSH secretion often results from a pituitary adenoma. This secondary form of hyperthyroidism is rare.

Thyroiditis

Thyroiditis (inflammation of the thyroid gland) is most often the result of a viral infection of the thyroid gland. The symptoms of thyroiditis are those of acute inflammation and the effects of increased TH. Thyroiditis is an acute disorder that may become

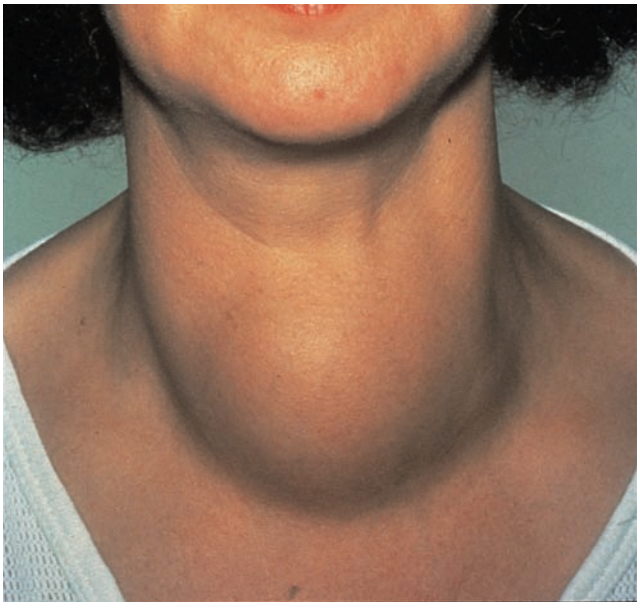


Figure 19–2 ■ Toxic multinodular goiter. The formation and growth of numerous nodules in the thyroid gland cause the characteristic massive enlargement of the neck.

Source: Custom Medical Stock Photo, Inc.

chronic, resulting in a hypothyroid state as repeated infections destroy gland tissue. See the discussion of Hashimoto's thyroiditis later in this chapter.

Thyroid Crisis

Thyroid storm (also called **thyroid crisis**) is an extreme state of hyperthyroidism that is rare today because of improved diagnosis and treatment methods (Porth, 2005). When it does occur, those affected are usually people with untreated hyperthyroidism (most often Graves' disease) and people with hyperthyroidism who have experienced a stressor, such as an infection, trauma, untreated diabetic ketoacidosis, or manipulation of the thyroid gland during surgery. Thyroid crisis is a life-threatening condition.

The rapid increase in metabolic rate that results from the excessive TH causes the manifestations of thyroid storm. The manifestations include hyperthermia, with body temperatures ranging from 102° to 106°F (39° to 41°C); tachycardia; systolic hypertension; and gastrointestinal symptoms (abdominal pain, vomiting, diarrhea). Agitation, restlessness, and tremors are common, progressing to confusion, psychosis, delirium, and seizures. The mortality rate is high. Rapid treatment of thyroid storm is essential to preserve life. Treatment includes cooling without aspirin (which increases free TH) or inducing shivering; replacing fluids, glucose, and electrolytes; relieving respiratory distress; stabilizing cardiovascular function; and reducing TH synthesis and secretion.

INTERDISCIPLINARY CARE

Treatment of hyperthyroidism focuses on reducing the production of TH by the thyroid gland, thus establishing a **euthyroid** (normal thyroid) state, and preventing or treating complications. Depending on the client's age and physical status, either medications, radioactive iodine therapy, or surgery may be used.

Diagnosis

Hyperthyroidism is diagnosed according to the manifestations of the specific disorders causing excessive TH, and by diagnostic test results. Elevated levels of TH (both T_3 and T_4) and increased radioactive iodine (RAI) uptake are diagnostic criteria of hyperthyroidism. Laboratory findings in hyperthyroidism were shown earlier in Table 19–1.

The following diagnostic tests may be ordered:

- *TA test.* Serum thyroid antibodies (TA) are measured to determine whether a thyroid autoimmune disease is causing the client's symptoms. TA is elevated in Graves' disease.
- *TSH test (sensitive assay).* Serum TSH levels are measured and compared with thyroxine (T_4) levels to differentiate pituitary from thyroid dysfunction. The best indicator of primary hyperthyroidism (such as in Graves' disease) is suppression of TSH below 0.1 mcg/mL. When the sensitive TSH is not suppressed, the hyperthyroidism is caused by a TSH-secreting pituitary tumor.
- *T_4 test.* Serum thyroxine levels are measured to determine TH concentration and to test thyroid gland function. T_4 levels are elevated in hyperthyroidism and in acute thyroiditis.
- *T_3 test.* Serum triiodothyronine (T_3) is measured by radioimmunoassay (T_3RIA), which measures bound and free forms of this hormone. This test is effective for the diagnosis of hyperthyroidism. T_3 levels may also be elevated in thyroiditis.
- *T_3 uptake test.* T_3 uptake (T_3RU) is measured by an *in vitro* test in which the client's blood is mixed with radioactive T_3; the results are elevated in hyperthyroidism.
- *RAI uptake test.* A radioactive iodine uptake test (thyroid scan) measures the absorption of ^{131}I or ^{123}I by the thyroid gland. A calculated dose of radioactive iodine is given orally or intravenously, and the thyroid is then scanned (often after 24 hours). The distribution of radioactivity in the gland is recorded (increased uptake of radioactive iodine is seen in Graves' disease). In addition, the scan reveals the size and shape of the gland.
- *Thyroid suppression test.* RAI and T_4 levels are measured first. The client then takes TH for 7 to 10 days, after which the tests are repeated. Failure of hormone therapy to suppress RAI and T_4 indicates hyperthyroidism.

Medications

Hyperthyroidism is treated by administering antithyroid medications that reduce TH production. Because these drugs do not affect the release or activity of hormone that is already formed, therapeutic effects may not be seen for several weeks. To rapidly decrease the cardiovascular symptoms associated with hyperthyroidism, propanolol (Inderal), a beta-blocker, is part of initial treatment. (See Chapter 31 ∞ for information on beta-blockers.) Some commonly prescribed drugs, their actions, and nursing implications are shown in the Medication Administration box on page 538.

Radioactive Iodine Therapy

Because the thyroid gland takes up iodine in any form, radioactive iodine (^{131}I) concentrates in the thyroid gland and damages or destroys thyroid cells so that they produce less TH. Radioactive iodine is given orally. Results typically occur in 6 to 8 weeks. In most instances, the client is not hospitalized during treatment and does not require radiation precautions. This type of therapy is contraindicated in pregnant women because radioactive iodine crosses the placenta and can have negative effects on the developing fetal thyroid gland. Because the amount of gland destroyed is not readily controllable, the client may become hypothyroid and require lifelong TH replacement. Adverse reactions include thyroiditis and cardiac instability due to liberation of stored thyroid hormone in the gland (Holcomb, 2006).

Surgery

Some hyperthyroid clients have such enlarged thyroid glands that pressure on the esophagus or trachea causes breathing or swallowing problems. In these cases, removal of all or part of the gland is indicated. A *subtotal thyroidectomy* is usually performed. This procedure leaves enough of the gland in place to produce an adequate amount of TH. A total **thyroidectomy** is performed to treat cancer of the thyroid; the client then requires lifelong hormone replacement (Kumrow & Dahlen, 2002).

MEDICATION ADMINISTRATION Hyperthyroidism

IODINE SOURCES

Strong Iodine Solution (Lugol's Solution)
Potassium iodide (SSKI, Thyro-Block, Pima)

Large doses of iodine for a short term inhibit TH synthesis and release. Iodine also makes the hyperplastic thyroid less vascular prior to surgery and hastens the ability of other antithyroid drugs to reduce natural hormone output. It is also used in thyroid storm. Potassium iodide is useful to protect the thyroid in radiation emergencies, blocking the uptake of radioactive iodine from nuclear fallout (Lehne, 2004).

Nursing Responsibilities

- Assess for hypersensitivity to iodine before giving medication; for example, ask client about allergies to shellfish.
- Dilute liquid iodine sources in water or orange juice to disguise bitter taste.
- Monitor for increased bleeding tendencies if the client is also taking anticoagulants; iodine increases their effect.

Health Education for the Client and Family

- The maximum effect of iodine in large doses usually occurs in 10 to 15 days.
- Long-term iodine therapy is not effective in controlling hyperthyroidism.

ANTITHYROID DRUGS

Methimazole (Tapazole)
Propylthiouracil (PTU, Propyl-Thyracil)

Antithyroid drugs inhibit TH production. They do not affect already formed hormones; thus, several weeks may elapse before the client experiences therapeutic effects. Methimazole can be taken in one daily dose; propylthiouracil must be taken in 3 doses daily; methimazole crosses the placenta and cannot be taken during pregnancy (Lehne, 2004).

Nursing Responsibilities

- Monitor for side effects: agranulocytosis, hypothyroidism, pruritus rash, elevated temperature, (for iodides) periorbital edema, anorexia, loss of taste, changes in menstruation.
- Administer drugs at the same time each day with meals to maintain stable blood levels.
- Monitor for symptoms of hypothyroidism: fatigue, weight gain.

Health Education for the Client and Family

- Watch for unusual bleeding, redness, swelling, nausea, loss of taste, or epigastric pain. Report any such symptoms to the physician.
- If you are also taking warfarin, report any signs of bleeding.
- If you are taking lithium, be aware of symptoms of hypothyroidism.
- It may take up to 12 weeks before you experience the full effects of the drugs. Take the medication regularly and exactly as prescribed. Do not discontinue abruptly.

Before surgery, the client should be in as nearly a euthyroid state as possible. The client may be given antithyroid drugs to reduce hormone levels and iodine preparations to decrease the vascularity and size of the gland (which also reduces the risk of hemorrhage during and after surgery). Nursing care of the client having a subtotal thyroidectomy is discussed in the box on the following page.

NURSING CARE

Health Promotion

Although hyperthyroidism is not preventable, it is important to teach clients the importance of regular healthcare provider visits and medication intake.

Assessment

The following data are collected through the health history and physical examination (see Chapter 18 ∞). Further focused assessments are described with nursing interventions.

- *Health history:* Other diseases, family history of thyroid disease, when symptoms began, severity of symptoms, intake of thyroid medications, menstrual history, changes in weight, bowel elimination.
- *Physical assessment:* Muscle strength, tremors, vital signs, cardiovascular and peripheral vascular systems, integument, size of thyroid, presence of bruit over thyroid, eyes and vision.

Nursing Diagnoses and Interventions

In planning and implementing nursing care for the client with hyperthyroidism, the nurse considers the client's responses to the systemic effects of the disorder. Although each client may have different needs, nursing diagnoses discussed in this section focus on the most common problems: cardiovascular problems, visual deficits, altered nutrition, and body image disturbance. See the accompanying Nursing Care Plan on page 540.

Risk for Decreased Cardiac Output

The client with hyperthyroidism is at risk for alterations in cardiac output. Excess TH directly affects the heart, resulting in increased rate and stroke volume. Increases in the metabolic demands and oxygen requirements of peripheral tissues increase the demands on the heart, and systolic hypertension, angina, arrhythmias, or cardiac failure may occur. The client often has palpitations and shortness of breath and is easily fatigued. The risk of complications is greater in clients with preexisting cardiovascular disorders.

- Monitor blood pressure, pulse rate and rhythm, respiratory rate, and breath sounds. Assess for peripheral edema, jugular vein distention, and increased activity intolerance. *Increased TH increases cardiac rate, stroke volume, and tissue demand for oxygen, causing stress on the heart. This may result in hypertension, arrhythmias, tachycardia, and congestive heart failure.*

NURSING CARE OF THE CLIENT HAVING A Subtotal Thyroidectomy

PREOPERATIVE CARE

- Administer ordered antithyroid medications and iodine preparations, and monitor their effects. *Antithyroid drugs are given before surgery to promote a euthyroid state. Iodine preparations are given to the client before surgery to decrease vascularity of the gland, thereby decreasing the risk of hemorrhage*.
- Teach the client to support the neck by placing both hands behind the neck when sitting up in bed, while moving about, and while coughing. *Placing the hands behind the neck provides support for the suture line.*
- Answer questions, and allow time for the client to verbalize concerns. *Because the incision is made at the base of the throat, clients (especially women) are often concerned about their appearance after surgery. Explain that the scar will eventually be only a thin line and that jewelry or scarves may be used to cover the scar*.
- Teach the client to expect hoarseness due to generalized swelling at the suture line. *This is expected to diminish with healing and is not caused by laryngeal nerve damage*.

POSTOPERATIVE CARE

- Provide comfort measures: Administer analgesic pain medications as ordered, and monitor their effectiveness; place the client in a semi-Fowler's position after recovery from anesthesia; support head and neck with pillows. *Analgesic medications reduce the perception of pain and reduce physical stress during the postoperative period. Positioning the client in a semi-Fowler's position and supporting the head and neck decrease strain on the suture line*.
- Perform focused assessments to monitor for complications:
  a. Hemorrhage. Assess dressing (if present) and the area under the client's neck and shoulders for drainage. Monitor blood pressure and pulse for symptoms of hypovolemic shock. Assess tightness of dressing (if present). *The vascularity of the gland increases the risk of hemorrhage. The location of the incision and the position of the client may cause the drainage to run back and under the client. The danger of hemorrhage is greatest in the first 12 to 24 hours after surgery.*
  b. Respiratory distress. Assess respiratory rate, rhythm, depth, and effort. Maintain humidification as ordered. Assist the client with coughing and deep breathing. Have suction equipment, oxygen, and a tracheostomy set available for immediate use. *Respiratory distress may result from hemorrhage and edema, which may compress the trachea; from tetany and laryngeal spasms resulting from decreased hormones due to removal or damage to the parathyroid glands; and from damage to the laryngeal nerve, causing spasms of the vocal cords. Stridor is heard in acute obstructions. This is a high-pitched, squeaky sound and is a sign of airway obstruction. Equipment must be immediately available if the client experiences respiratory distress that requires interventions and treatment*.
  c. Laryngeal nerve damage. Assess for the ability to speak aloud, noting quality and tone of voice. *The location of the laryngeal nerve increases the risk of damage during thyroid surgery. Although hoarseness may be due to edema or the endotracheal tube used during surgery and will subside, permanent hoarseness or loss of vocal volume is a potential danger*.
  d. Tetany. Assess for signs of latent tetany due to calcium deficiency, including tingling of toes, fingers, and lips; muscular twitches; positive Chvostek's and Trousseau's signs; and decreased serum calcium levels. Serum calcium levels will be monitored in the postoperative period. Keep calcium gluconate or calcium chloride available for immediate intravenous use, if necessary. *The parathyroid glands are located in and near the thyroid gland; surgery of the thyroid gland may injure or remove parathyroid glands, resulting in hypocalcemia and tetany. Tetany may occur in 1 to 7 days after thyroidectomy*.

- Suggest keeping the environment as cool and free of distraction as possible. Decrease stress by explaining interventions and teaching relaxation procedures. *A physically comfortable and psychologically calm environment can reduce stimuli and stressors. Stress increases circulating catecholamines, which further increase cardiac workload*.
- Encourage the client to balance activity with rest periods. *Rest periods decrease energy expenditure and tissue requirements for oxygen, decreasing demands on the heart by decreasing cardiac workload*.

Disturbed Sensory Perception: Visual

Visual changes that occur in clients with hyperthyroidism include difficulty in focusing, diplopia (double vision), or visual loss. If the client is unable to close the eyelids because of exophthalmos, the risk of corneal dryness with resultant infection or injury increases. Visual deficits may also result from pressure on the optic nerve from retro-orbital edema and the shortening of eye muscles. Although treatment of hyperthyroidism may stop the progression of eye changes, not all symptoms are reversible.

- Monitor visual acuity, photophobia, integrity of the cornea, and lid closure. *The cornea is at risk for dryness, injury, conjunctivitis, and corneal infections. Injury and infection of the cornea can result in further loss of visual acuity*.
- Teach measures for protecting the eye from injury and maintaining visual acuity:
  - Use tinted glasses or shields as protection.
  - Use artificial tears to moisten the eyes.
  - Use cool, moist compresses to relieve irritation
  - Cover or tape eyelids shut at night if they do not close.
  - Elevate the head of the bed to 45 degrees to promote periorbital fluid decrease
  - Promptly report any pain or changes in vision.

NURSING CARE PLAN A Client with Graves' Disease

Mrs. Juanita Manuel is a 33-year-old mother of four small children. She is a second-year student at the local community college, within one semester of completing the requirements for an associate degree in child care. For the past 3 months, Juanita has been constantly hungry and has eaten more than usual, but she has still lost 15 lb (6.8 kg). She has repeated bouts of diarrhea and often feels nauseated. Her hands shake, she can feel her heart beating rapidly, and she finds herself laughing or crying for no apparent reason.

Mrs. Manuel makes an appointment with her family physician. The nurse at the office completes a health history and physical assessment. When asked how she has been feeling, Mrs. Manuel replies, "Well, I don't know what's wrong with me—but I keep losing weight and I cry at the drop of a hat. I am also just so hot all the time, and I've never had that problem before. I hope I find out what's wrong and it's nothing serious."

ASSESSMENT

The health history indicates that although her appetite has increased, Mrs. Manuel has lost 15 lb (6.8 kg). She states that she has had diarrhea, nausea, palpitations, heat intolerance, and mood changes. Physical assessment findings include the following: T 101°F (38.3°C), P 110, R 24, and BP 162/86. Her skin is moist and warm, her hair thin and fine. She has visible tremors in her hands. Her eyeballs protrude, and she is unable to close her eyelids completely. Her thyroid is enlarged and palpable. Diagnostic tests reveal the following abnormal results: T_3, 350 g/dL (normal range: 80 to 200 ng/dL), T_4, 15.1 mg/dL (normal range: 5 to 12 mg/dL). A thyroid scan demonstrates an enlarged thyroid with increased iodine uptake. After the medical diagnosis of Graves' disease is made, Mrs. Manuel is started on the antithyroid medication propylthiouracil, 150 mg orally every 8 hours.

DIAGNOSES

- *Risk for Imbalanced Nutrition: Less than Body Requirements* related to weight loss of 15 lb (6.8 kg), with present weight 10% less than normal for height
- *Diarrhea* related to increased peristalsis as evidenced by 8 to 10 liquid stools per day
- *Risk for Disturbed Sensory Perception: Visual* related to an inability to close the eyelids completely
- *Anxiety* related to a lack of knowledge about disease process

EXPECTED OUTCOMES

- Gain at least 1 lb (0.45 kg) every 2 weeks.
- Regain normal bowel elimination patterns.
- Maintain normal vision (with no evidence of corneal damage) and verbalize measures to protect her eyes.
- Verbalize medical treatment and self-care needs.
- Verbalize a decrease in anxiety.

PLANNING AND IMPLEMENTATION

- Request that she keep a record of daily weight.
- Discuss adopting a high-kilocalorie diet. Identify food likes and dislikes, as well as foods that increase diarrhea, before instituting a plan to increase food intake.
- Request that she keep a stool chart, noting the time, type, and precipitating factors for diarrhea stools. Teach comfort measures for irritated anal area (clean washcloth and soap, nonirritating ointment).
- Teach how to apply eyedrops (artificial tears).
- Explain the need to elevate the head of the bed to 45 degrees at night, and tape eye shields over eyes before sleep.
- Teach about Graves' disease, the medication's effects and side effects, and the need for continued medical care.

EVALUATION

By her next office visit, Mrs. Manuel has gained 1 lb (0.45 kg) and has discussed her dietary needs with the nurse and her husband. She is having diarrhea less often. She has safely applied the eyedrops and states that she uses the eye shields and elevates the head of her bed at night. The office nurse reviewed the written and verbal information about Graves' disease and the medication prescribed. Mrs. Manuel verbalizes her understanding, stating, "I'll always take my medicine—I never want to feel like that again!" She also says that she feels much less anxious now that she understands what has happened.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the pathophysiologic basis for Mrs. Manuel's abnormal vital signs?
2. What is the rationale for having the client with exophthalmos elevate the head of the bed at night?
3. Outline a teaching plan that could be given to clients for home care following a subtotal thyroidectomy.

See Evaluating Your Response in Appendix C.

PRACTICE ALERT

Teach the client to cover or tape the eyelids shut at night if they do not close and to sleep with the head of the bed elevated.

The measures outlined decrease the risk of injury, provide comfort, decrease periorbital edema that can further compromise vision, and ensure immediate care for problems, thereby minimizing the risk of further visual loss.

Imbalanced Nutrition: Less than Body Requirements

The hypermetabolic state that occurs in hyperthyroidism causes gastrointestinal hypermotility, with nausea, vomiting, diarrhea, and abdominal pain. Although the client may have an increased appetite and eat more than usual, weight loss continues.

- Ask the client to weigh daily (at the same time each day), and keep a record of results. *The inability to meet metabolic demands results in loss of body weight. Regular monitoring detects continued weight loss.*
- In collaboration with a dietitian, teach the client the need for a diet high in carbohydrates and protein and including between-meal snacks. Six small meals a day may be more desirable than three large meals. Caloric intake may need to be increased to 4000 kcal/day if weight loss exceeds 10% to 17% for height and frame. *Increased nutrients as part of a well-balanced diet are necessary to meet metabolic*

demands. Clients are often better able to increase food intake by eating frequent, small meals. A 1-lb weight gain requires approximately 3500 extra kilocalories.

- Monitor nutritional status through results of laboratory data. Serum albumin, transferrin, and total lymphocyte counts are commonly lower than normal in nutritional deficits. *A negative nitrogen balance signifies a catabolic state in which protein is lost and metabolic demands are not being met.*

Disturbed Body Image

Physical changes common in hyperthyroidism include exophthalmos, goiter, tremors, hair loss, increased perspiration, loss of strength, fatigue, weight loss, and changes in reproductive and sexual function (amenorrhea in women, impotence in men, and increased libido in both men and women). In addition, the client often has mood changes and insomnia and is constantly nervous and anxious. There may even be periods of psychosis. These changes are frightening not only for the client but also for family members.

- Establish a trusting relationship; encourage the client to verbalize feelings about self and to ask questions about the illness and treatment. Provide reliable information and clarify misconceptions. *Establishing trust facilitates open sharing of feelings and perceptions.*

Community-Based Care

Clients with hyperthyroidism primarily provide self-care at home. Teaching is individualized to meet the client's needs. Address the following topics:

- The client taking oral medications must understand the need for lifelong treatment.
- The client who has a thyroidectomy requires information about postoperative wound care.
- The client having radioactive iodine therapy needs to know the symptoms of hypothyroidism.
- Depending on the age of the client and the support systems available, referral to community healthcare agencies may be necessary.
- In addition, suggest the following resources which are accessible via the Internet:
  - American Thyroid Association
  - Thyroid Foundation of Canada
  - Endocrine Society.

THE CLIENT WITH HYPOTHYROIDISM

Hypothyroidism is a disorder that results when the thyroid gland produces an insufficient amount of TH. Because a decrease in TH levels decreases metabolic rate and heat production, hypothyroidism affects all body systems (see the *Multisystem Effects of Hypothyroidism* on page 542). Hypothyroidism is common in women between ages 30 and 60; the incidence rises after age 50. However, the disorder can occur at any stage of life. Careful evaluation of symptoms is important in the older adult because manifestations of hypothyroidism are often thought to be the result of aging instead of a pathologic process.

The hypothyroid state in adults is sometimes called **myxedema**. The term reflects the characteristic accumulation of nonpitting edema in the connective tissues throughout the body. The edema is the result of water retention in mucoprotein (hydrophilic proteoglycans) deposits in the interstitial spaces. The face of a client with myxedema appears puffy, the tongue is enlarged, and the voice is hoarse and husky (Porth, 2005).

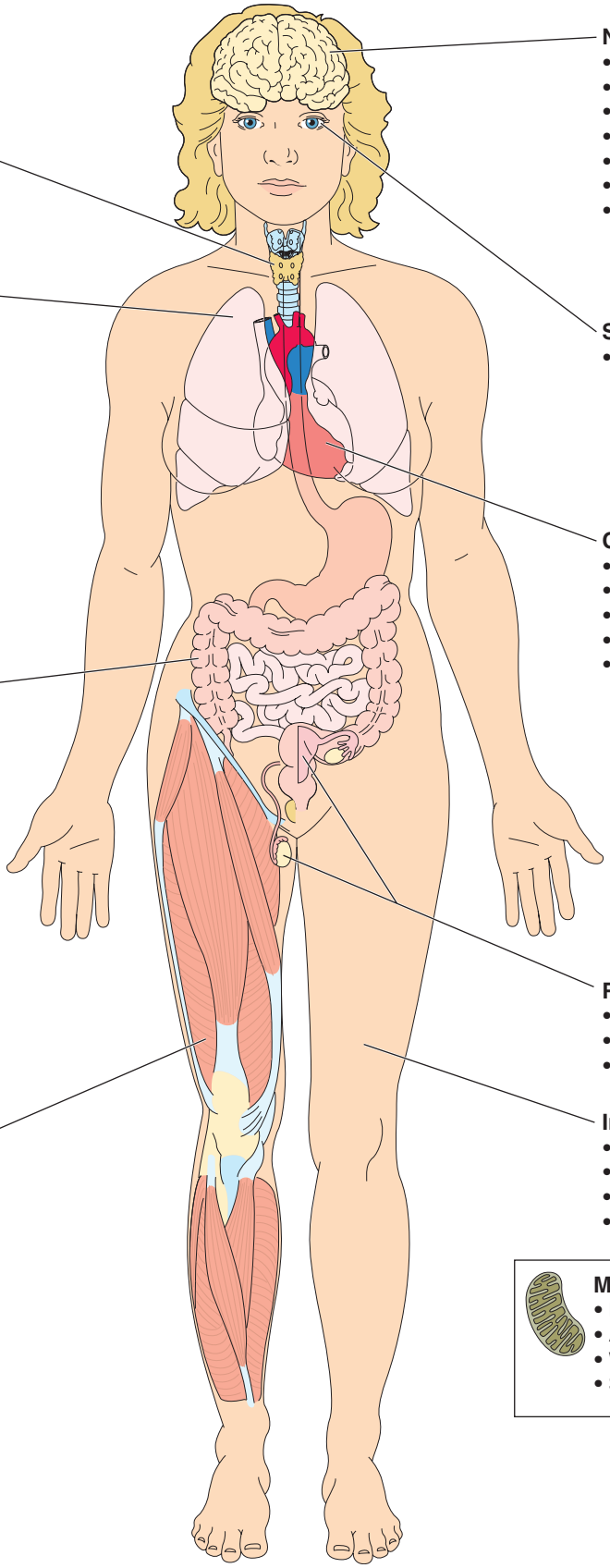
Pathophysiology and Manifestations

Hypothyroidism may be either primary or secondary. Primary hypothyroidism (which is more common) may be caused by congenital defects in the gland, loss of thyroid tissue following treatment for hyperthyroidism with surgery or radiation, antithyroid medications, thyroiditis, or endemic iodine deficiency. The cardiac drug amiodarone (Cordarone), which contains 75 mg of iodine per 200 mg tablet, is increasingly being implicated in causing thyroid problems (Porth, 2005). Clofibrate, estrogens, methadone, amiodarone, and birth control pills increase T_4 measurement; anabolic steroids, androgens, lithium, phenytoin, propanolol, interferon alpha, and interleukin-2 decrease T_4 measurement in thyroid tests. Of course, the drugs propylthiouracil and methimazole, which are used to treat hyperthyroidism, decrease T_4 measurement (Medline Plus, 2006). Secondary hypothyroidism may result from pituitary TSH deficiency or peripheral resistance to thyroid hormones. Hypothyroidism has a slow onset, with manifestations occurring over months or even years. With treatment, the mental and physical symptoms rapidly reverse in clients of all ages.

When TH production decreases, the thyroid gland enlarges in a compensatory attempt to produce more hormone. The goiter that results is usually a simple or nontoxic form. People living in certain areas of the world where the soil is deficient in iodine, the substance necessary for TH synthesis and secretion, are more prone to become hypothyroid and develop simple goiter. (Iodine deficiency is discussed later.) Geriatric clients have a decrease in T_4 production of approximately 30% but serum levels are usually maintained because of the age-related decrease in T_4 degradation (Weissel, 2006).

Hypothyroid clients characteristically have the manifestations of goiter, fluid retention and edema, decreased appetite, weight gain, constipation, dry skin, dyspnea, pallor, hoarseness, and muscle stiffness. Many clients have a decreased sense of taste and smell, menstrual disorders, anemias, and cardiac enlargement. The pulse is typically slow. Deficient amounts of TH cause abnormalities in lipid metabolism, with elevated serum cholesterol and triglyceride levels. As a result, the client is at increased risk for atherosclerosis and cardiac disorders. Decreased renal blood flow and glomerular filtration rate reduce the kidney's ability to excrete water, which may cause hyponatremia. Sleep apnea is more common in clients with hypothyroidism. Factors that result in decreased TH (in addition to those described) include iodine deficiency and Hashimoto's thyroiditis. A severe state of hypothyroidism is

MULTISYSTEM EFFECTS OF **Hypothyroidism**



Endocrine
- Goiter

Respiratory
- Pleural effusion

Gastrointestinal
- Constipation

Musculoskeletal
- Muscle stiffness
- Weakness
- Fatigue

Neurologic
- Hand and foot paresthesias
- Lethargy
- Somnolence
- Confusion
- ↓ reflexes
- Slow speech
- Memory impairment

Sensory
- Periorbital edema

Cardiovascular
- Hypotension
- Bradycardia
- Dysrhythmias
- Enlarged heart
- Anemia

Reproductive
- Menorrhagia (female)
- Infertility (female)
- ↓ libido (male)

Integumentary
- Hair loss
- Brittle nails
- Coarse, dry skin
- Nonpitting edema

Metabolic Processes
- Hypothermia
- Anorexia
- Weight gain
- Systemic edema

called *myxedema coma* and is characterized by a shallow edema especially around the eyes, hands, and feet.

Iodine Deficiency

Iodine is necessary for TH synthesis and secretion. Iodine deficiency may result from certain goitrogenic drugs (which block TH synthesis); lithium carbonate, used to treat bipolar mental disorders; and antithyroid drugs. Goitrogenic compounds in foods such as turnips, rutabagas, and soybeans may also block TH synthesis if consumed in sufficient quantities. In areas of the world where the soil is deficient in iodine, dietary intake of iodine may be inadequate. However, the use of iodized salt has reduced this risk in the United States.

Hashimoto's Thyroiditis

Hashimoto's thyroiditis is the most common cause of goiter and primary hypothyroidism in adults and children. In this autoimmune disorder, antibodies develop that destroy thyroid tissue. Functional thyroid tissue is replaced with fibrous tissue, and TH levels decrease. In addition, decreasing levels of TH in the early stages of the disease prompt the gland to enlarge to compensate, causing a goiter. However, as the disease progresses, the thyroid gland becomes smaller. This disorder is more common in women and has a familial link.

Myxedema Coma

Myxedema coma is a life-threatening complication of long-standing, untreated hypothyroidism usually triggered by an acute illness or trauma (Tierney et al., 2005). It is characterized by severe metabolic disorders (hyponatremia, hypoglycemia, lactic acidosis), hypothermia, cardiovascular collapse, impaired mentation, and coma. Myxedema coma, although rare, most commonly occurs during the winter months in older women with chronic hypothyroidism (Porth, 2005).

Myxedema coma may be precipitated by trauma, infection, failure to take thyroid replacement medications, the use of central nervous system depressants, and exposure to cold temperatures (Porth, 2005). The treatment of myxedema coma addresses the precipitating factors and manifestations and involves maintaining a patent airway; maintaining fluid, electrolyte, and acid–base balance; maintaining cardiovascular status; increasing body temperature; and increasing TH levels. If untreated, the mortality rate is high (Tierney et al., 2005).

INTERDISCIPLINARY CARE

The treatment of the client with hypothyroidism focuses on diagnosis, prevention or treatment of complications, and replacement of the deficient TH. With early and continued treatment, both appearance and mental function return to normal.

Diagnosis

Hypothyroidism is diagnosed by the clinical manifestations and by a decrease in TH, especially T_4. See Table 19–2. TSH concentration often is increased, because the negative hormonal feedback from TH is lost. The same laboratory and diagnostic tests used to diagnose hyperthyroidism are also used to diagnose hypothyroidism, with opposite results in most cases.

TABLE 19–2 Laboratory Findings in Hypothyroidism

TEST	NORMAL VALUES	FINDINGS
Serum TA	None to 1:20	Normal
Serum TSH	2–10 mU/ml mU = microunit	Increased in primary hypothyroidism
Serum T_4	5 to 12 mcg/dL	Decreased
Serum T_3	80 to 200 ng/dL	Decreased
T_3 uptake (T_3RU)	25 to 35 relative percentage	Decreased
Thyroid suppression		No change in RAI uptake or T_4 levels

Medications

Hypothyroidism is treated with medications that replace TH. Levothyroxine (thyroxine, T_4) is the treatment of choice (Tierney et al., 2005). Medications commonly used to treat hypothyroidism and their nursing implications are shown in the Medication Administration box on page 544. In the geriatric clients, an age-related decrease in serum albumin and renal excretion can increase the amount of available drug and cause an exaggerated pharmacologic effect. Therefore, the older client may require less thyroid medication than a younger client.

Surgery

If the hypothyroid client has a goiter large enough to cause respiratory difficulties or dysphagia, a subtotal thyroidectomy may be performed (see page 539).

NURSING CARE

Health Promotion

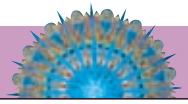
One of the most critical factors in preventing hypothyroidism is education of the public about the necessity of an adequate dietary intake of iodine. The use of iodized salt meets the requirements for hormone production. It is important to teach clients the importance of regular healthcare provider visits and medication intake. See the Nursing Research box on page 545.

Assessment

Collect the following data through the health history and physical examination (see Chapter 18 ∞). Further focused assessments are described with nursing interventions below. When assessing the older client, be aware of normal changes with aging, outlined in the box on page 545.

- *Health history:* Pituitary diseases, when symptoms began, severity of symptoms, treatment of hyperthyroidism with medications or radioactive iodine, thyroid surgery, treatment of head or neck cancer with radiation, diet, use of iodized salt, bowel elimination, respiratory difficulties.
- *Physical assessment:* Muscle strength, deep tendon reflexes, vital signs, cardiovascular and peripheral vascular systems, integument, thyroid gland, weight.

MEDICATION ADMINISTRATION Hypothyroidism



THYROID PREPARATIONS

Levothyroxine sodium (T_4) (Levoxyl, Levothroid, Synthroid)
Liothyronine sodium (T_3) (Cytomel)
Liotrix (T_3–T_4) (Euthyroid, Thyrolar)

Thyroid preparations increase blood levels of TH, thus raising the client's metabolic rate. As a result, cardiac output, oxygen consumption, and body temperature increase. Levothyroxine speeds the elimination of vitamin K–dependent clotting factors enhancing the effects of warfarin (Coumadin). Patients are at increased risk of bleeding if warfarin dosages are appropriately reduced (Lehne, 2004). The dosage depends on the drug chosen and the client's degree of thyroid dysfunction, sensitivity to TH, age, body size, and health. The older adult may require lower doses.

Nursing Responsibilities

- Give 1 hour before meals or 2 hours after meals for best absorption.
- Thyroid preparations potentiate the effect of anticoagulant drugs. If the client is also receiving an anticoagulant, monitor for bruising, bleeding gums, and blood in the urine.
- Thyroid medications potentiate the effect of digitalis. If the client is also receiving a digitalis preparation, monitor for signs of digitalis toxicity.
- Monitor for symptoms of coronary insufficiency: chest pain, dyspnea, tachycardia.
- If the client has insulin-dependent diabetes, monitor the effects of insulin. The effect of the insulin may change as thyroid function increases.
- During dose adjustment, take pulse before administering drug. Report pulse >100.

Health Education for the Client and Family

- Do not substitute brands of drugs or use generic equivalents without the physician's approval.
- The medications must be taken for the rest of one's life.
- Report symptoms of excess thyroid hormone to the physician: excess weight loss, palpitations, leg cramps, nervousness, or insomnia.
- If you have diabetes and use insulin, monitor blood glucose levels closely; the thyroid medications may alter the amount of insulin required.
- Thyroid preparations increase the risk of iodine toxicity. Do not use iodized salt or over-the-counter drugs containing iodine.
- If you are also taking an anticoagulant, report any signs of bleeding.
- Report any changes in menstrual periods.
- Take the thyroid preparation each morning to decrease the possibility of insomnia.
- Closely monitor blood pressure and pulse (older clients).
- Avoid excessive intake of foods that are known to inhibit TH utilization such as turnips, cabbage, carrots, spinach, and peaches.

Nursing Diagnoses and Interventions

In planning and implementing care for clients with hypothyroidism, the nurse takes into account that the disorder affects all organ systems. Although many nursing diagnoses might be valid, this section focuses on client problems with cardiovascular function, elimination, and skin integrity. See the accompanying Nursing Care Plan on page 546.

Decreased Cardiac Output

A TH deficit causes a reduction in heart rate and stroke volume, resulting in decreased cardiac output. There may also be an accumulation of fluid in the pericardial sac (from the edema characteristic of hypothyroidism), and coronary artery disease may be present, further compromising cardiac function.

- Monitor blood pressure, rate and rhythm of apical and peripheral pulses, respiratory rate, and breath sounds. *Hypotension indicates decreasing peripheral blood. Fluid in the pericardial sac restricts cardiac function. Monopolysaccharide deposits in the respiratory system decrease vital capacity and cause hypoventilation.*
- Suggest the client avoid chilling; increase room temperature, use additional bed covers, and avoid drafts. *Chilling increases metabolic rate and puts increased stress on the heart.*
- Explain the need to alternate activity with rest periods. Ask the client to report any breathing difficulties, chest pain, heart palpitations, or dizziness. *Activity increases demands on the heart and should be balanced with rest. Symptoms of cardiac stress include dyspnea, chest pain, palpitations, and dizziness.*

Constipation

The hypothyroid client is likely to have a reduced appetite and decreased food intake, a diminished activity level because of muscle aches and weakness, and reduced peristalsis to the point that fecal impactions may occur.

- Encourage a fluid intake of up to 2000 mL per day. Discuss preferred liquids and the best times of day to drink fluids. If kilocalorie intake is restricted, ensure that liquids have no kilocalorie or are low in kilocalorie. *Sufficient fluid intake is necessary to promote proper stool consistency.*
- Discuss ways to maintain a high-fiber diet. *Diets high in fiber and fluid produce soft stools. Fiber that is not digested absorbs water, which adds bulk to the stool and assists in the movement of fecal material through the intestines.*
- Encourage activity as tolerated. *Activity influences bowel elimination by improving muscle tone and stimulating peristalsis.*

PRACTICE ALERT
High-fiber foods include beans, potatoes, fruits, breads, cereal, crackers, popcorn, and rice.

Risk for Impaired Skin Integrity

The client with hypothyroidism is at risk for impaired skin integrity related to the accumulation of fluid in the interstitial spaces and to dry, rough skin. Decreased peripheral circulation, decreased activity levels, and slow wound healing further in-

NURSING RESEARCH Evidence-Based Practice: Self-Medicating with Thyroid Hormone

Michel, Neafsey, and Dzurec (2004) conducted a telephone survey of 38 women taking Neafsey levothyroxine for hypothyroidism. Clients taking levothyroxine have an increased risk of developing osteoporosis and are encouraged to take a calcium supplement to prevent bone loss. Prior research has shown that taking calcium carbonate within 4 hours of taking levothyroxine may decrease the absorption of the levothyroxine. The authors asked the subjects if they were taking calcium or multiple vitamin supplements and how they timed the ingestion of levothyroxine and the supplements. They also inquired about concurrent diseases and found that 13% had osteoporosis, 33% had hypertension, 27% had depression, 27% also had hyperlipidemia, and 24% had arthritis. Only 2 of the 38 subjects reported that calcium and levothyroxine should be administered separately. They learned this by patient information on pharmacy printouts. Those who were taking calcium (47%) reported taking it within 4 hours of taking levothyroxine; 17% took them together and 50% separated administration by 1 hour. Fifteen subjects taking calcium reported that a healthcare professional recommended it.

IMPLICATIONS FOR NURSING

Patient education about medication self-administration is within the scope of nursing practice. It is difficult to know the complex details about the vast number of medications and it is easy to overlook a supplement that is not prescribed by the healthcare provider. Nurses regularly review medication administration when clients are discharged from acute care facilities and levothyroxine is a common prescription. In fact, it is the second most commonly prescribed medication in the United States (Michel et al., 2004). Perhaps the best practice is to recommend clients read the pharmacy printout carefully for details about taking each medication and consult the pharmacist or healthcare provider if they have questions.

CRITICAL THINKING IN CLIENT CARE

1. How is hypothyroidism related to osteoporosis? What specific pathophysiologic processes increase the risk of osteoporosis? How does a calcium supplement prevent the development of osteoporosis?
2. What other factors contribute to the complexity of medication self-administration in the client with hypothyroidism?
3. What counsel about levothyroxine is important for clients with hypothyroidism? Should the client who has had the thyroid gland removed because of goiter or cancer receive the same counsel?

crease the risk. These interventions are outlined for the older client who is hospitalized for surgery or severe hypothyroidism.

- Monitor skin surfaces for redness or lesions, especially if the client's activity is greatly reduced. Use a pressure ulcer risk assessment scale to identify clients at risk. *Hypothyroidism causes dry, rough, edematous skin conditions that increase the risk of skin breakdown.*
- Provide or teach the immobile client measures to promote optimal circulation:
  - Use a turning schedule if the client is on bed rest, or teach the client to change position every 2 hours.
  - Limit the time for sitting in one position; shift weight or lift the body using arm rests every 20 to 30 minutes.
  - Use pillows, pads, or sheepskin or foam cushions for bed and/or chair.
  - Teach and implement a schedule of range-of-motion exercises.

  Prolonged pressure, especially in clients with edema and circulatory impairment, can occlude capillaries and cause hypoxic tissue damage.
- Provide or teach the client measures to maintain skin integrity:
  - Take baths only as necessary; use warm (not hot) water.
  - Use gentle motions when washing and drying skin.
  - Use alcohol-free skin oils and lotions.

  Dry skin and edema increase the risk of skin breakdown. Hot water, rough massage, and alcohol-based preparations may increase skin dryness, further impairing the body's ability to maintain skin integrity.

PRACTICE ALERT

Lift the client up in bed to prevent tissue damage from shearing forces.

NURSING CARE OF THE OLDER ADULT Variations in Assessment Findings—Hypothyroidism

Normal Changes with Aging

- The thyroid gland undergoes some degree of atrophy, fibrosis, and nodularity.
- Hair growth decreases.
- Nails are often thick, brittle, and yellow.
- Facial skin sags, and bones become more prominent.
- Deep tendon reflexes decrease.
- Response to questions may be slower.

Community-Based Care

Clients with hypothyroidism require lifelong care, primarily at home. Address the following topics:

- The need to take medications for the rest of one's life
- The need for periodic dosage reassessments
- If the client is older or does not have a support system, helpful community resources
- Additional resources:
  - American Thyroid Association
  - Thyroid Foundation of Canada
  - Endocrine Society.

MEDIALINK Hypothyroidism Resources

NURSING CARE PLAN A Client with Hypothyroidism

Jane Lee is a 60-year-old retired nurse living with her husband and daughter on a farm that has been in the family for four generations. Mrs. Lee has gained 10 lb (4.5 kg) in the past few months, even though she is rarely hungry and eats much less than normal. She is always tired and weak—so tired that she has not even been able to help with the chores on the farm or do housework. She is concerned about her appearance and the way she sounds when she talks. Her face is puffy, and her tongue always feels thick. Mr. Lee convinces his wife to make an appointment at a health center in a nearby town.

ASSESSMENT

Brian Henning, RN, completes the health assessment for Mrs. Lee at the health center. He finds that she now weighs 150 lb (68 kg), an increase of 10 lb (4.5 kg) over her weight at her last visit 6 months earlier. Mrs. Lee states that she always feels cold, tired, and weak. She also states that she is constipated, has difficulty remembering things, and looks different. Physical assessment findings include a palpable and bilaterally enlarged thyroid; dry, yellowish skin; nonpitting edema of the face and lower legs; and slow, slurred speech. Diagnostic tests revealed the following abnormal findings: T_3, 56 ng/dL (normal range: 80 to 200 ng/dL); T_4, 3.1 (normal range: 5 to 12 mg/dL); TSH increased. The medical diagnosis of hypothyroidism is made, and Mrs. Lee is started on levothyroxine 0.05 mg daily.

DIAGNOSES

- *Constipation* related to decreased peristalsis, as evidenced by hard, formed stools every 4 days
- *Impaired Verbal Communication* related to changes in speech patterns and enlarged tongue
- *Low Self-Esteem* related to changes in physical appearance and activity intolerance

EXPECTED OUTCOMES

- Regain normal bowel elimination patterns, having a soft, formed stool at least every other day.
- Experience improvement in verbal communication.
- Regain positive self-esteem as medication reduces physical changes and fatigue.

PLANNING AND IMPLEMENTATION

- Teach to increase fluids, bulk, and fiber in the diet to help regain a normal bowel elimination pattern of a soft, formed stool every other day.
- Take medication as prescribed and do not expect immediate reversal of symptoms affecting speech.
- Plan activities around rest periods. Encourage husband and daughter to help with housecleaning and cooking.

EVALUATION

On return to the health center 2 months later, Mrs. Lee reports that she is no longer constipated but that she is continuing to drink six glasses of water and eating oatmeal every day. She no longer feels cold, is regaining her normal energy, and even feels well enough to plant her garden. Her speech is clear and easy to understand. As she leaves the examining room, Mrs. Lee says, "It's hard to believe that I have changed so much—now I look and feel like the 'old' me!"

CRITICAL THINKING IN THE NURSING PROCESS

1. What physical changes that normally occur with aging are similar to the manifestations of hypothyroidism?
2. Describe the factors that put Mrs. Lee's safety at risk. What alterations in her home environment would you suggest to promote safety until the prescribed medication takes effect?
3. The client taking oral thyroid medications may become hyperthyroid. List the manifestations you would include in a teaching plan to signal this condition.

See Evaluating Your Response in Appendix C.

THE CLIENT WITH CANCER OF THE THYROID

Thyroid cancer is relatively rare, with an estimated rate of 25,690 new cases annually. Thyroid cancer accounts for approximately 1490 cancer deaths a year (American Cancer Society, 2005). The most consistent risk factor is exposure to ionizing radiation to the head and neck during childhood. For example, many adults in their 50s and 60s received x-ray treatments for colds and sinus infections during childhood.

Of the several types of thyroid cancer, the most common types are listed here:

- Papillary thyroid carcinoma is the most common thyroid malignancy. It is usually detected as a single nodule, but may arise from a multinodular goiter. The average age of diagnosis is 42, with 70% of cases occurring in women. Risks for the development of this form are exposure to external x-ray treatments to the head or neck as a child, childhood exposure to radioactive isotopes of iodine in nuclear fallout, and a family history. Papillary thyroid carcinoma is the least aggressive type, but does metastasize to the local and regional lymph nodes and lungs.
- Follicular thyroid cancer is the second most common thyroid malignancy. The average age of diagnosis is 50, with 75% of cases occurring in women. This form is more aggressive, with potential for vascular invasion and spread to lung and bone.

Thyroid cancer is manifested by a palpable, firm nontender nodule in the thyroid. If undetected, the tumor may grow and impinge on the esophagus or trachea, causing difficulty in swallowing or breathing. Most people with thyroid cancer do not have elevated thyroid hormone levels. The diagnosis is made by measuring thyroid hormones, performing thyroid scans, and by fine-needle biopsy of the nodule. The usual treatment is subtotal or total thyroidectomy. TSH suppression therapy with levothyroxine may be conducted prior to surgery. Radioactive iodine therapy (^{131}I) and chemotherapy are additional therapeutic options. The 5-year survival rate, if the tumor has not metastasized, is 97% (American Cancer Society, 2005). Nursing care for the client with cancer is discussed in Chapter 14 ∞.

DISORDERS OF THE PARATHYROID GLANDS

Disorders of the parathyroid glands, hyperparathyroidism and hypoparathyroidism, are not as common as those of the thyroid gland. Hypercalcemia and hypocalcemia (the primary results of alterations in parathyroid function) are discussed in Chapter 10.

THE CLIENT WITH HYPERPARATHYROIDISM

Hyperparathyroidism results from an increase in the secretion of parathyroid hormone (PTH), which regulates normal serum levels of calcium. The increase in PTH affects the kidneys and bones, resulting in the following pathophysiologic changes:

- Increased resorption of calcium and excretion of phosphate by the kidneys, which increases the risk of hypercalcemia and hypophosphatemia
- Increased bicarbonate excretion and decreased acid excretion by the kidneys, which increases the risk of metabolic acidosis and hypokalemia
- Increased release of calcium and phosphorus by bones, with resultant bone decalcification
- Deposits of calcium in soft tissues and the formation of renal calculi.

Pathophysiology and Manifestations

Hyperparathyroidism occurs more often in older adults and is three times more common in women. The disorder itself is not common. The three types of hyperparathyroidism are as follows:

- Primary hyperparathyroidism occurs when there is hyperplasia or an adenoma in one of the parathyroid glands. These disorders interrupt the normal regulatory mechanism between serum calcium levels and PTH secretion and increase the absorption of calcium through the gastrointestinal tract.
- Secondary hyperparathyroidism is a compensatory response by the parathyroid glands to chronic hypocalcemia. It is characterized by an increased secretion of PTH.
- Tertiary hyperparathyroidism results from hyperplasia of the parathyroid glands and a loss of response to serum calcium levels. This disorder is most often seen in clients with chronic renal failure.

Many clients with hyperparathyroidism are asymptomatic. When symptoms occur, they are related to hypercalcemia and various musculoskeletal, renal, and gastrointestinal manifestations. Bone reabsorption results in pathologic fractures, while elevated calcium levels alter neural and muscular activity, leading to muscle weakness and atrophy. Proximal renal tubule function is altered, and metabolic acidosis, renal calculi formation, and polyuria occur.

Manifestations of the effect of hypercalcemia on the gastrointestinal tract include abdominal pain, constipation, anorexia, and peptic ulcer formation. Hypercalcemia also affects the cardiovascular system, causing arrhythmias, hypertension, and increased sensitivity to cardiotonic glycosides (e.g., digitalis preparations). The manifestations of hyperparathyroidism are summarized in the following box.

MANIFESTATIONS of Hyperparathyroidism

MUSCULOSKELETAL SYSTEM
- Bone pain (back, joints, shins)
- Pathologic fractures (women)
- Muscle weakness
- Muscle atrophy

RENAL EFFECTS
- Renal calculi
- Polyuria
- Polydipsia

GASTROINTESTINAL SYSTEM
- Abdominal pain
- Peptic ulcers
- Pancreatitis
- Nausea
- Constipation

CARDIOVASCULAR SYSTEM
- Arrhythmias
- Hypertension

CENTRAL NERVOUS SYSTEM
- Paresthesias
- Depression
- Psychosis

METABOLIC EFFECTS
- Acidosis
- Weight loss

INTERDISCIPLINARY CARE

Hyperparathyroidism is diagnosed by excluding all other possible causes of hypercalcemia; by at least a 6-month history of symptoms; and by laboratory analysis of levels of serum calcium and PTH levels (Tierney et al., 2005).

Treatment of hyperparathyroidism focuses on decreasing the elevated serum calcium levels. Clients with mild hypercalcemia are urged to drink fluids and keep active. They should avoid immobilization, thiazide diuretics, large doses of vitamins A and D, antacids containing calcium, and calcium supplements. Severe hypercalcemia requires hospitalization and intensive treatment with intravenous saline. Medications to inhibit bone resorption and reduce hypercalcemia, such as pamidronate (Aredia), alendronate (Fosamax), and zoledronate (Zometa), are used for short-term treatment and may relieve bone pain (Tierney et al., 2005). Calcitonin, a hormone produced by the thyroid gland, decreases plasma levels of calcium by inhibiting bone resorption and increasing calcium excretion by the kidney (Lehne, 2004). A form of calcitonin from salmon is available as a nasal spray or IM/SQ injection.

Surgical removal of the parathyroid glands affected by hyperplasia or adenoma treats primary hyperparathyroidism. The preoperative and postoperative nursing care of the client having surgery of the parathyroids is essentially the same as that for the client having a thyroidectomy (see page 539).

NURSING CARE

Nursing care of the client with hypercalcemia is discussed in Chapter 10 ∞.

THE CLIENT WITH HYPOPARATHYROIDISM

Hypoparathyroidism results from abnormally low PTH levels. The most common cause is damage to or removal of the parathyroid glands during thyroidectomy. The lack of circulating PTH causes hypocalcemia and an elevated blood phosphate level.

Pathophysiology and Manifestations

Reduced levels of PTH result in impaired renal tubular regulation of calcium and phosphate. In addition, decreased activation of vitamin D results in decreased absorption of calcium by the intestines. The low calcium levels cause changes in neuromuscular activity, affecting peripheral motor and sensory nerves. Hypocalcemia lowers the threshold for nerve and muscle excitability; a slight stimulus anywhere along a nerve or muscle fiber initiates an impulse.

The neuromuscular manifestations that result include numbness and tingling around the mouth and in the fingertips, muscle spasms of the hands and feet, convulsions, and laryngeal spasms. Tetany, a continuous spasm of muscles, is the primary symptom of hypocalcemia. In severe cases of tetany, death may occur. Assessments for **tetany** include Chvostek's sign and Trousseau's sign (see Chapter 18 ∞). The manifestations of hypoparathyroidism are summarized in the box on this page.

MANIFESTATIONS of Hypoparathyroidism

MUSCULOSKELETAL SYSTEM
- Muscle spasms
- Facial grimacing
- Carpopedal spasms
- Tetany or convulsions

INTEGUMENTARY SYSTEM
- Brittle nails
- Hair loss
- Dry, scaly skin

GASTROINTESTINAL SYSTEM
- Abdominal cramps
- Malabsorption

CARDIOVASCULAR SYSTEM
- Arrhythmias

CENTRAL NERVOUS SYSTEM
- Paresthesias (lips, hands, feet)
- Mood disorders (irritability, depression, anxiety)
- Hyperactive reflexes
- Psychosis
- Increased intracranial pressure

INTERDISCIPLINARY CARE

Hypoparathyroidism is diagnosed by low serum calcium levels and high phosphorous levels in the absence of renal failure, an absorption disorder, or a nutritional disorder.

Treatment of hypoparathyroidism focuses on increasing calcium levels. Intravenous calcium gluconate is given immediately to reduce tetany. Long-term therapy includes supplemental calcium, increased dietary calcium, and vitamin D therapy.

NURSING CARE

Nursing care for the client with hypocalcemia is discussed in Chapter 10 ∞.

DISORDERS OF THE ADRENAL GLANDS

Disorders of the adrenal cortex or adrenal medulla result in changes in the production of adrenocorticotropic hormone (ACTH). Hormones of the adrenal cortex are essential to life. They maintain homeostasis in response to stressors. Disorders of the adrenal cortex result in complex physical, psychologic, and metabolic alterations that are potentially life threatening. Hormones of the adrenal medulla are not essential to life, because the sympathetic nervous system produces similar body responses. The disorders that occur are hyperfunction and hypofunction of the adrenal cortex and hyperfunction of the adrenal medulla.

THE CLIENT WITH HYPERCORTISOLISM (CUSHING'S SYNDROME)

Cushing's syndrome is a chronic disorder in which hyperfunction of the adrenal cortex produces excessive amounts of circulating cortisol or ACTH. Cushing's syndrome is more common in women, with the average age of onset between 30 and 50 years (Figure 19–3 ■). However, the disorder may occur at any age, especially as the result of pharmacologic therapy. People who take steroids for long periods of time (e.g., for

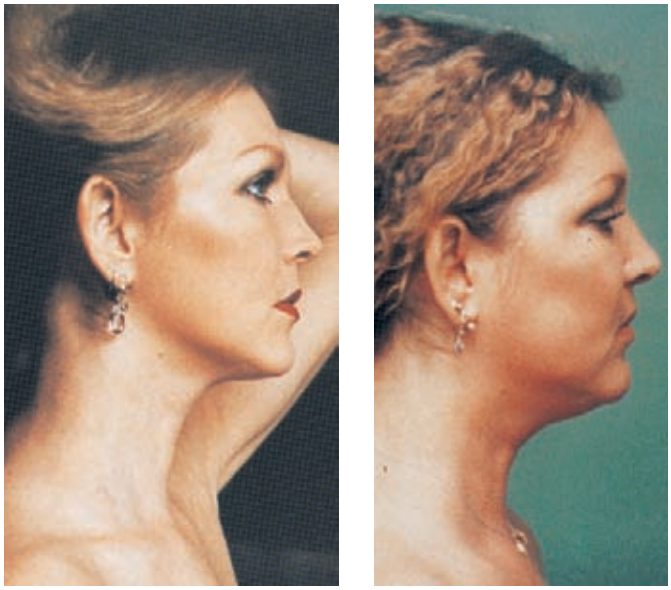


Figure 19–3 ■ A woman before and after developing Cushing's syndrome. In the photo at right, notice the swollen facial features.

Courtesy of Dr. Charles Wilson, University of California, San Francisco.

the treatment of arthritis, after an organ transplant, or as an adjunct to chemotherapy) are at increased risk for developing the disorder.

Pathophysiology

Cushing's syndrome may be the result of various causes. The most common etiologies of the disorder are as follows (Porth, 2005):

- The pituitary form, with ACTH hypersecretion by a tumor of the pituitary (called *Cushing's disease*). This is most commonly caused by a small pituitary adenoma, with persistent but disorderly and random overproduction of ACTH. Forty-three percent of Cushing's disease is due to hypersecretion of ACTH by the pituitary.
- The ectopic form, caused by ACTH-secreting tumors (such as small-cell lung cancer). In this form, the ACTH is also random and episodic, but greater than in Cushing's disease.
- The adrenal form, resulting from excessive cortisol secretion by a benign or malignant adrenal tumor. The excess secretion suppresses pituitary ACTH production, resulting in atrophy of the uninvolved adrenal cortex. Thirty-two percent of Cushing's disease is due to excessive autonomous secretion of cortisol by the adrenal glands, independent of ACTH (Tierney et al., 2005).
- Iatrogenic Cushing's syndrome, resulting from long-term therapy with potent pharmacologic glucocorticoid preparations.

Manifestations

The manifestations of Cushing's syndrome result from the ACTH or cortisol excess, and manifest as exaggerated cortisol actions. Obesity and a redistribution of body fat result in fat deposits in the abdominal region (central obesity), fat pads under the clavicle, a "buffalo hump" over the upper back, and a round "moon" face. Changes in protein metabolism cause muscle weakness and wasting, especially in the extremities. Glucocorticoid excess inhibits fibroblasts, resulting in loss of collagen and connective tissue. Thinning of skin, abdominal striae (reddish purple "stretch marks"), easy bruising, poor wound healing, and frequent skin infections result. Glucose metabolism is altered in the majority of clients, and diabetes mellitus may occur. Electrolyte imbalances also occur with the increased hormone levels. Changes in calcium absorption result in osteoporosis, compression fractures of the vertebrae, fractures of the ribs, and renal calculi. Hypokalemia and hypertension occur as potassium is lost and sodium is retained. Inhibited immune responses increase the risk of infection, and increased gastric acid secretion increases the risk of peptic ulcers. Emotional changes range from depression to psychosis. In women, increasing androgen levels cause hirsutism (excessive facial hair in particular), acne, and menstrual irregularities. The manifestations and effects of Cushing's syndrome are grouped by body system in the box on this page.

The complications of untreated Cushing's syndrome include electrolyte imbalances (hyperglycemia, hypernatremia, and hypokalemia), hypertension, and emotional disturbances. Increased susceptibility to infections is also a factor. Compression fractures from osteoporosis and aseptic necrosis of the femoral head may result in serious disability. If the client undergoes a bilateral adrenalectomy as a treatment for Cushing's syndrome, an acute deficit of cortisol (Addisonian crisis) may result.

MANIFESTATIONS of Cushing's Syndrome

MUSCULOSKELETAL SYSTEM
- Weakness
- Muscle wasting
- Osteoporosis

INTEGUMENTARY SYSTEM
- Thin, easily bruised skin
- Skin infections
- Poor wound healing
- Eccyhmosis
- Purple striae (around thighs, breasts, abdomen)
- Hirsutism

CENTRAL NERVOUS SYSTEM
- Emotional lability
- Psychoses

GASTROINTESTINAL SYSTEM
- Peptic ulcers

CARDIOVASCULAR SYSTEM
- Hypertension

RENAL EFFECTS
- Renal calculi
- Polyuria
- Polydipsia
- Glycosuria

METABOLIC EFFECTS
- Hypokalemia
- Hypernatremia
- Truncal obesity

REPRODUCTIVE SYSTEM
- Oligomenorrhea or amenorrhea
- Impotence
- Decreased libido

INTERDISCIPLINARY CARE

The treatment of Cushing's syndrome includes medications, radiation therapy, or surgery, depending on the etiologic origin of the disorder.

Diagnostic Tests

Cushing's syndrome is diagnosed through a variety of diagnostic tests. Findings are shown in Table 19–3 and in the following bulleted summary:

- *Plasma cortisol levels* are measured. If Cushing's disease is present, test results show a loss of the normal diurnal variations of higher levels in the morning and lower levels in the afternoon.
- *Plasma ACTH levels* are measured to determine the etiology of the syndrome. Normally, plasma ACTH levels are highest from 7 A.M. to 10 A.M. and lowest from 7 P.M. to 10 P.M. In secondary Cushing's syndrome, ACTH is elevated; in primary Cushing's syndrome, ACTH is decreased.
- *24-hour urine tests* (17-ketosteroids [17-KS] and *17-hydroxycorticosteroids*) are conducted to measure free cortisol and androgens; these hormones are increased in Cushing's syndrome. Because synthesis and circulation of adrenal hormones are diurnal and episodic, 24-hour urine collections

TABLE 19–3 Laboratory Findings in Cushing's Syndrome

	TEST	NORMAL VALUES	FINDINGS
Serum	Cortisol	8 A.M. to 10 A.M.: 5 to 23 mcg/dL 4 P.M. to 6 P.M.: 3 to 13 mcg/dL	Increased
	Blood urea nitrogen (BUN)	5 to 25 mg/dL	Normal
	Sodium	135 to 145 mEq/L	Increased
	Potassium	3.5 to 5.0 mEq/L	Increased
	Glucose (serum)	70 to 100 mg/dL	Increased
Urine	17-KS	Male: 5 to 25 mg/24h Female: 5 to 15 mg/24h >Age 65: 4 to 8 mg/24h	Increased

more correctly reflect total hormone than serum levels drawn intermittently.

- *Serum potassium, calcium, and glucose levels* are measured to identify electrolyte imbalances.
- *ACTH suppression test* may be conducted to identify the cause of the disorder. A synthetic cortisol (dexamethasone) is given to suppress the production of ACTH, and plasma cortisol levels are measured. If an extremely high dose of cortisol is necessary to suppress ACTH, the primary disorder is adrenal cortex hyperplasia. If ACTH is not suppressed with the synthetic cortisol, an adrenal tumor is suspected (Kirk et al., 2002).

Medications

Cushing's syndrome that results from a pituitary tumor is treated by medications as an adjunct to surgery or radiation. Medications are also used for clients with inoperable pituitary or adrenal malignancies. Although the drugs control symptoms, they do not effect a cure. Examples of some commonly prescribed drugs follow:

- Mitotane directly suppresses activity of the adrenal cortex and decreases peripheral metabolism of corticosteroids. It is used to treat metastatic adrenal cancer.
- Aminogluthemide or ketoconazole (or both) inhibit cortisol synthesis by the adrenal cortex and may be administered to clients with ectopic ACTH-secreting tumors that cannot be surgically removed.
- Somatostatin analog (octreotide) suppresses ACTH secretion in some clients.

Surgery

When Cushing's syndrome is caused by an adrenal cortex tumor, an adrenalectomy may be performed to remove the tumor. Only one adrenal gland is usually involved; however, if an ACTH-producing ectopic tumor is involved, a bilateral adrenalectomy is performed. Lifelong hormone replacement is necessary if both adrenal glands are removed. Nursing care of the client having an adrenalectomy is discussed below.

Surgical removal of the pituitary gland (hypophysectomy) is indicated when Cushing's syndrome is the result of a pituitary disorder. The gland is removed either by a transsphenoidal route or by a craniotomy. Nursing care for the client having cranial surgery is discussed in Chapter 44 ∞.

NURSING CARE

Health Promotion

Stress the risk of developing Cushing's syndrome for clients taking long-term steroids. The risk of abruptly discontinuing the medications is an essential component of teaching. For a review of glucocorticoid administration see Chapter 12 ∞.

NURSING CARE OF THE CLIENT HAVING AN Adrenalectomy

PREOPERATIVE CARE

- Request a dietary consultation to discuss with the client about a diet high in vitamins and proteins. If hypokalemia exists, include foods high in potassium. *Glucocorticoid excess increases catabolism. Vitamins and proteins are necessary for tissue repair and wound healing following surgery.*
- Use careful medical and surgical asepsis when providing care and treatments. *Cortisol excess increases the risk of infection.*
- Monitor the results of laboratory tests of electrolytes and glucose levels. *Electrolyte and glucose imbalances are corrected before the client has surgery.*
- Teach the client to turn, cough, and perform deep-breathing exercises. *Although they are important for all surgical clients, these activities are even more important for the client who is at risk for infection. Having the client practice and demonstrate the activities increases postoperative compliance.*

POSTOPERATIVE CARE

- Take and record vital signs, measure intake and output, and monitor electrolytes on a frequent schedule, especially during the first 48 hours after surgery. *Removal of an adrenal gland, especially a bilateral adrenalectomy, results in adrenal insufficiency. Addisonian crisis and hypovolemic shock may occur. Cortisol is often given on the day of surgery and in the postoperative period to replace inadequate hormone levels. Intravenous fluids are also administered.*
- Assess body temperature, WBC levels, and wound drainage. Change dressings using sterile technique. *Impaired wound healing increases the risk of infection in clients with adrenal disorders. Use aseptic technique to decrease this risk.*

Assessment

Collect the following data through the health history and physical examination (see Chapter 18 ∞). Further focused assessments are described with nursing interventions below.

- *Health history:* History of pituitary, adrenal, pancreatic, or pulmonary tumor; frequent infections; gastrointestinal bleeding; stress fractures; pain, changes in weight distribution; change in height; fatigue; weakness; change in appearance; bruising; skin infections; menstrual history; sexual function.
- *Physical assessment:* Vital signs, behavior, appearance, fat distribution, face, skin, hair quantity and distribution, muscle size and strength, gait.

Nursing Diagnoses and Interventions

The nurse caring for the client with Cushing's syndrome must take a holistic approach to plan and implement interventions for a wide variety of responses, including problems related to fluid and electrolyte balance, injury, infection, and body image. For additional information about clients with alterations in fluid and electrolyte balance, see Chapter 10. See accompanying Nursing Care Plan below.

Fluid Volume Excess

The excess cortisol, not mineralocorticoid, secretion associated with Cushing's syndrome results in sodium and water

MEDIALINK Care Plan Activity: Cushing's Syndrome

NURSING CARE PLAN A Client with Cushing's Syndrome

Sara Domico is a 30-year-old lawyer living in a major metropolitan area. She has never been married, and she shares her life with her cat, Beau, and her parents, who live nearby. Her physician recently diagnosed Ms. Domico as having Cushing's syndrome and admits her to the hospital for surgery for an adrenal cortex tumor (adrenalectomy). She has been having increased muscle weakness, so much so that she has difficulty climbing the one flight of stairs to her apartment. She has also had difficulty sleeping, irregular menstrual periods, and hypertension. Ms. Domico is especially concerned about her protruding abdomen, round face, development of facial hair, and the numerous bruises that have appeared on her skin.

ASSESSMENT

When Ms. Domico arrives at the hospital the morning of surgery, she is admitted by her case manager, Ann Sprengel, RN, CNS. Ann completes a physical assessment that includes abnormal findings of thin lower extremities, an enlarged abdomen, purple striae over the abdomen and buttocks, a round face, and obvious facial hair. Her blood pressure is 160/96. Ms. Domico tells Ann that she is always tired and that sometimes it "just wears me out to walk from the bedroom to the kitchen." Diagnostic tests conducted prior to admission reveal the following abnormal findings (all except cortisol levels are corrected before surgery).

Glucose: 186 mg/dL (normal range: 70 to 110 mg/dL)
Sodium: 152 mEq/L (normal range: 135 to 145 mEq/L)
Potassium: 3.2 mEq/L (normal range: 3.5 to 5.0 mEq/L)
Calcium: 4.3 mEq/L (normal range: 4.5 to 5.5 mEq/L)
Cortisol: 35 mg/dL (normal for A.M.: 5 to 23 mg/dL)

DIAGNOSES

- *Fluid Volume Excess* related to sodium retention causing edema and hypertension
- *Risk for Injury* related to generalized fatigue and weakness
- *Risk for Infection* related to impaired immune response and edema
- *Disturbed Body Image* related to physical changes secondary to Cushing's syndrome

EXPECTED OUTCOMES

- Regain a normal body fluid balance.
- Remain free of injury.
- Remain free of infection.
- Verbalize understanding of the physical effects of the disease process and realistic expectations of desired changes in appearance.

PLANNING AND IMPLEMENTATION

- Weigh each morning, using the same scale.
- Maintain an accurate record of intake and output.
- Ensure adequate lighting in the room, and wear glasses and shoes when getting out of bed.
- Develop a written schedule of rest and activity periods.
- If agreeable, provide a private room, and restrict visitors to parents at this time.
- Use strict medical and surgical asepsis when providing care.
- Provide time for discussion of the disease and treatment; encourage verbalization of feelings and identify successful coping mechanisms used in the past.
- Encourage turning, coughing, and deep breathing and/or incentive spirometry every 2–4 hours.

EVALUATION

Ms. Domico states that she is "ready to have surgery and start feeling better." She has not fallen or injured herself, and she has remained free of infection. Although edema is still present, she has lost 8 lb (3.6 kg), and her blood pressure is decreased. Ms. Domico has openly discussed her concerns about the way she looks and feels; she understands that symptoms will improve following surgery. She has strong religious beliefs and family support, both of which provide strength and help her cope with the effects of the disorder and the need for any further treatment.

CRITICAL THINKING IN THE NURSING PROCESS

1. When Ms. Domico was admitted to the hospital, several of her test results were abnormal. Describe the pathophysiologic reason for those results.
2. List the assessments that nurses can make to determine body fluid balance.
3. Develop a plan of care for this client for the nursing diagnosis *Fatigue.*

See Evaluating Your Response in Appendix C.

reabsorption, causing fluid volume excess (Porth, 2005). The client will have weight gain, edema, and hypertension.

- Ask the client to weigh at the same time each day, and maintain a record of results. *Body weight is an accurate indicator of fluid status. One liter of fluid retention corresponds to about 2 lb (0.9 kg) of body weight.*
- Monitor blood pressure, rate and rhythm of pulse, respiratory rate, and breath sounds. Assess for peripheral edema and jugular vein distention. *Extracellular fluid volume excess resulting from sodium and water retention is manifested by hypertension and a bounding, rapid pulse. There may also be crackles and wheezes, dependent edema, and venous distention.*
- Teach the client and family the reasons for restricting fluid and the importance of limiting fluids if ordered. *Restricting fluid can help decrease the risk of fluid volume excess. Involving the client and family in the plan of care and teaching the rationale for interventions helps achieve goals.*

Risk for Injury

The client with Cushing's syndrome is at risk for injury from several causes. Excess cortisol causes increased absorption of calcium and demineralization of bones, resulting in risk of pathologic fractures. Muscle weakness and fatigue are common, increasing the potential for accidental falls.

- Teach the client and family to maintain a safe environment:
  - Keep unnecessary clutter and equipment out of the way and off the floor.
  - Ensure adequate lighting, especially at night.
  - Encourage the use of assistive devices for ambulation or to ask for help if needed.
  - If the client wears corrective lenses, be sure they are available and clean.
  - Encourage the use of nonskid slippers or shoes.
  - Monitor for signs of fatigue (increased pulse and respirations); plan rest periods.

  A well-lighted environment free of clutter decreases the risk of falls and injury. Sensory and motor deficits increase the risk of falls; corrective lenses, assistive devices, and nonslip footwear can decrease this risk. Rest relieves fatigue. To reduce energy expenditure, include alternating periods of rest and activity in daily schedules.

Risk for Infection

Elevated cortisol levels impair the immune response and put the client with Cushing's syndrome at increased risk for infection. Increased cortisol also affects protein synthesis, causing delayed wound healing, and inhibits collagen formation, which results in epidermal atrophy, further inhibiting resistance to infection. In addition, impaired blood flow to edematous tissue results in altered cellular nutrition, which increases the potential for infection. The following interventions are outlined for the client with Cushing's syndrome who is hospitalized:

- Place in a private room, and limit visitors. *The client must avoid exposure to environmental infection.*
- Monitor vital signs and verbalizations of subjective manifestations (e.g., the client's response to "How do you feel?") every 4 hours. *Increased body temperature and pulse are systemic indicators of infection; however, because Cushing's syndrome impairs the normal inflammatory response, the usual indicators of inflammation may not be present.*
- Use principles of medical and sterile asepsis when caring for the client, conducting procedures, or providing wound care. *Impaired skin and tissues make aseptic techniques even more necessary to decrease the risk of infection. Intact skin is the first line of defense against infection; if invasive procedures are performed or a wound is present, this defense is lost.*
- If wounds are present, assess the color, odor, and consistency of wound drainage, and look for increased pain in and around the wound. *Cortisol excess delays wound healing and closure.*
- Teach the importance of increasing intake of protein and vitamins C and A. *Protein, vitamin C, and vitamin A are necessary to collagen formation; collagen helps support and repair body tissues.*

PRACTICE ALERT

A generalized feeling of malaise may be the primary manifestation of infection.

Disturbed Body Image

The client with Cushing's syndrome has obvious physical changes in appearance. The abnormal fat distribution, moon face, buffalo hump, striae, acne, and facial hair (in women) all contribute to disruptions in the way clients with this disorder perceive themselves.

- Encourage clients to express feelings and to ask questions about the disorder and its treatment. *The loss of one's normal body image may prompt feelings of hopelessness, powerlessness, anger, and depression. Understanding the disease and adapting to changes from that disease are the first steps in regaining control of one's own body.*
- Discuss strengths and previous coping strategies. Enlist the support of family or significant others in reaffirming the client's worth. *Disturbances in body image are often accompanied by low self-esteem. Self-esteem derives from one's perception of competence and from appraisals of others.*
- Discuss signs of progress in controlling symptoms; for example, decreased facial edema or increased activity tolerance. *Many physical changes from cortisol excess disappear with treatment. Clearly communicate this fact, because the client may believe changes are permanent.*

Using NANDA, NIC, and NOC

Chart 19–1 shows links between NANDA, NIC, and NOC when caring for the client with Cushing's syndrome.

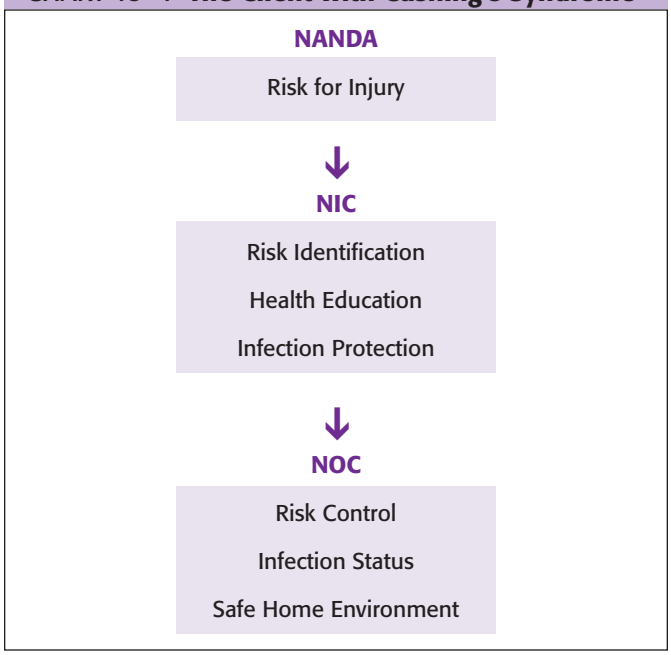
Community-Based Care

The client with Cushing's syndrome requires education about self-care at home specific to the type of treatment given. Address the following topics:

- Safety measures to prevent falls if fatigue, weakness, and osteoporosis are present
- Taking medications as prescribed, with information about side effects. Clients often require medications for the rest of their lives, and dosage changes are highly likely

NANDA, NIC, AND NOC LINKAGES

CHART 19–1 **The Client with Cushing's Syndrome**



Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

- Having regular health assessments
- Wearing a medical ID indicating the client has Cushing's syndrome
- Helping the older client with referrals to social services or community health services because of the complexity of the treatment and care required
- Providing helpful resources:
  - American Association of Clinical Endocrinologists
  - Endocrine Society.

THE CLIENT WITH CHRONIC ADRENOCORTICAL INSUFFICIENCY (ADDISON'S DISEASE)

Addison's disease is a disorder resulting from destruction or dysfunction of the adrenal cortex. The result is chronic deficiency of cortisol, aldosterone, and adrenal androgens, accompanied by skin pigmentation. It can occur at any age, although it is more common in adults under the age of 60. Like many endocrine disorders, Addison's disease is more common in women.

Pathophysiology

There are many possible causes of Addison's disease. The etiologies include:

- Autoimmune destruction of the adrenals. This is the most common cause, accounting for about 80% of spontaneous cases (Holcomb, 2006; Tierney et al., 2005). It may occur alone, or as part of a polyglandular autoimmune syndrome (PGA). Type 2 PGA is seen in adults, often associated with autoimmune thyroid disease (usually hypothyroidism), type 1 diabetes, primary ovarian or testicular failure, and pernicious anemia.
- Clients who are taking anticoagulants, have major trauma, sepsis, or are having open heart surgery. Such clients may have bilateral adrenal hemorrhage.
- Adrenoleukodystrophy, an X-linked disorder characterized by an accumulation of very long chain fatty acids in the adrenal cortex, testes, brain, and spinal cord.
- ACTH deficit, resulting from pituitary tumors, pituitary surgery or irradiation, and the use of exogenous steroids.
- Clients who are abruptly withdrawn from long-term, high-dose steroid therapy. Other clients at risk are those with tuberculosis or acquired immune deficiency syndrome (AIDS); the pathogens responsible for either disease can infiltrate and destroy adrenal tissue.

Adrenocortical destruction initially causes a decrease in adrenal glucocorticoid reserve. Basal glucocorticoid secretion is normal, but does not increase in response to stress and surgery. Trauma or infection can precipitate an adrenal crisis. As the destruction of the adrenal cortex continues, even basal secretion of glucocorticoids and mineralocorticoids is deficient. Decreasing plasma cortisol reduces the feedback inhibition of pituitary ACTH and plasma ACTH rises.

Secondary adrenocortical insufficiency occurs when either large doses or prolonged therapy with glucocorticoids are given for their anti-inflammatory and immunosuppressive effects to treat diseases such as arthritis and asthma. If the steroid medications are suddenly discontinued, the hypothalamus and pituitary cannot respond normally to the reduced level of circulating glucocorticoids. The client may develop manifestations of chronic adrenocortical insufficiency or, if subjected to stress, adrenal crisis (Tierney et al., 2005).

Manifestations

The onset of Addison's disease is slow in most cases; the client experiences symptoms after about 90% of the function of the gland is lost. The primary manifestations are the result of elevated ACTH levels and decreased aldosterone and cortisol (see the box on page 554). Aldosterone deficiency affects the ability of the distal tubules of the nephron to conserve sodium. Sodium is lost, potassium is retained, extracellular fluid is depleted, and the blood volume is decreased. Postural hypotension and syncope are common, and hypovolemic shock may occur. Hyponatremia causes dizziness, confusion, and neuromuscular irritability. Hyperkalemia causes cardiac arrhythmias.

Cortisol insufficiency also causes decreased hepatic gluconeogenesis with hypoglycemia. The client tolerates stress poorly and experiences lethargy, weakness, anorexia, nausea, vomiting, and diarrhea. The increased ACTH levels stimulate hyperpigmentation in more than 90% of clients with Addison's disease (Porth, 2005). In Caucasian clients, the skin looks deeply suntanned or bronzed in both exposed and unexposed areas.

Addisonian Crisis

Addisonian crisis is a life-threatening response to acute adrenal insufficiency and occurs in about 25% of patients (Holcomb, 2006). Triggers include surgery, acute systemic illness,

MANIFESTATIONS of Addison's Disease

INTEGUMENTARY SYSTEM
- Delayed wound healing
- Hyperpigmentation

CARDIOVASCULAR SYSTEM
- Postural hypotension
- Tachycardia
- Arrhythmias

CENTRAL NERVOUS SYSTEM
- Lethargy
- Emotional lability
- Tremors
- Confusion

MUSCULOSKELETAL SYSTEM
- Weakness
- Joint pain
- Muscle wasting
- Muscle pain

GASTROINTESTINAL SYSTEM
- Anorexia
- Diarrhea
- Nausea and vomiting

REPRODUCTIVE SYSTEM
- Menstrual changes

METABOLIC EFFECTS
- Hyperkalemia
- Hypoglycemia
- Hyponatremia

trauma, or abrupt withdrawal of long-term corticosteroid therapy. The disorder is chronic after the acute episode resolves.

This response can occur in any person with Addison's disease; however, it is most commonly precipitated by major stressors, especially if the disease is poorly controlled. Addisonian crisis may also occur in clients who are abruptly withdrawn from glucocorticoid medications or who have hemorrhage into the adrenal glands from either septicemia or anticoagulant therapy.

The client with Addisonian crisis may have any of the manifestations of Addison's disease, but the primary symptoms develop rapidly and are a high fever; weakness; severe, penetrating pain in the abdomen, lower back, and legs; severe vomiting; diarrhea; and hypotension; and circulatory collapse, shock, and coma.

Treatment of the crisis is rapid intravenous replacement of fluids and glucocorticoids. Fluid balance is usually restored in 4 to 6 hours.

INTERDISCIPLINARY CARE

The client with Addison's disease requires early diagnosis and treatment. Medical treatment includes cortisol replacement therapy.

Diagnostic Tests

Addison's disease is diagnosed through findings of decreased levels of cortisol, aldosterone, and urinary 17-KS. Dehydration may result in increased hematocrit and blood urea nitrogen (BUN). Blood glucose levels are decreased, and potassium is increased. A list of laboratory findings in Addison's disease is shown in Table 19–4. The following diagnostic tests are used:

- *Serum cortisol levels,* which are decreased in adrenal insufficiency

TABLE 19–4 Laboratory Findings in Addison's Disease

	TEST	NORMAL VALUES	FINDINGS
Serum	Cortisol	8 A.M. to 10 A.M.: 5 to 23 mcg/dL 4 P.M. to 6 P.M.: 3 to 13 mcg/dL	Decreased
	Blood urea nitrogen (BUN)	5 to 25 mg/dL	Increased
	Sodium	135 to 145 mEq/L	Decreased
	Potassium	3.5 to 5.0 mEq/L	Increased
	Glucose (serum)	70 to 100 mg/dL	Decreased
Urine	17-KS	Male: 5 to 25 mg/24h Female: 5 to 15 mg/24h >Age 65: 4 to 8 mg/24h	Low/absent

- *Blood glucose levels,* which are decreased in adrenal insufficiency
- *Serum sodium levels,* which are decreased in adrenal insufficiency
- *Serum potassium levels,* which are increased in adrenal insufficiency
- *BUN levels,* which are increased in adrenal insufficiency
- *Urinary 17-hydroxycorticoids* and *17-KS levels,* which are decreased in adrenal insufficiency
- *Plasma ACTH levels,* which are increased in primary adrenal insufficiency but decreased in secondary adrenal insufficiency
- Possibly *ACTH stimulation test* (Cortisol levels rise with pituitary deficiency but do not rise in primary adrenal insufficiency.)
- *CT scans* of the head, which identify any intracranial lesion impinging on the pituitary gland.

Medications

The primary medical treatment of Addison's disease is replacement of corticosteroids and mineralocorticoids, accompanied by increased sodium in the diet. Hydrocortisone (Cortef) is given orally to replace cortisol; fludrocortisone (Florinef) is given orally to replace mineralocorticoids. Nursing implications in cortisol replacement are given in the box on the following page.

NURSING CARE

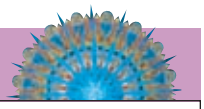
Health Promotion

Health promotion interventions for the client with or at risk for Addison's disease focus on careful assessments during anticoagulant therapy, open heart surgery, and trauma treatment. If the disease is present, teaching to prevent or treat an Addisonian crisis is essential.

Assessment

Collect the following data through the health history and physical examination (see Chapter 18 ∞). Further focused assessments are described with nursing interventions in the following text.

MEDICATION ADMINISTRATION ADDISON'S DISEASE



CORTISOL REPLACEMENTS
Cortisone (Cortone, Cortogen)
Hydrocortisone (Cortisol, Hydrocortone, Cortef)
Fludrocortisone acetate (Florinef, F-Cortef)
Dexamethasone (Decadron, Hexadrol, Dexasone)
Prednisone (Meticorten, Deltasone, Orasone)
Prednisolone (Meticortelone)

NURSING ALERT
Prednisolone and predinsone are equivalent in potency; dexamethasone (Decadron) is four to five times more potent.

Methylprednisolone (Medrol, Solu-Medrol)
Adrenocorticosteroids are used for replacement therapy in acute and chronic adrenal insufficiency. These drugs have anti-inflammatory and immunosuppressant effects. They also facilitate coping with stress.

Because corticosteroids are immunosuppressants, their use is contraindicated when an infection is suspected because they mask the signs of infection. Immunizations with live vaccines should not be attempted. Corticosteroids are also contraindicated in many other disorders, including peptic ulcer, Cushing's syndrome, cardiac disease, hyperthyroidism, hypothyroidism, and tuberculosis. Concurrent use with NSAIDs is contraindicated because of the effect on the gastrointestinal tract.

When these drugs are administered in small doses for replacement therapy, side effects are uncommon. Large doses or prolonged therapy may cause a Cushing-like syndrome, with atrophy of the adrenal cortex. Older clients, especially postmenopausal women, are more prone to develop hypertension and osteoporosis when undergoing glucocorticoid therapy. These drugs are used with caution in children and the older adult and are not usually administered to pregnant women.

Nursing Responsibilities
- Establish baseline data, including mental status, neurologic function, vital signs, and weight.
- Identify medications that might interact with corticosteroids: antidiabetic agents, cardiac glycosides, oral contraceptives, anticoagulants, NSAIDs.
- Document and report increased blood pressure, edema or weight gain, bleeding or bruising, weakness, or manifestations of Cushing's syndrome.
- Administer oral forms of the drug with food to minimize its ulcerogenic effect.
- Monitor electrolyte levels for increased sodium and decreased potassium.
- Monitor capillary blood glucose for hyperglycemia in the diabetic client.

Health Education for the Client and Family
- Take medications with food or milk, and report any gastric distress or dark stools.
- Most people need to take the medications for the rest of their lives.
- Consume a diet that is low in potassium, and higher in sodium and protein.
- Weigh yourself each day at the same time, and report any consistent weight gain, which indicates fluid retention.
- Use safety measures in the home to prevent falls and injuries.
- Corticosteroids may impair the effectiveness of oral contraceptives.
- Take the medication regularly and continuously. *Abruptly discontinuing the medication is dangerous.*
- Obtain a Medic-Alert bracelet.
- Monitor for increased stressors (infection, dental work, personal crisis) and increase the dose as indicated by the physician.
- Anticoagulant drugs or insulin may decrease the effectiveness of corticosteroids.
- Report the following to the physician: dizziness on sitting or standing, nausea and vomiting, pain, thirst, feelings of anxiety, malaise, infections.

- *Health history:* Weight loss, changes in skin color, nausea and vomiting, anorexia, diarrhea, abdominal pain, weakness, amenorrhea, changes in sexual desire, confusion, intolerance of stress.
- *Physical assessment:* Height and weight, vital signs, skin, hair quality and distribution, muscle size and strength.

Nursing Diagnoses and Interventions

The client with Addison's disease requires nursing care for a wide variety of responses to the decrease in cortisol levels. Nursing diagnoses discussed in this section are directed toward problems with fluid and electrolyte balance and compliance with lifelong self-care. See the accompanying Nursing Care Plan on page 556.

Deficient Fluid Volume

Fluid volume deficit in the client with Addison's disease results from loss of water and sodium, as well as from vomiting and diarrhea. Extracellular fluid volume deficit, decreased cardiac output, hypotension, and hypovolemic shock may occur, especially in crisis situations. Interventions for this diagnosis are outlined for the client who is hospitalized.

- Monitor intake and output, and assess for signs of dehydration: dry mucous membranes; thirst; poor skin turgor; sunken eyeballs; scanty, dark urine; increased urine specific gravity; weight loss; and increased hemoconcentration (increased hematocrit and BUN). *Glucocorticoid and mineralocorticoid depletion causes fluid volume deficit. Fluid volume deficit may reach crisis levels if undetected, causing altered tissue perfusion and hypovolemic shock.*
- Monitor cardiovascular status: Take and record vital signs, assess character of pulses, monitor potassium levels and ECGs. *Fluid volume deficit may lead to hypotension and a rapid, weak, or thready pulse. As aldosterone levels fall, renal excretion of potassium decreases, increasing blood levels of potassium.*

NURSING CARE PLAN A Client with Addison's Disease

A 51-year-old unemployed salesman, Mr. Don Sardoff, is brought to the emergency room by his wife, Ellen, at 8 A.M. Mrs. Sardoff tells the emergency room nurse that her husband has not been feeling well for the last week, but that when he got up this morning, he was so weak he couldn't dress himself and didn't know where he was. Mrs. Sardoff also tells the nurse that her husband has been taking a cortisone drug for treatment of his rheumatoid arthritis for the past 2 years, but notes, "We didn't have the money to buy it this month."

ASSESSMENT

On admission to the emergency room, Mr. Sardoff is dehydrated, with dry oral mucous membranes and tongue, poor skin turgor, and sunken eyeballs. His blood pressure is 94/44, and his pulse is rapid and thready. He is weak, dizzy, and disoriented about time and place. Diagnostic tests reveal the following abnormal findings at 8:30 A.M:

- EKG: widening QRS complex and increased PR interval
- Sodium: 129 mEq/L (normal range: 135 to 145 mEq/L)
- Glucose: 54 mg/dL (normal range: 70 to 110 mg/dL)
- Potassium: 5.3 mEq/L (normal range: 3.5 to 5 mEq/L)
- Cortisol: 2 mg/dL (normal for A.M.: 5 to 23 mg/dL)

The medical orders for Mr. Sardoff include intravenous administration of 5% dextrose in normal saline (D_5NS) at 250 mL/h and hydrocortisone (Solu-Cortef) 200 mg. After the fluids and medication are initiated, Mr. Sardoff is moved to an in-hospital medical bed.

DIAGNOSES

- *Deficient Fluid Volume* related to hypovolemia secondary to adrenal insufficiency
- *Ineffective Tissue Perfusion: Peripheral* related to fluid volume deficit
- *Anxiety* related to lack of knowledge about the effects and treatment of adrenal insufficiency

EXPECTED OUTCOMES

- Regain normal fluid balance.
- Regain normal peripheral perfusion with blood pressure within normal range.
- Verbalize knowledge of the causes and effects of adrenal insufficiency.

PLANNING AND IMPLEMENTATION

- Monitor intake and output closely.
- Take and record weight at the same time daily.
- Monitor blood pressure, pulses, and skin turgor every 2 hours until stable, then four times a day.
- Monitor electrolytes, and report abnormal results.
- Discuss a diet that is high in sodium, low in potassium, and has an increased fluid intake (3000 mL per day). Discuss the types of fluids desired and the best times for intake of increased fluids.
- Assist during activity to prevent falls.
- Provide verbal and written instructions, and encourage verbal feedback about the causes and effects of the disease, the effects of medications, the effects of not taking long-term cortisone drugs, the diet, and self-care at home.

EVALUATION

Following treatment for acute adrenal insufficiency, Mr. Sardoff is no longer dehydrated, and his blood pressure has returned to his normal reading of 132/88. He is alert and oriented, and anxious to learn to care for himself at home. After dietary instructions and teaching for self-care that included his wife, Mr. Sardoff verbalizes an understanding of his illness and the need to take his medication carefully and accurately. A referral is made to a social worker for assistance with costs of medications.

CRITICAL THINKING IN THE NURSING PROCESS

1. Adrenal insufficiency is often diagnosed only when the client becomes seriously ill in response to a stressor. Explain why this statement is or is not true.
2. Describe the physical assessments that are found in the severely dehydrated client.
3. Outline a teaching plan for Mr. Sardoff with foods for a high-sodium, low-potassium diet.

See Evaluating Your Response in Appendix C.

- Weigh the client daily at the same time and in the same clothing. *Dehydration is manifested by weight loss.*
- Encourage an oral fluid intake of 3000 mL per day and an increased salt intake. *Cortisol deficiency increases fluid loss, leading to extracellular fluid volume depletion. Oral fluid replacement is necessary to balance this loss. An increase in dietary sodium can decrease the hyponatremia characteristic of adrenal insufficiency.*
- Teach to sit and stand slowly, and provide assistance as necessary. *Extracellular fluid volume deficit causes orthostatic hypotension, dizziness, and possible loss of consciousness. These manifestations increase the risk of injury from falls.*

PRACTICE ALERT
Hyperkalemia causes changes in cardiac muscle function, which are reflected in ECG changes.

Risk for Ineffective Therapeutic Regimen Management

Clients with Addison's disease must learn to provide lifelong self-care that involves varied components: medications, diet, and recognizing and responding to responses to stress. Changes in lifestyle are difficult to maintain permanently.

- Teach the effects of illness and treatment. Discuss client and family concerns. *Lack of knowledge about the illness, as well as the possibility of complications from disregarding or altering the treatment, can negatively affect compliance.*
- Include the following in the teaching plan:
  - Self-administration of steroids
  - The importance of carrying at all times an emergency kit containing parenteral cortisone and a syringe/needle
  - Wearing a Medic-Alert bracelet that says "Adrenal insufficiency—takes hydrocortisone"
  - Increasing oral fluid intake

- Maintaining a diet high in sodium and low in potassium
- The necessity of altering the medication dose when experiencing emotional or physical stressors
- The importance of continuing health care.

One of the most important components of caring for the client with Addison's disease is teaching both the client and family to provide care. The length of treatment and the side effects of medications can discourage compliance.

Community-Based Care

The client with Addison's disease provides self-care at home. One of the most important components of caring for the client with Addison's disease is teaching both the client and family to provide care. Family stability, an awareness of the serious nature of the disease, and the effectiveness of treatment all promote compliance. The length of treatment and the side effects of medications, however, can discourage compliance. In addition to the information in the teaching topics included with nursing diagnoses and interventions, include the following topics:

- The importance of continuing health care
- Referral to social worker, if appropriate
- Referral to community agencies for continued education and support
- Helpful resources:
  - National Institute of Diabetes and Digestive and Kidney Diseases (Addison's disease)
  - Endocrine Society
  - American Association of Clinical Endocrinologists.

MEDIALINK Addison's Disease Resources

THE CLIENT WITH PHEOCHROMOCYTOMA

Pheochromocytomas are tumors of chromaffin tissues in the adrenal medulla. These tumors, which are usually benign, produce catecholamines (epinephrine or norepinephrine) that stimulate the sympathetic nervous system. Although many organs are affected, the most dangerous effects are peripheral vasoconstriction and increased cardiac rate and contractility with resultant paroxysmal hypertension. Systolic blood pressure may rise to 200 to 300 mmHg, the diastolic to 150 to 175 mmHg. Attacks are often precipitated by physical, emotional, or environmental stimuli. This condition is life threatening.

A pheochromocytoma is diagnosed by increased catecholamine levels in the blood or urine, by x-ray studies, and by surgical exploration. Vanillylmandelic acid is the end product of metabolism of all catecholamine substances and is collected in a 24-hour urine. Because catecholamine secretion is episodic, a 24-hour urine is a better surveillance method than serum catecholamines (Pagana & Pagana, 2002). Surgical removal of the tumor(s) by adrenalectomy is the treatment of choice.

DISORDERS OF THE PITUITARY GLAND

The pituitary gland produces hormones that affect multiple body systems through regulation of endocrine function. Target tissues include the thyroid, adrenal cortex, ovary, uterus, mammary glands, testes, and kidneys. Disorders result from an excess or deficiency of one or more of the pituitary hormones due to a pathologic condition within the gland itself or to hypothalamic dysfunction.

Although disorders of the pituitary cause diverse and serious problems, they are not as common as disorders of other endocrine glands. Hyperpituitarism and hypopituitarism are discussed in this section.

THE CLIENT WITH DISORDERS OF THE ANTERIOR PITUITARY GLAND

Hyperfunction of the anterior pituitary gland, characterized by excess production and secretion of one or more trophic hormones, is usually the result of a pituitary tumor or pituitary hyperplasia. The most common cause of hyperpituitarism is a benign adenoma. The manifestations result from pressure on the optic nerve causing visual changes or an excess of growth hormone (GH), prolactin (PRL), ACTH, or TSH. Typically, 70% to 90% of the anterior pituitary is damaged before clinical manifestations develop (Porth, 2005).

Hypofunction of the anterior pituitary gland results in a deficiency of one or more of the gland's hormones. Conditions causing hypopituitarism include pituitary tumors, surgical removal of the pituitary gland, radiation, and pituitary infarction, infection, or trauma.

Pathophysiology and Manifestations

Growth hormone (also called somatotropin) is produced by cells in the anterior pituitary throughout life. GH is necessary for growth and also contributes to metabolic regulation. GH stimulates all aspects of cartilage growth, and one of its major effects is to stimulate the growth of the epiphyseal cartilage plates of long bones. In addition, other body tissues respond to the metabolic effect of GH with increases in bone width and the growth of visceral and endocrine organs, skeletal and cardiac muscle, skin, and connective tissue. Gigantism and acromegaly (discussed next) result from overstimulation. Growth retardation and short stature result from deficient production of GH.

Hypersecretion of PRL affects reproductive and sexual function. Women may have irregular or absent menses, difficulty becoming pregnant, and decreased libido. Men may be impotent and have decreased libido. PRL deficiency in postpartal women causes a failure to lactate.

An excess secretion of ACTH overstimulates the adrenal cortex, which in turn increases secretion of adrenal hormones. The result is Cushing's syndrome. Deficiencies of TSH are uncommon, but cause hypothyroidism.

Gigantism

Gigantism occurs when GH hypersecretion begins before puberty and the closure of the epiphyseal plates. The person becomes abnormally tall, often exceeding 7 ft (213 cm) in height, but body proportions are relatively normal. Most often the result of a tumor, the condition is rare today as a result of improved diagnosis and treatment.

Acromegaly

Acromegaly, which literally means "enlarged extremities," occurs when sustained GH hypersecretion begins during adulthood, most commonly because of pituitary tumors. As a result of constant stimulation, bone and connective tissue continue to grow. The forehead enlarges, the maxilla lengthens, the tongue enlarges, and the voice deepens (Figure 19–4 ■). Overgrowth of bone and soft tissue in the hands and feet causes clients to buy increasingly larger rings, gloves, and shoes. This condition is different from Marfan's syndrome, a genetic disorder that results in elongated bones, optic changes, and severe cardiovascular effects. (See Chapter 32 for nursing care of clients with cardiovascular disorders.)

Other manifestations include peripheral nerve damage from entrapment of nerves, headache, hypertension, congestive heart failure, seizures, and visual disturbances. Impaired glucose tolerance and diabetes may also occur. Arthralgias develop secondary to the bone and connective tissue growth and may be relieved by treatment that halts excessive GH production (Lehne, 2004; Munzer & Fiebach, 2002).

INTERDISCIPLINARY CARE

Acromegaly is treated by surgical removal or irradiation of the pituitary tumor. A transsphenoidal or transfrontal surgical procedure is most commonly used. Octreotide (Sandostatin) suppresses the anterior pituitary gland and decreases GH levels. It is expensive and gastrointestinal side effects are common for the first couple of weeks. About 25% of patients develop cholesterol gallstones within a year (Lehne, 2004).

NURSING CARE

Clients with anterior pituitary disorders require interventions to help in coping with physical and emotional changes, as well as to prevent complications involving other organs and functions of the endocrine system. Nursing care for the client having cranial surgery is discussed in Chapter 44 ∞.

THE CLIENT WITH DISORDERS OF THE POSTERIOR PITUITARY GLAND

Disorders of the posterior pituitary are related primarily to excessive or deficient antidiuretic hormone (ADH) secretion. The disorders discussed here are the syndrome of inappropriate ADH secretion and diabetes insipidus.

Pathophysiology and Manifestations

Antidiuretic hormone is secreted in response to serum osmolality, which is monitored by osmoreceptors in the hypothalamus. When a condition of hyperosmolality occurs, ADH secretion increases, and renal water is reabsorbed. Hypo-osmolality causes the suppression of ADH, and renal water excretion increases.

Syndrome of Inappropriate ADH Secretion

The **syndrome of inappropriate ADH secretion (SIADH)** is characterized by high levels of ADH in the absence of serum hypo-osmolality. This disorder is most often caused by the ectopic production of ADH by malignant tumors (e.g., oat cell carcinoma of the lung, pancreatic carcinoma, leukemia, and Hodgkin's disease). A transient form may follow a head injury, pituitary surgery, or the use of medications such as barbiturates, anesthetics, or diuretics.

Figure 19–4 ■ Manifestations of acromegaly. Progressive alterations in facial appearance include enlargement of the cheekbones and jaw along with thickening of soft-tissue structures such as the nose, lips, cheeks, and the flesh above the brows.

Courtesy of Clinical Pathological Conference, American Journal of Medicine.

Manifestations of SIADH occur as a result of water retention, hyponatremia, and serum hypo-osmolality. Blood volume expands, but the plasma is diluted. Aldosterone is suppressed; as a result, renal excretion of sodium increases. Water moves from the hypotonic plasma and the interstitial spaces into the cells.

Manifestations of SIADH (see Chapter 10 ∞) are usually nonspecific but are related to hyponatremia and water intoxication. Brain cells swell, causing neurologic symptoms including headache, changes in mental status or personality, lethargy, and irritability. Weight gain results from the retention of fluid (Held-Warmkessel, 2005). Usually no edema is present, because water is distributed between the intracellular and extracellular spaces.

Treatment addresses the low serum sodium and intracellular swelling. Besides keeping the client safe, nursing care involves teaching the client about restricting fluids to 1 L/day. Fluid restriction continues for 3 to 10 days until the malignant source of ADH is successfully destroyed. Demeclocycline (Declomycin) is a tetracycline antibiotic with the unique property of creating excessive urine flow. It is used as a treatment for SIADH (Lehne, 2004).

Diabetes Insipidus

Diabetes insipidus is the result of ADH insufficiency. The two types are as follows:

- *Neurogenic diabetes insipidus* can either result from a disruption of the hypothalamus and pituitary gland (as from trauma, irradiation, or cranial surgery) or be idiopathic.
- *Nephrogenic diabetes insipidus* is a disorder in which the renal tubules are not sensitive to ADH. This may be familial in origin or the result of renal failure.

Diabetes insipidus may result from brain tumors or infections, pituitary surgery, cerebral vascular accidents, and renal and organ failure. It is also a complication of closed-head trauma with increased intracranial pressure.

A deficit of ADH causes excretion of large amounts of dilute urine (*polyuria*), in some instances as much as 12 L/day. The client has extreme thirst and drinks large volumes of water (*polydipsia*). If unable to replace the water loss, the client becomes dehydrated and hypernatremic. Even though hyperosmolality is present, the urine is dilute and has a low specific gravity.

If this disorder is caused by cerebral injury, symptoms commonly appear 3 to 6 days after the initial injury and last for 7 to 10 days. If the increased intracranial pressure is relieved, symptoms of diabetes insipidus usually disappear. However, diabetes insipidus may also be a chronic illness requiring lifelong treatment and care. See Table 19–5 for a comparison of posterior pituitary gland disorders.

INTERDISCIPLINARY CARE

SIADH is treated by correcting underlying causes, treating the hyponatremia with intravenous hypertonic saline, and restricting oral fluids to less than 800 mL/day.

Diabetes insipidus is also treated by correcting underlying causes, if possible. Other medical interventions include administering intravenous hypotonic fluids, increasing oral fluids, and replacing ADH hormone. Desmopressin acetate, administered intranasally, orally, or parenterally, is the treatment of choice (Tierney et al., 2005).

NURSING CARE

Nursing care for the client with SIADH and diabetes insipidus focuses on client problems with fluid and electrolyte balance, as discussed in Chapter 10 ∞.

TABLE 19–5 **Comparison of Posterior Pituitary Gland Disorders**

SIADH	DIABETES INSIPIDUS
Excessive ADH	Deficient ADH
Fluid volume excess	Fluid volume deficit
Restrict fluid intake	Encourage fluid intake
Demeclocycline (Declomycin) (oral agent) causes excessive urination	Desmopressin (DDAVP) (nasal spray) causes increased water reabsorption

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CHAPTER HIGHLIGHTS

- Hormones regulate growth, development, and metabolism. Homeostasis is dependent on a balanced level of each type of hormone. Not only do hormones affect organ function, they interact and when excesses or deficits occur, signs and symptoms are manifested.
- The pituitary gland, in conjunction with the hypothalamus, is the master gland of the body. Fifteen hormones and regulatory factors are synthesized in the anterior pituitary and hypothalamus; many are trophic hormones that stimulate the release of other hormones.
- Thyroid disorders are the most common endocrine disorders. Occurring mainly among women, these diseases change body image and impose upsets to energy levels creating fatigue and exhaustion.
- Diagnostic tests and therapies are available to identify and treat thyroid disorders. Surgery, radiation therapy, and medications support good quality of life, but the medications must be used throughout the lifetime.
- The parathyroid glands synthesize parathormone, which maintains serum calcium. Thyroid tissues surrounding the parathyroids synthesize a hormone, calcitonin, which decreases serum calcium to normal levels. These glands provide the proper level of serum calcium, which is vital for cardiac function, bone stability, nerve conduction, and muscle contraction.
- The adrenal glands regulate energy and fluid balance through corticosteroids and mineralocorticoids. Cushing's and Addison's disease are polar opposites and treatment eliminates the signs and symptoms of one and creates the manifestations of the other. Clients with these diseases require patient education until they fully grasp the significance of the condition and the importance of adhering to the treatment plan.

TEST YOURSELF NCLEX-RN® REVIEW

1 Graves' disease, the most common cause of hyperthyroidism, is categorized as what type of disorder?
   1. autoimmune
   2. infectious
   3. allergic
   4. genetic

2 What principle supports the treatment of hyperthyroidism with radioactive iodine?
   1. Radioactive iodine reduces the vascularity of the thyroid gland.
   2. Doses of radioactive iodine are too small to be hazardous to other body parts.
   3. The thyroid gland takes up iodine in any form.
   4. Irradiation of the thyroid gland decreases the risk of hypothyroidism.

3 You assess a client with newly diagnosed hypothyroidism as having an enlarged thyroid gland (goiter). What physiologic process causes this enlargement?
   1. an excess of TH stimulates thyroid follicles
   2. an increased dietary iodine intake
   3. a compensatory effort to produce more TH
   4. tissue hypertrophy in response to increased TH

4 Mrs. Jonah has taken cortisone for her rheumatoid arthritis for several years. What endocrine disorder is she most at risk for developing?
   1. hyperthyroidism
   2. hypothyroidism
   3. acromegaly
   4. Cushing's syndrome

5 Which statement illustrates that the client with Addison's disease understands your teaching?
   1. "I will be sure to stop taking my medications when I have an infection."
   2. "I have purchased an emergency kit and keep it with me all the time."
   3. "I know I should never alter my dose of medications."
   4. "I wonder why I look suntanned all the time."

6 Signs and symptoms of hyponatremia found in SIADH include:
   1. weight loss.
   2. irritability.
   3. hyperkalemia.
   4. constipation.

7 A home health nurse is caring for a client with hyperparathyroidism and osteoporosis. Which nursing diagnosis has priority with this client?
   1. *Risk for Fear*
   2. *Risk for Injury*
   3. *Risk for Isolation*
   4. *Risk for Chronic Low Self-Esteem*

8 A nurse is monitoring a client for signs of hypercalcemia. Which of the following is a sign of hypercalcemia?
   1. oliguria
   2. positive Chvostek's sign
   3. diminished bowel sounds
   4. hyperactive deep tendon reflexes

9 A client with increased ACTH levels and Addison's disease is likely to manifest:
   1. tremor.
   2. hair loss.
   3. gingival hyperplasia.
   4. dermal hyperpigmentation.

10 Clients treated with glucocorticoids are at risk for Addisonian crisis due to:
   1. rapid withdrawal of glucocorticoids.
   2. excessive ACTH.
   3. sodium retention.
   4. hypokalemia.

See Test Yourself answers in Appendix C.

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