Chapter 3: Disorders of the Endocrine System:
Anatomy, Physiology, and Current Treatment Initiatives

10 Contact Hours

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Learning objectives

- Outline the anatomy and physiology of the endocrine system.
- Identify diseases and disorders of the endocrine system.
- Explain the pathophysiology of diseases and disorders of the endocrine system.
- Identify the diagnostic process for diseases and disorders of the endocrine system.
- Describe treatment initiatives for diseases and disorders of the endocrine system.
- Discuss nursing interventions for patients dealing with diseases and disorders of the endocrine system.

Introduction

Allison is a registered nurse enrolled in a master’s degree program. She hopes to pursue a career in nursing professional development. Allison is studying advanced anatomy and physiology this semester. Her current focus is the endocrine system. Allison is preparing a term paper that she wants to adapt as a continuing education program for the nurses on her unit.

The endocrine system is quite complex and works in conjunction with the nervous system to maintain the delicate balance that ensures homeostasis. It is imperative for nurses to understand how the endocrine system functions and how alterations in functioning can lead to a number of pathologies. This education program provides information on the anatomy and physiology of the endocrine system, disorders of the endocrine system, treatment options for endocrine pathologies, and nursing considerations related to care of patients suffering from such pathologies.

ANATOMY AND PHYSIOLOGY OF THE ENDOCRINE SYSTEM

Components of the endocrine system

The endocrine system, in conjunction with the nervous system, is responsible for regulating and integrating the metabolic activities of the body. It consists of endocrine glands, hormones, and receptors [1,2].

The endocrine glands secrete specific hormones produced by the body to regulate cell and organ activity [3]. The primary glands of the endocrine system are the [1]:

- Pituitary gland.
- Thyroid gland.
- Parathyroid gland.
- Adrenal glands.
- Pancreas.
- Thymus.
- Pineal gland.
- Gonads (ovaries and testes).

The hormones that are secreted by the glands of the endocrine system are chemical messengers that transfer information from one set of cells to another to coordinate bodily functions [3]. Hormones cause changes in the metabolic activities in specific cells while nerve impulses cause gland secretion and muscle contraction. Hormonal action is rather slow, but of prolonged duration. The action of nerve impulses, on the other hand, is rapid but of short duration [2].

Anatomy and physiology alert! There are two types of hormones.

Group I hormones are those that bind to intracellular receptors and are lipophilic (have a strong affinity for lipids) such as the steroid hormones. Group II hormones are those that bind to cell surfaces and are hydrophilic (readily absorbing or dissolving in water) such as polypeptides, glycoproteins, and catecholamines [3].

Receptors, the third component of the endocrine system, are protein molecules. Receptors bind with other molecules (such as hormones) to cause specific physiologic changes in target cells [1].

Negative and positive feedback

The endocrine system depends on both negative and positive feedback for its regulation. Negative feedback takes place when the rate of production of a particular product decreases as the concentration of that product increases. Negative feedback manages the rate of production to avoid accumulation of a particular product. For example, as the amount of some hormones reach the desired level, the body stops or reduces the rate of their production to avoid excessive accumulation [1,3].

Positive feedback occurs when the rate of production of a particular product increases as the concentration of that product increases. Positive feedback is less common in the body than negative feedback. An example of positive feedback is the secretion of oxytocin that stimulates uterine muscle contraction during labor. As labor progresses, pressure on the cervix continues to stimulate oxytocin release, which continues to stimulate uterine muscle contraction [3].
### GLANDS OF THE ENDOCRINE SYSTEM AND THEIR SECRETIONS

#### Pituitary gland

The pituitary gland (also referred to as the hypophysis) is generally considered to be the most important gland of the endocrine system. It is responsible for the production of the hormones that control many functions of other endocrine glands [1,2]. Because of its importance, the pituitary gland is often called the master gland [1].

The pituitary gland is quite small, only about the size of a pea. It is located on the inferior side of the brain in the sella turcica of the sphenoid bone and is attached to the hypothalamus of the brain by the pituitary stalk [3].

The pituitary gland is divided into two primary regions: the anterior lobe (adenohypophysis) and the posterior lobe (neurohypophysis) [1,3].

The anterior pituitary is the larger of the two regions and produces six hormones that are regulated by the hypothalamus [1,2,3]:

- **Growth hormone (GH):** GH, also referred to as somatotropin, stimulates growth of bone and tissue. It accelerates the rate of body growth by stimulating the uptake of amino acid by the cells of the body, increasing tRNA synthesis, and promoting protein synthesis [3]. Deficiency of GH in children causes growth failure. In adults, GH deficiency leads to difficulty maintaining adequate amounts of body fat, muscle, and bone mass. This hormone is also linked to emotional well-being [2].
- **Thyroid-stimulating hormone (TSH):** TSH, or thyrotropin, stimulates the synthesis and release of thyroid hormones from the thyroid gland [1,3]. A lack of thyroid hormones because of a defect in the pituitary gland or in the thyroid itself is called hypothyroidism [2].
- **Adrenocorticotropic hormone (ACTH):** ACTH stimulates the adrenal cortex to produce and secrete various steroid hormones [2,3].

#### Thyroid gland

The thyroid gland is located in the neck immediately below the larynx and partially in front of the trachea. The two lateral lobes of the thyroid are found on either side of the trachea and are joined by a narrow bridge of tissue called the isthmus. This “joining” gives the gland its characteristic butterfly shape [1,2,3].

The thyroid hormones regulate the metabolism of the body and help maintain normal blood pressure, heart rate, digestion, muscle tone, and reproductive functions. The thyroid gland also contributes to bone growth and nervous system development in children [1,2].

The two thyroid lobes function as one unit to produce triiodothyronine (T3), thyroxine (T4), and calcitonin [2]. TSH, which is produced by the pituitary gland, triggers secretion of T3 and T4, which are collectively known as thyroid hormone [1,3].

Thyroid hormone is the major metabolic hormone of the body. It regulates metabolism by increasing the speed of cellular respiration. T3 and T4, referred to collectively as thyroid hormone [3]:

- Increase metabolic rate.
- Increase consumption of oxygen.
- Increase glucose absorption.
- Increase body temperature.
- Affect growth and development.
- Improve the effects of the sympathetic nervous system.

Calcitonin is responsible for maintaining the calcium level of the blood. It accomplishes this by slowing the release of calcium from bone. Calcitonin secretion is controlled by the concentration of calcium in the fluid surrounding thyroid cells [1].

#### Parathyroid glands

The parathyroid glands are the smallest endocrine glands and are embedded in the posterior surface of the thyroid glands [1,3]. These glands work together as one entity and produce parathyroid hormone (PTH) [1].

The primary function of PTH is to regulate calcium balance in the blood by adjusting the rate at which calcium and magnesium ions are lost in the urine. PTH release is stimulated by decreased levels of calcium in the blood [3]. PTH increases plasma calcium levels by [3]:

- Stimulating the formation and action of osteoclasts. Osteoclasts cause the breakdown of bone tissue, which releases calcium from the bones into the blood.
- Triggering kidney tubules to increase calcium reabsorption.
- Facilitating increased calcium absorption from the gastrointestinal (GI) tract.

**Anatomy and physiology alert!** PTH also increases the transport of phosphate ions from the blood to urine for excretion from the body [1].

#### Adrenal glands

The two adrenal glands each lie embedded in adipose tissue on the top of each kidney [1,3]. They are triangular in shape and consist of two distinct structures: the outer adrenal cortex and the inner adrenal medulla. These structures function as separate endocrine glands [1,3].

The majority of the adrenal glands is made up of the adrenal cortex, which has three zones [1]:

- **Zona glomerulosa:** This is the outermost zone of the adrenal cortex. It produces mineralocorticoids (aldosterone and...
The pancreas

The pancreas is a triangular shaped organ located in the abdomen along the curve of the duodenum. It extends from behind the stomach to the spleen [1,3].

The pancreas performs both endocrine and exocrine functions. Its endocrine function is to secrete hormones. Its exocrine function is to secrete digestive enzymes. The pancreas is composed primarily of acinar cells, which regulate pancreatic exocrine function [1,2,3].

The islet cells or islets of Langerhans are the pancreatic endocrine cells. They occur in clusters of cells scattered among acinar cells. The islets contain alpha, beta, and delta cells that produce the following hormones [1,3]:

- Glucagon: Glucagon is produced by the alpha cells. Glucagon is a hormone that stimulates glycogenolysis, which raises the blood glucose level by causing the breakdown of glycogen to glucose. Glucagon helps maintain blood glucose levels during fasting or starvation [1,3].
- Insulin: Insulin is secreted by beta cells that are innervated by adrenergic fibers. Insulin lowers the blood glucose level by stimulating the movement of blood glucose across cells, converting glucose to glycogen [1,3].
- Somatostatin: Somatostatin is secreted by delta cells and inhibits the release of GH, corticotrophin, and certain other hormones [1].

The thymus

The thymus is found below the sternum and contains lymphatic tissue. The thymus, which reaches its maximum size at puberty and then begins to atrophy, secretes the hormones thymosin and thymopoietin. These hormones promote peripheral lymphoid tissue growth [1,3].

Anatomy and physiology alert! The major role of the thymus seems to be related to the immune system since it produces T cells, which are critical to cell-mediated immunity [1].

The pineal gland

The pineal gland is located in the middle of the brain at the back of the third ventricle. It produces melatonin, which is believed to regulate circadian rhythms as part of the sleep-wake cycle, body temperature, cardiovascular function, and reproduction [1,2].

Gonads

The gonads are the primary source of sex hormones and include the ovaries in females and the testes in males [1,2].

- Ovaries: The ovaries are paired, oval-shaped glands located on either side of the uterus. The ovaries produce eggs (ova) and steroid hormones estrogen and progesterone. These hormones promote development and maintenance of female sex characteristics, regulate the menstrual cycle, maintain the uterus for pregnancy, and in conjunction with other hormones, prepare the mammary glands for lactation [1,2].
- Testes: The testes are paired structures located in the scrotum in males. The testes produce spermatozoa and the male sex hormone testosterone, which stimulates and maintains male sex characteristics and incites the male sex drive [1,2].

Hormonal release mechanisms

There are four main mechanisms that control the release of hormones [1,2].

**Pituitary – target gland axis**
The pituitary gland constantly monitors hormone levels. If levels decrease, the pituitary responds by increasing trophic hormones, which trigger their target glands to increase production of specific hormones. Trophic hormones include [1]:

- Corticotropin (regulates adrenocortical hormones).
- TSH (regulates T3 and T4).
- LH (regulates gonadal hormones).

If levels are increased, secretion of trophic hormones decreases [1].

**Hypothalamic-pituitary-target gland axis**
The hypothalamus produces trophic hormones that regulate anterior pituitary hormones. Thus, the hypothalamus controls anterior pituitary hormones, which regulate the hormones of their specific target glands [1].

**Chemical regulation**
Endocrine glands that are not controlled by the pituitary gland might be controlled by substances that stimulate gland secretions. An example of chemical regulation is that of blood glucose levels, which regulate glucagon and insulin release [1].

Nervous system regulation
The central nervous system (CNS) assists in the hormonal regulation by [1]:

- The hypothalamus directly controls the secretion of ADH and oxytocin.
- Nervous system stimuli (such as pain, stress, and some drugs) affect ADH levels.
- The autonomic nervous system (ANS) controls catecholamine secretion by the adrenal medulla.
- Stress stimulates sympathetic stimulation, which, in turn, triggers the pituitary to release corticotrophin.

Anatomy and physiology alert! Normal age-related changes in the endocrine system include decreased progesterone production, a 50% reduction in serum aldosterone levels, and a 25% decrease in cortisol secretion rate. Also, in stressful situations, an elderly person's blood glucose level is higher and remains elevated for a longer period of time than that of a younger adult's [1].
Arguably, with the exception of diabetes and malignancies of the thyroid, diseases and disorders of the endocrine system are not particularly well understood or even well known by many health care professionals (HCPs). This education program provides information about the pathophysiology, diagnosis, treatment, and nursing considerations of nonmalignant diseases and disorders of the endocrine system.

### Hypopituitarism

Hypopituitarism is a complicated clinical syndrome of deficiency in pituitary hormone production. This can occur when disorders affect the pituitary gland, hypothalamus, or surrounding structures [4]. Partial hypopituitarism and complete hypopituitarism (panhypopituitarism) occur in both adults and children. In children, these conditions may lead to dwarfism and delayed puberty. The prognosis may be good with prompt recognition, appropriate replacement therapy, and correction of underlying causes.1

**Hypopituitarism alert!** Panhypopituitarism is characterized by involvement of all pituitary hormones. However, it is more likely that only one or more pituitary hormones are involved. This leads to only isolated or partial hypopituitarism [4].

### Pathophysiology

Hypopituitarism can impair some or all production of the hormones produced or stored by the pituitary. Here is a review of the hormones associated with the pituitary [1,4].

- The anterior pituitary secretes TSH, FSH, LH, GH, ACTH, and prolactin.
- The posterior pituitary stores and secretes two hormones produced by the hypothalamus: vasopressin (ADH) and oxytocin.

The hormones of the pituitary target specific glands to stimulate hormones produced and secreted by those glands (e.g., TSH stimulates the thyroid to produce T3 and T4). Therefore, the function of the pituitary is assessed not by measuring pituitary hormones in isolation, but by the functioning of the target glands [4].

### Etiology

The most common cause of primary hypopituitarism in adults is pituitary tumor or adenomas. Additional causes include traumatic brain injury, infection, irradiation, partial or total hypophysectomy during surgery, chemical agents, and, more rarely, granulomatous disease such as tuberculosis [5]. In children, congenital defects, trauma, or other underlying conditions may lead to the disease [4,5]. Secondary hypopituitarism is caused by a deficiency of releasing hormones produced by the hypothalamus as a result of trauma, infection, tumor, or an unknown cause. Sometimes hypopituitarism may have no identifiable etiology [4,5].

### Clinical presentation

Primary hypopituitarism usually presents as a rather slow, predictable pattern of hormonal failures as the effects of anterior pituitary destruction become evident [4,5]. Clinical presentation depends on the severity of the disease and the number of hormones that are deficient. Clinical features usually begin with hypogonadism due to decreased FSH and LH levels [4,5]. In adults, this causes menstrual cessation in females and impotence in males [4,5]. Other effects include infertility and decreased libido [4].

Birth but initial signs and symptoms appear in the first few months of life, and by 6 months, growth retardation is apparent [5]. Children with dwarfism may appear chubby because of fat deposits in the lower trunk. They experience a delay in secondary tooth eruption and growth generally continues at less than half the normal rate, which can linger into the patient’s 20s or 30s. The average height of these individuals is 4 feet (122 cm), and body proportions are normal [5]. If hypopituitarism occurs prior to puberty, the development of secondary sex characteristics is prevented [4,5]. In males, these include [4,5]:
- Lack of facial and body hair.
- Undersized penis, testes, and prostate gland.
- Failure to initiate and maintain an erection.

In females, these include [4,5]:
- Lack of pubic hair.
- Lack of axillary hair.
- Failure to develop mature breasts.
- Primary amenorrhea.

Panhypopituitarism causes a significant number of both physiological and mental problems including [4,5]:
- Psychosis.
- Lethargy.
- Bradycardia.
- Orthostatic hypotension.
- Anorexia.
- Anemia.

Tumors of the pituitary can cause headache, vision problems (even blindness), and hemianopia. Hypopituitarism related to infection or surgery causes fever, vomiting, hypotension, and hypoglycemia [5].
Hyperpituitarism alert! Clinical signs and symptoms of hypopituitarism do not usually become apparent until 75% of the gland is destroyed [5].

Diagnosis and epidemiology

Hyperpituitarism is a rare disorder. According to the National Institutes of Health (NIH), it affects less than 200,000 persons in the United States and has an international incidence of 4.2 cases per 100,000 annually [4].

In any suspected case of hypopituitarism it is essential to rule out organic, non-endocrine causes of short stature, decreased/delayed growth, and other presenting clinical manifestations. Thyroid function must be carefully evaluated to determine if, for example, decreased levels of thyroid hormone is related to dysfunction of the thyroid, pituitary, or hypothalamus [5,6].

Treatment and nursing considerations

Replacement of hormones secreted by the pituitary’s target glands is essential. Complete loss of all anterior pituitary hormones is fatal without treatment [5]. Treatment also involves treatment of any underlying cause, such as surgical removal of tumors.

Hormone replacement therapy may include the following medications:

- Corticosteroids: Corticosteroids such as hydrocortisone or prednisone are used to replace adrenal hormones that are not being produced because of ACTH deficiency. Corticosteroids are administered orally [5,7].
- Levothyroxine: Levothyroxine (brand names Synthroid, Levoxyl) is administered to replace thyroid hormone caused by low or deficient TSH production [7].
- Sex hormones: For patients of child-bearing age, sex hormone administration may be beneficial. These may include testosterone in men and estrogen or a combination of estrogen and progesterone in women. Testosterone is administered via injection or through the skin with a patch or gel. Estrogen and progesterone can be administered via pills, gel, or patches [7]. Gonadotropins (LH and FSH) can be administered by injection to trigger ovulation in women and sperm production in men [7].
- Growth hormone: GH replacement is recommended for children as well as adults. GH or somatropin is administered as daily subcutaneous injections of one of several recombinant deoxyribonucleic acid GHs. Administration is accompanied by monitoring of serum IGF-1 levels. In children, administration can produce more normal height. In adults, administration of a growth hormone may have some benefit but will not make adults taller [5,7].

There are a number of nursing considerations related to treatment of hypopituitarism, including [4, 5, 6,7]:

- Emphasize the need for life-long hormone replacement. Assess knowledge by having patients and families explain why hormone replacement is necessary, consequences of not taking replacements prescribed, and any possible adverse effects and what to do about them. Have patients/families demonstrate how to accurately take prescribed medications.
- Emphasize the importance of keeping HCPs informed of any and all medications the patient is taking in addition to hormone replacement therapy. Explain that this includes not only prescription medications but OTC (OTC) medications, herbal supplements, vitamins, minerals, weight loss products, and other supplements. Any or all of these products can interact with each other and have the potential to cause adverse side effects.
- Until hormone replacement therapy is complete, monitor patients for signs of thyroid deficiency (lethargy, bradycardia, dry skin and hair, and constipation), adrenal deficiency (weakness, orthostatic hypotension, fatigue, weight loss, and hypoglycemia), and deficiency of gonadotropins (decreased libido, apathy, and lethargy).
- Since anorexia can be a significant problem, initiate a dietary consult to help patients and families develop a diet that patients find appealing. Patients should be monitored for changes in weight.
- Since orthostatic hypotension is a possibility, teach the patient to move slowly when changing positions, especially when going from lying to sitting and sitting to standing.
- Instruct patients to wear a medical identification bracelet.
- Refer the families of children with dwarfism to mental health resources for counseling. This disorder can cause significant stress for patients and families.

Hyperpituitarism

Emily is a registered nurse who works on a busy medical-surgical unit. She attends graduate school two evenings a week. Thanks to her busy schedule she has lost touch with many of her neighbors. As she prepares to leave for school one early-summer evening, she notices her next-door neighbor watering his lawn. As she waves hello she notices that his movements seem slow and painful. Feeling concerned, Emily stops to chat. To her surprise she notices that his facial features seem quite different compared to the last time she saw him. His face seems “bigger” and he seems to be sweating excessively even though the evening is actually rather cool. He tells her that he thinks he must be getting arthritis since his “joints ache.” His voice seems deeper and huskier than Emily recalls, and he mentions that he has made an appointment with “my eye doctor since I don’t see as well as I used to and I am getting awful headaches.” Emily is concerned. She is currently studying endocrine disorders and wonders if her neighbor is suffering from hyperpituitarism.

Hyperpituitarism, also known as acromegaly and gigantism, is a chronic, progressive disease characterized by hormonal dysfunction and disturbing skeletal overgrowth [5]. It occurs when the pituitary gland produces excessive amounts of GH [5,8].

It is important to differentiate between acromegaly and gigantism. The defining difference is the age of persons experiencing pituitary overproduction. Acromegaly affects adults. In adults, the bones increase in size including those of the hands, feet, and face. In children, excessive growth hormone leads to gigantism characterized by exaggerated bone growth and abnormal increases in height [5,8].
Acromegaly occurs after closure of the epiphyseal (rounded end of long bones). This leads to thickening of the bones, transverse bone growth, and visceromegaly (enlargement of the internal abdominal organs) [5].

Giantism starts before epiphyseal closure and causes proportional excessive growth of all body tissues. As the disease advances, loss of gland. The tumors themselves secrete excessive amounts of GH leading to clinical signs and symptoms. Neurological signs and symptoms such as headache and vision disturbances are the result of a tumor pressing on brain tissue [5,8]. Occasionally, non-pituitary tumors can cause hyperpituitarism. For example, tumors of the lungs adrenal glands, or pancreas may secrete GH. In some cases, tumors may produce growth hormone-releasing hormone (GH-RH). This hormone stimulates the pituitary to produce more GH [8].

Incidence and etiology

Acromegaly is a rare disorder that affects males and females equally, usually between the ages of 30 and 50. About three to four people per every million are diagnosed annually [5].

Giantism can affect infants as well as children, but, fortunately, it is a very rare disorder with only 100 reported cases to date. Affected patients may reach as much as three times the normal height for their age. Adults may reach a height of more than 80 inches [5].

In adults, the most common cause of excessive GH production is a tumor. Most of these tumors are benign adenomas of the pituitary gland. The tumors themselves secrete excessive amounts of GH leading to clinical signs and symptoms. Neurological signs and symptoms such as headache and vision disturbances are the result of a tumor pressing on brain tissue [5,8]. Occasionally, non-pituitary tumors can cause hyperpituitarism. For example, tumors of the lungs adrenal glands, or pancreas may secrete GH. In some cases, tumors may produce growth hormone-releasing hormone (GH-RH). This hormone stimulates the pituitary to produce more GH [8].

Signs and symptoms

Acromegaly develops slowly with early signs not readily apparent. It may take years for signs and symptoms to become evident [5,8].

**Acromegaly alert! Since the disease develops so slowly some people may notice physical changes in appearance only by comparing old and current photographs [8].**

One of the most common initial signs of acromegaly is enlargement of the hands and feet. Patients may complain that their shoe size has steadily increased and that their rings no longer fit [8].

Symptoms can vary among patients but some general characteristics are evident. Excessive secretion of GH causes [5,8]:

- Overgrowth of cartilage and connective tissue, which results in a characteristic hulking appearance, enlarged nose, enlarged feet, thickened lips, tongue, fingers, and ears, changes in the shape of the face such as a protruding lower jaw and brow, and wider spacing between the teeth.
- Laryngeal hypertrophy and enlargement of the paranasal sinuses, which causes the voice to sound deep, husky, and hollow.

Diagnosis

Diagnosis is based on patient history, presenting signs and symptoms, and the results of various blood tests. Fasting blood samples for GH and insulin-like growth factor-1 (IGF-1) are obtained. Elevated levels of these hormones suggest acromegaly. However, the results from random samples are not conclusive [5,8].

The definitive test for verifying acromegaly is GH suppression test [8]. Baseline GH and glucose levels are obtained. A prescribed dose of glucose is then administered, after which GH and glucose levels are drawn at 10, 60, and 120 minutes after glucose ingestion [9]. Under normal conditions glucose suppresses GH secretion. So if a glucose infusion does not suppress GH levels to below accepted normal values, and these results are accompanied by characteristic clinical signs and symptoms, a diagnosis of acromegaly is likely [5,8].

**Acromegaly alert! Patients should not be emotionally or physically stressed when obtaining blood samples for the GH suppression test since stress can elevate GH levels [9].**

Various imaging tests may also be performed to diagnosis acromegaly. Skull x-rays, CT scans, MRI, and arteriography are may be used to determine the presence and extent of pituitary lesions. X-rays of the bones can show bone thickening and osteoporosis [5,8].

Treatment and nursing considerations

Treatment focuses on reducing GH production and decreasing the adverse effects of excessive amounts of GH. Surgical removal or reduction of a tumor is essential when severe pathophysiology is present.

Surgical removal of pituitary tumors is complex and should be performed by surgeons who have experience in this procedure. Most of these types of tumors are removed via transphenoidal surgery. During this procedure the surgeon removes the tumor via the nose and sphenoid sinus [5,8]. Tumor removal can normalize GH production, relieve pressure on surrounding tissues, and eliminate signs and symptoms. However, it may not be possible to remove the entire tumor depending on its size and location. In these cases, additional treatment may be necessary [5,8].

**Acromegaly alert! Vital signs and neurological status must be carefully monitored following surgery. Alterations in levels of consciousness, vision disturbances, unequal pupil size, vomiting, elevated blood pressure, or decreasing pulse rate, should be reported**
immediately. These signs may indicate increased intracranial pressure as a result of intracranial bleeding or cerebral edema [5].

If tumor cells remain following surgery radiation therapy may be prescribed to destroy any remaining cells and to gradually reduce GH levels. However, it may take some time, even years, for radiation to cause noticeable improvement in the signs and symptoms of acromegaly [8].

Radiation may be administered in one of two ways [5,8]:
- Conventional radiation therapy: Conventional radiation therapy is usually administered over a 4- to 6-week period. Note that the full effect of this type of radiation therapy may not be achieved for as long as 10 or more years post-treatment.
- Stereotactic radiosurgery: Stereotactic radiosurgery is also known as Gamma Knife radiosurgery. A high dose of radiation directed at the tumor cells is administered in a single dose while limiting radiation exposure to surrounding normal cells. This type of radiation therapy may bring GH levels to within normal limits within 3 to 5 years.

Acromegaly alert! Administration of stereotactic radiosurgery requires an extremely high level of technical skill and is available at only a few U.S. health care facilities. The type of radiation therapy used depends on the size and location of remaining tumor cells and the IGF-I levels [8].

Medications may also be prescribed to lower production or block the action of GH and include the following agents [8]:
- Somatostatin analogues: These drugs (e.g., octreotide and lanreotide) are synthetic forms of the hormone somatostatin. They act by interfering with excessive secretion of GH and promoting reduction in GH levels. Patients are initially injected subcutaneously three times a day with a short-acting form of octreotide to identify any resulting side effects and to evaluate its effectiveness. If the medication is tolerated, a long-acting form is administered intramuscularly once a month. Lanreotide is administered subcutaneously once a month.
- GH antagonists: Pegvisomant, a GH antagonist, acts by blocking its effectiveness. If the medication is tolerated, a long-acting form is administered intramuscularly once a month.
- GH antagonists: Pegvisomant, a GH antagonist, acts by blocking its effectiveness. If the medication is tolerated, a long-acting form is administered intramuscularly once a month.

Diabetes insipidus

Mark is a 35-year-old marketing executive. Since he sits at a desk for the majority of his working hours he enjoys hiking and hiking during his leisure time. One afternoon he takes a nasty fall from his bicycle while cycling with his two young sons. He strikes his head on the sidewalk and sustains what his physician describes as a mild concussion. Several days later Mark experiences an abrupt onset of weakness and dizziness. Mark’s wife insists on taking him to the emergency department where he is diagnosed as having diabetes insipidus.

Diabetes insipidus is a disorder of water metabolism as the result of a deficiency of antidiuretic hormone (ADH), also called vasopressin [5,11]. There are two primary forms of diabetes insipidus: central diabetes insipidus and nephrogenic diabetes insipidus [10].
- Central diabetes insipidus: Also called neurogenic, pituitary, or neurohypophyseal, central diabetes insipidus is marked by a decreased secretion of ADH.
- Nephrogenic diabetes insipidus: Nephrogenic diabetes insipidus is characterized by a decreased ability to concentrate urine because of resistance to the action of ADH in the kidneys.

Diabetes insipidus alert! Nephrogenic diabetes insipidus is a rare congenital disturbance of water metabolism due to renal tubular resistance to ADH (vasopressin)/[5].

Incidence and etiology

Diabetes insipidus may begin in childhood or early adulthood and is more common in males than in females. The disease is characterized by excessive urination and excessive thirst accompanied by significant fluid intake [5,11]. Incidence is slightly higher today than in the past, but it is still a rare disorder affecting one in 25,000 people [5].

Central diabetes insipidus is the result of [5,11]:
- Intracranial neoplastic tumors.
- Metastatic lesions.
- Surgical removal of the pituitary or other neurosurgery.
- Skull fracture.
- Head trauma.

Nephrogenically, diabetes insipidus occurs due to [5,11]:
- Infection.
- Granulomatous disease.
- Vascular lesions.

In some cases, the exact cause of the disease may not be able to be identified [5].

Nephrogenic diabetes insipidus is the result of a defect in the areas of the kidneys that reabsorb water back into the bloodstream. It is less common central diabetes insipidus [5,11].
Nephrogenic diabetes insipidus may also be due to [5,10]:
- An inherited disorder wherein boys inherit the abnormal gene on the X chromosome from their mothers.
- Renal disease.
- Effects of drugs such as lithium.
- Hypercalcemia.

Diabetes insipidus alert! Gestational diabetes insipidus occurs during pregnancy when an enzyme produced by the placenta destroys the mother’s ADH [5].

Pathophysiology
Under normal conditions ADH is manufactured in the hypothalamus and stored in the posterior pituitary gland. When serum osmolality increases and circulating volume decreases, ADH is released into the general circulation. ADH increases water permeability of the distal and collecting tubules of the kidneys. This leads to reabsorption of water and increased serum osmolality and increased circulating volume. These changes cause the release of ADH to stop [11].

Diagnosis
Diagnosis is based on history, signs and symptoms, and various diagnostic tests.

Characteristic signs and symptoms of diabetes insipidus include [5,11]:
- Abrupt onset of extreme polyuria generally between 4 to 6 L/day. However, urinary output may be as high as 30 L/day.
- Extreme thirst.
- Nocturia.
- Extreme fatigue as the result of lost sleep due to nocturia.
- Dehydration.
- Poor skin turgor.
- Muscle weakness.
- Constipation.
- Dizziness.
- Hypotension.

Symptoms usually have an abrupt onset, starting within one to two days after surgery, skull fracture, or stroke. As cerebral edema or increased intracranial pressure is relieved, symptoms stop as quickly as they began [5].

A number of complications are associated with prolonged polyuria. These include [5,11]:
- Hypovolemia.
- Hyperosmolality.
- Circulatory collapse.
- Loss of consciousness.
- CNS damage.
- Bladder distension.
- Enlarged calyces.
- Hydroureter (distention of the ureter).
- Hydronephrosis (collection of urine in the kidney).

Various diagnostic tests may help in the diagnosis of diabetes insipidus. Urinalysis reveals [5,11]:
- Almost colorless urine.
- Low urine osmolality (50 to 200 mOsm/kg of water, less than that of plasma).
- Low specific gravity (less than 1.005).

Additional diagnostic study results that suggest diabetes insipidus include [5,11]:
- Serum osmolality of 300 mOsm/kg.
- Serum sodium of 147 mEq/L.

Dehydration (or water deprivation) test is performed to differentiate ADH deficiency from other types of polyuria by comparing urine osmolality after dehydration and after ADH administration [5,11]. The dehydration test procedure involves [5,11]:
- Baseline vital signs, weight, and urine and plasma osmolalities are obtained.
- Patients are deprived of fluids. They must be monitored to be sure that they do not drink any fluids.
- Urine output, body weight, urine osmolality, urine specific gravity, and plasma osmolality are measured hourly.
- Vital signs are monitored for the duration of the test to detect orthostatic hypotension.

Diabetes insipidus is diagnosed if “the increase in urine osmolality after ADH administration exceeds nine percent [11, pg 261].”

Patients with pituitary diabetes insipidus have decreased urinary output and increased urine specific gravity. Patients with nephrogenic diabetes have no response to the vasopressin administration [5,11].

Plasma or urinary evaluation of ADH may also be performed. Fluid restriction or the infusion of hypertonic saline infusion are conducted to establish if the origin of the diabetes insipidus is the result of damage to the posterior pituitary gland (neurogenic) or failure of the kidneys to respond to ADH (nephrogenic). In neurogenic diabetes insipidus ADH levels are decreased. In nephrogenic diabetes insipidus ADH levels are elevated [11].

Treatment and nursing considerations
Prognosis for patients with diabetes insipidus is generally good, depending on the underlying cause [5,10]. Mild cases of diabetes insipidus may require no treatment other than fluid replacement. Severe cases require that the underlying cause be identified and corrected or treated satisfactorily. Until this is accomplished, various types of vasopressin or of a vasopressin stimulant are administered to control fluid balance and to prevent dehydration [5,11].
There are several medications that can be used to treat diabetes insipidus, including:

- Aqueous vasopressin is used as part of the initial management of diabetes following head trauma or neurological procedure. The drug is administered subcutaneously or intramuscularly several times a day because it is only effective for 2 to 6 hours [5,11].
- Desmopressin acetate (DDAVP) is a synthetic, long-acting vasopressin analogue that is effective for 8 to 20 hours. It is administered via nasal spray and is absorbed through the mucous membranes. DDAVP can also be given subcutaneously, intravenously, or orally in tablet form administered at bedtime or in divided doses [5,11].
- Lypressin is a synthetic vasopressin replacement. It is administered as a short-acting nasal spray. However, there are several side effects associated with the drug that can be problematic. These include nasal congestion, nasal irritation, ulceration of nasal passages, substernal tightness of the chest, coughing, and dyspnea with large doses. Additionally, the drug has a variable absorption rate [11].

**Treatment alert!** If nephrogenic diabetes insipidus is caused by medication, discontinuing the medication allows the kidneys to recover [5].

The prognosis is good for patients who have uncomplicated diabetes insipidus as long as they receive adequate fluid replacement. But the presence of a serious underlying cause (such as cancer) can alter the prognosis depending on how successful treatment initiatives prove to be [5,11].

Nursing care emphasizes meticulous monitoring of intake and output, patient safety, and patient/family education. It is essential to facilitate fluid intake to prevent severe dehydration. Patients must be weighed daily and vital signs monitored carefully. Nurses must also be alert for the development of signs of hypovolemic shock such as cool, clammy skin, anxiety, confusion, rapid breathing, and generalized weakness [5,11].

**Patient safety actions and education initiatives include [5]:**

- Instruct patients/families that patients may be weak and/or dizzy. They should be helped to ambulate as needed and cautioned to change positions slowly, especially when moving from lying to sitting or standing positions.
- Provide, and teach patients/families to provide, meticulous skin and mouth care since skin and mucous membranes may become dry and cracked.
- Explain that constipation is a possibility. Encourage the addition of more high-fiber foods to the patient’s diet.
- Teach patients/families how to monitor patients’ intake and output.
- Instruct patients/families to report weight gain since this may indicate the need for a decrease in medication dosage.
- Teach patients/families to report any return of polyuria. This may indicate that the dosage of medication is too low.
- Teach patients to wear a medical alert bracelet.
- Instruct patients to carry their medication with them at all times.
- Explain the importance of adhering to prescribed medication regimens. As previously mentioned, instruct patients never to stop taking medication unless told to do so by their HCPs. They also need to be instructed to apprise their HCPs of any medications they are taking including OTC medications, herbs, vitamins, minerals, and weight loss products.
- Ask patients/families to demonstrate their knowledge of their medications by having them explain how to take medications, side effects, and what to do if side effects occur. Patients/families should demonstrate how to safely administer their medications.

### Childhood hypothyroidism

**Jack and Maura are the proud parents of a baby girl, their first child. Maura gave birth yesterday afternoon and is preparing for discharge. Her obstetrician and the baby’s pediatrician enter her room. They explain that as part of the routine newborn screening program, Maura’s baby has been evaluated for congenital hypothyroidism. Results of the screening, unfortunately, show that the baby has congenital hypothyroidism.**

#### Incidence and etiology

There are several types of hypothyroidism that affect children, including:

- **Congenital hypothyroidism (CH):** Occurs when the thyroid gland fails to develop or function normally before birth [12].
- **Acquired hypothyroidism-autoimmune hypothyroidism:** Occurs as the result of an autoimmune disorder called chronic lymphocytic thyroiditis (CLT). In CLT the child’s immune system “attacks” the thyroid gland, causing damage and reduced functioning. Patients who have other types of autoimmune diseases (most often insulin-dependent diabetes) are at higher risk for developing CLT. It is estimated that 20% to 30% of people with diabetes will develop CLT. Thus, annual screening for CLT is often a routine part of diabetes care [12].
- **Acquired hypothyroidism-latrogenic hypothyroidism:** Occurs in people who have had their thyroid glands surgically removed or medically destroyed [12].

CH is a common problem, occurring in about one in every 2,500 to 3,000 babies. Currently, all states in the United States test for CH as part of the routine newborn screening initiatives [12].

Infantile cretinism (congenital or neonatal hypothyroidism) is three times more common in females than in males. Early diagnosis and treatment are essential for the best possible patient outcomes. If treatment begins before the age of 3 months the infant usually experiences normal growth and development. However, if treatment is not initiated within that timeframe and children remain untreated beyond the age of 2, irreversible mental retardation occurs. However, skeletal abnormalities are reversible with treatment [5].

Infant cretinism is most often the result of defective embryonic development of the thyroid gland. The next most common cause of the disorder is related to an inherited enzymatic defect in the synthesis of thyroxine (T4). Less often, anti-thyroid drugs administered during pregnancy cause cretinism in infants. Cretinism in children over the age of 2 is usually due to chronic autoimmune thyroiditis [5].

Complications of untreated hypothyroidism in children are severe mental retardation and skeletal malformations including dwarfism and bone and muscle dystrophy [5].
Clinical presentation

At birth, the weight and length of the newborn with infantile cretinism appear normal. However, by the age of 3 to 6 months, the infant displays characteristic signs of hypothyroidism [5].

**Hypothyroidism in children alert!** Breast-fed infants with infantile cretinism experience a delayed onset of symptoms because breast milk contains small amounts of thyroid hormone [5].

Typical characteristics of hypothyroidism in children include the following signs [5,12]:
- The infant:
  - Sleeps excessively and is inactive.
  - Seldom cries, but when he/she does, the cry is hoarse.
  - Has a lowered metabolism and progressive mental impairment.
  - Exhibits abnormally deep tendon reflexes and hypotonic abdominal muscles.
  - Has a puffy and swollen face and droopy eyelids.

Diagnosis

Diagnosis is based on the results of thyroid function screening, which measures thyroid hormone and serum TSH levels. Hypothyroidism is diagnosed when TSH levels are above normal and thyroid hormone levels are below normal [12].

Treatment and nursing considerations

Hypothyroidism in children (as well as adults) is generally treated with thyroid hormone replacements. Replacement therapy for children who are less than age one involves administration of oral levothyroxine. Initial doses are of moderate strength and are gradually increased to levels appropriate for life-long maintenance [5,12].

**Hypothyroidism treatment alert!** A too rapid increase in the dosage of thyroid hormone can trigger thyrotoxicity. Signs and symptoms of thyrotoxicity include tachycardia, vomiting, hypotension, tremor, weakness, shortness of breath, cough, swollen extremities, and coma. Thyrotoxicity can reach crisis levels (thyrotoxic crisis or thyroid storm) and is fatal without treatment that includes anti-thyroid medications, correction of electrolyte imbalance, and treatment of any cardiac arrhythmias [5,11].

Note that doses of thyroid replacement therapy are higher in children compared to adults because children metabolize thyroid hormone much more quickly [5].

Parents of children with hypothyroidism need support and encouragement as they learn to deal with their child’s need for life-long treatment and monitoring. Early detection and treatment are essential if mental retardation is to be avoided.

**Hypothyroidism alert!** When working with parents of newborns be alert to any comments they may make about how “quiet” and “good” their babies are. Parents may mistake lack of activity, sleeping for long periods of time, and lack of crying as signs of a “good” baby, when these behaviors may actually indicate hypothyroidism [5].

**Adult hypothyroidism**

Lauren is an RN who is pursuing a doctorate in nursing practice. As part of her work in endocrinology she has been asked to write a hypothetical clinical study of an adult with hypothyroidism. She would like to find something “different” to focus on as part of her research. Lauren decides to develop a case study based on the clinical presentation of an adult who has hypothyroidism caused by chronic autoimmune thyroiditis or Hashimoto’s disease. Hashimoto’s disease is not frequently discussed even though it is a common thyroid gland disorder and should provide Lauren the challenge she is looking for.

Many endocrine orders are not especially prevalent (compared to other diseases and disorders) and may go unrecognized because of HCPs’
lack of knowledge or exposure to such disorders. Even diseases of the thyroid, that are, arguably, more easily recognized and prevalent, can go undetected for lengthy periods of time causing delays in treatment that can not only be frustrating for the patient, but, at times, dangerous as well.

## Incidence and etiology

Hypothyroidism is more common in females than in males and in people with Down syndrome, and frequency increases with age. There has been a significant increase in incidence in the United States among persons aged 40 to 50 [5,11].

There are two classifications of hypothyroidism: primary and secondary. Primary hypothyroidism is due to a disorder of the thyroid gland itself. Secondary hypothyroidism is the result of a failure to stimulate normal thyroid function. [11].

The most common cause of hypothyroidism is the primary form and is usually caused by, in order of frequency [6]:
- Autoimmune disease: Autoimmune disease hypothyroidism is also referred to as chronic thyroiditis or Hashimoto's disease. It occurs at any age but is most often found in middle-aged women and in persons who have a family history of thyroid disease. Autoimmune hypothyroidism is caused by a reaction of the immune system against the body's thyroid gland and affects between 0.1% and 5% of all adults in Western countries. In rare cases this disease is related to other endocrine disorders caused by the immune system such as adrenal insufficiency and type 1 diabetes. Hashimoto's disease begins and progresses slowly, taking months or even years for a diagnosis to be made [13].
- Overtreatment with anti-thyroid drugs: Too large a dose of anti-thyroid drugs (used to treat hyperthyroidism) can lead to hypothyroidism [5,6].
- Subacute thyroiditis: Subacute thyroiditis is a self-limiting, painful inflammation of the thyroid gland. It is most often associated with viral infections [6].
- Lithium therapy: Lithium may decrease thyroid hormone levels [5,14].
- Overtreatment with anti-thyroid drugs: Too large a dose of anti-thyroid drugs (used to treat hyperthyroidism) can lead to hypothyroidism [5,6].
- Secondary hypothyroidism occurs as a result of insufficient secretion of TSH caused by disease or trauma of the pituitary gland [6].

## Pathophysiology

When the thyroid gland fails to produce adequate amounts of thyroid hormone, or when the thyroid is not adequately stimulated to produce thyroid hormone, there is a general reduction in the rate of all physical and mental processes. Cellular metabolic activity decreases, which reduces oxygen consumption. Thus, there is less oxidation of nutrients for energy and less body heat [6].

**Hypothyroidism alert!** Even though hypothyroidism is not an uncommon disorder, it can go unrecognized for long periods of time. This is because initial signs and symptoms can be vague and nonspecific, making recognition and diagnosis a challenge [5,6].

## Complications

There are a number of potential complications of hypothyroidism. These include [5,6]:
- Benign intracranial hypertension.
- Bleeding tendencies.
- Cardiovascular disease such as arteriosclerosis, impaired peripheral circulation, ischemic heart disease, heart failure, and cardiomegaly (enlarged heart).
- Carpal tunnel syndrome.
- Deafness.
- Fertility problems.
- GI problems such as achlorhydria (absence of hydrochloric acid), pernicious anemia, megaloclon (abnormal dilation of the colon), and intestinal obstruction.
- Iron deficiency anemia.
- Psychiatric disturbances.

The most serious and dramatic complication of hypothyroidism is myxedema coma, which commonly causes death. Myxedema coma usually progresses slowly. However, stressors such as infection, trauma, exposure to cold, or myocardial infarction can intensify hypothyroidism, causing myxedema coma to develop abruptly [5,6].

The respirations of patients in myxedema coma are quite depressed, leading to an increase in the partial pressure of carbon dioxide in arterial blood. Cardiac output is decreased, and cerebral hypoxia occurs and progresses. Heart rate slows, and blood pressure drops. The patient becomes hypothermic and stuporous [6].

**Myxedema coma alert!** Myxedema coma is a medical emergency and requires life-saving actions. Patients are admitted to the intensive care unit. Most experts recommend the intravenous administration of thyroid hormones. Electrolyte and volume disturbances must also be corrected [5,6].

## Clinical presentation and diagnosis

The early clinical manifestations of hypothyroidism are vague and nonspecific. These include fatigue, lethargy, unexplained weight gain, menstrual changes, forgetfulness, reduced attention span, constipation, and sensitivity to cold, especially of the hands and feet [5,6].

As the disease progresses, signs and symptoms more characteristic of hypothyroidism become evident. These include [5,6,11]:
- Anorexia.
- Decreased libido.
- Drooping upper eyelids.
- Dry, flaky, thick skin.
- Muscular weakness.
- Thinning, dry hair.

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- Anorexia.
- Decreased libido.
- Drooping upper eyelids.
- Dry, flaky, thick skin.
- Muscular weakness.
- Thinning, dry hair.
Treatment alert! Because initial symptoms are vague it is important that nurses and other HCPs be alert to the possibility of hypothyroidism.

In addition to clinical manifestations, several tests are used to confirm the diagnosis of hypothyroidism.

In primary hypothyroidism (hypothyroidism is due to a disorder of the thyroid gland itself), TSH levels are elevated. In secondary hypothyroidism (hypothyroidism is due to failure to stimulate normal thyroid function as a result of hypothalamic or pituitary insufficiency), TSH levels are decreased [5,6]. Radioimmunoassay shows low T3 and T4 levels [5,6]. Serum cholesterol, alkaline phosphatase, and triglyceride levels are elevated. Normochromic normochromic anemia may be evident [5]. Electrocardiogram (ECG) shows sinus bradycardia, low voltage of QRS complexes, and flat or inverted T waves [6].

Additional signs, symptoms, and complications related to specific body systems eventually develop [5,6,11].

- Cardiovascular system: Elevated cholesterol, arteriosclerotic and ischemic heart disease, heart failure, cardiomegaly, poor peripheral circulation, and pericardial and pleural effusions.
- CNS: Ataxia, intention tremors, carpal tunnel syndrome, gradually progressing mental impairment, and psychiatric disturbances.
- GI system: Achlorhydria (absence of hydrochloric acid), pernicious anemia, adynamic (weak) colon, megacolon, and obstruction of the intestine.
- Hematologic system: Anemia, iron deficiency anemia, and bleeding tendencies.
- Reproductive system: Impaired fertility.
- Senses: Deafness and nystagmus (rapid, involuntary movement of the eyes).

Severe hypothyroidism is referred to as myxedema. Its characteristic traits include thickened facial features, rough, hard, dough-like, cool skin, bradycardia, weak pulse, muscle weakness, delayed reflexes, and sacral and/or peripheral edema. Hyponatremia may also be present, and the thyroid tissue may not be readily palpable [5,11].

Treatment and nursing considerations

Treatment for mild cases of hypothyroidism involves gradual thyroid replacement with levothyroxine (Levoxyl, Synthroid), a synthetic form of the T4 thyroid hormone. This medication is a stable form of the thyroid hormone and is given orally once a day [5,14,15]. Occasionally, iotryonine is given for inadequate T3 levels [5].

Other thyroid hormone replacements are available, but as of this writing are not often recommended for replacement therapy. These medications include desiccated thyroid hormone, T3 (triiodothyronine), and various combinations of thyroid hormones T3 and T4 [15].

For severe cases of hypothyroidism such as myxedema coma it is essential to provide more aggressive quick-acting treatment including [6]:

- Administration of T3 since it acts more quickly than T4. In unconscious patients it is administered via nasogastric tube.
- Administration of sodium levothyroxine (Synthroid) parenterally for the restoration of T4 levels. Parenteral administration continues until the patient regains consciousness.
- Administration of oral thyroid hormone after the patient regains consciousness and is able to swallow oral preparations.
- Initiate steroid therapy if the rapid administration of thyroid hormone triggers adrenal insufficiency.

Treatment alert! It is imperative to monitor vital signs carefully when levothyroxine is administered. Rapid correction of hypothyroidism can trigger cardiac problems. Elderly patients are at particular risk for hypertension and heart failure. Chest pain and/or tachycardia should be reported immediately. Teach patients and families to report any signs of cardiovascular disease such as chest pain and rapid heart rate as well [5].

Nurses must be alert to signs and symptoms of hyperthyroidism after thyroid hormone replacement begins. There is always a danger of over-correction leading to abnormally high thyroid hormone levels. Teach patients and families about these signs and symptoms (restlessness, sweating, and unexplained excessive weight loss) and to report their occurrence to their HCPs immediately [5,6].

Treatment alert! Warn patients and families that thyroid hormone replacement therapy may increase the effects of digoxin and anticoagulants. Teach them to monitor the patient’s pulse and to monitor for signs of bleeding such as bleeding gums and blood in stools [6].

Encourage patients to wear medical alert bracelets at all times. Warn patients and families to take thyroid replacement therapy exactly as prescribed and to never discontinue taking their medication unless told to do so by the prescribing physicians. Emphasize that they must tell any physician or HCP (such as dentists or nurse practitioners) who prescribes medications for them about their hypothyroidism [5,6].

As always, teach patients and families not to take any additional medications or supplements without the approval of the physician who is supervising their hormone replacement therapy. This includes OTC medications, herbal preparations, vitamins, minerals, weight loss products, or any other supplements [5,6].

Treatment alert! Explain to patients and families that patients will need to take life-long hormone replacement therapy. Warn them that replacement therapy must not be discontinued even when they begin to feel better and signs and symptoms begin to subside and resolve [5,6].

Teach patients and families to make sure that patients take good care of their skin, which is generally dry and flaky. Emphasize that special attention should be paid to bony prominences [5].

Make sure that patients and families are aware of the signs and symptoms of worsening hypothyroidism, myxedema, and myxedema coma and to seek immediate medical help if they occur [5,6]. Caution patients and families to report any infection or occurrence of other diseases and disorders since these problems can affect hypothyroidism and medication effectiveness [5,6].

Patients may be concerned about weight gain and begin to diet or to use weight loss products without health care supervision. Warn patients that it is essential to have an adequate nutritional intake. Work with patients and families to plan a well-balanced, low calorie diet to help with appropriate weight loss. Remind them that excessive rapid weight loss may be a sign of hyperthyroidism and to report such an occurrence [5,6].

Constipation is common among patients suffering from hypothyroidism. To reduce and/or prevent constipation encourage adequate fluid intake and a diet high in fiber. Stool softeners may be prescribed, as well as cathartics, as needed [5,6].

Help patients and families access community resources available for support and education. Include reliable, accurate Internet sites as part of this education. Caution patients and families not to believe everything they may read on the Internet as it relates to hypothyroidism. Help patients and families to critique Internet sites for reliability and validity.
Hyperthyroidism

Janice is a 32-year-old fashion consultant. She majored in fashion design with a minor in marketing in college. Janice’s husband, Dennis, is a business major, and it has been their dream to start their own business, establishing an exclusive upscale fashion boutique. Dennis’ parents are prominent members of the community and have agreed to invest in this business venture with the condition that his mother also works in the boutique “to keep an eye on things.” Initially things go well, but as time goes on Janice feels increasingly stressed. Her mother-in-law is constantly criticizing Janice’s decisions and tells her that “my friends will never come here unless you make this a really exclusive establishment.” Dennis is sympathetic but tells Janice that they must be patient until they earn enough money to buy his parents out of their share of the business. Janice begins to lose a considerable amount of weight despite having an increased appetite. She complains of feeling “jittery” and that her “heart pounds every time I have to go to work.” Janice also develops an extreme intolerance to heat. She and Dennis attribute these symptoms to stress. One cold winter afternoon, after listening to another round of her mother-in-law’s criticism, Janice states that she simply can’t stand how hot it is in the boutique. She goes outside and, despite the cold and significant snowfall, stands on the sidewalk in her sleeveless dress in an attempt to become more comfortable. Dennis, summoned from the office by his mother, finds Janice and insists that he take her to see their family physician at once. After listening to Janice’s recent history and conducting a few diagnostic tests, the physician diagnoses Janice with Graves’ disease, a form of hyperthyroidism.

Hyperthyroidism (also referred to as thyrotoxicosis or Graves’ disease) is a metabolic imbalance characterized by excessive amounts of thyroid hormone in the bloodstream [5,6]. Fortunately, with treatment, most patients lead normal lives [5].

Incidence, etiology, and pathophysiology

Hyperthyroidism is more common in women than in men and affects about 2% of the female population [6].

A number of factors can increase the risk for development of hyperthyroidism. These include [5,11,16]:

- **Family history:** It is believed that genetic factors can make people more vulnerable to the disease. There is a marked increased incidence of the disease in monozygotic twins.
- **Gender:** The disease is more prevalent among women.
- **Age:** Most patients are over the age of 40. Only 5% of patients with hyperthyroidism are younger than 15 years of age. The incidence of Graves’ disease, however, is highest between the ages of 30 and 40.
- **Coexistence of other autoimmune disorders:** People with other autoimmune disorders such as type 1 diabetes or rheumatoid arthritis are at increased risk for the development of hyperthyroidism.
- **Smoking:** Cigarette smoking can increase risk for hyperthyroidism.
- **Pregnancy:** Women who are pregnant or who have recently given birth are at increased risk, especially if they also have genetic predisposition to the disease.
- **Stress:** Both physical and emotional stress can trigger the onset of hyperthyroidism.
- **Excessive dietary intake of iodine:** Excessive dietary intake of iodine can trigger hyperthyroidism onset.

The most common type of hyperthyroidism is Graves’ disease, which is characterized by diffuse hyperfunction of the thyroid gland, increased thyroxine (T4) production, enlargement of the thyroid gland, and multiple system changes [5,6]. Graves’ disease is associated with ophthalmopathy, when the tissues and muscles behind the eye become swollen causing the eyelids to protrude. Graves’ disease may subside spontaneously [6]. Its exact cause is unknown but it occasionally coexists with abnormal iodine metabolism and other types of endocrine disorders [5].

The second most common type of hyperthyroidism is toxic adenoma, which is a small, benign nodule in the thyroid gland that secretes thyroid hormone. Its incidence is highest in the elderly, but its etiology is unknown. Toxic adenoma’s clinical manifestations are similar to those of Graves’ disease in many respects, but it does not cause ophthalmopathy [5,11]. Nor does it cause pretibial myxedema (localized skin lesions) or acropachy (soft tissue swelling with underlying bone changes at the site of new bone formation), which are also associated with Graves’ disease [5,11].

Remaining types of hyperthyroidism include [5,11]:

- **Thyrotoxicosis factitia** is a form of hyperthyroidism that is due to chronic ingestion of thyroid hormone. The hormone is ingested by patients with thyroid carcinoma in an attempt to suppress TSH or by patients who are abusing thyroid hormone in an attempt to lose weight.
- **Functioning metastatic thyroid carcinoma** is a rare disease. It causes the thyroid gland to produce excessive amounts of thyroid hormone.
- **A TSH-secreting pituitary tumor** also causes excessive production of thyroid hormone.
- **Subacute thyroiditis** is a granulomatous inflammation of the thyroid that is triggered by a virus. It causes transient hyperthyroidism, fever, pain, pharyngitis, and thyroid gland tenderness.
- **Silent thyroiditis** is a transient form of hyperthyroidism that is self-limiting.

**Hyperthyroidism alert! Clinical hyperthyroidism can be triggered by excessive dietary intake of iodine or by stress in patients who have latent hyperthyroidism [5].**

In Graves’ disease, an autoimmune reaction causes thyroid-stimulating antibodies to bind to and stimulate the TSH receptors of the thyroid gland. The cause of this autoimmune response is unknown. Disease development is associated with genetic factors, other autoimmune disorders, and the production of auto-antibodies formed because of a fault in suppressor T-lymphocyte function [11].

Complications

There are a number of complications associated with hyperthyroidism. These include [5]:

- Muscle weakness.
- Myasthenia gravis.
- Osteoporosis.
- Paralysis.

Cardiovascular complications such as arrhythmias, cardiac insufficiency, and cardiac decompression may occur. Cardiovascular complications are most common in elderly patients [5].
Thyroid storm is the most serious complication of hyperthyroidism. Also referred to as thyrotoxic crisis, thyroid storm usually occurs in patients with preexisting, though often undiagnosed, thyrotoxicosis. Untreated, it is usually fatal [5,11]. When excessive amounts of T3 and T4 are produced systemic adrenergic activity increases, which leads to overproduction of epinephrine. Excessive amounts of epinephrine cause significant hypermetabolism that, in turn, leads to rapid cardiac, GI, and sympathetic nervous system decompensation. Hypertension, tachycardia, vomiting, extreme irritability, and temperature up to 106°F can occur. Thyroid storm can progress to delirium, coma, and death. The onset of thyroid storm is abrupt and triggered by stressors such as trauma, surgery, infection, or serious events such as stroke, myocardial infarction, preeclampsia, or pulmonary embolism [11].

Clinical presentation

The characteristic signs and symptoms of hyperthyroidism are [5,6,11]:

- Enlarged thyroid gland (also referred to as goiter).
- Exophthalmos (abnormally protruding eyes and a characteristic staring gaze).
- Heat intolerance.
- Nervousness.
- Inability to sit still.
- Weight loss even though appetite is increased.
- Diaphoresis.
- Diarrhea.
- Tremors.
- Palpitations.

**Hyperthyroidism alert!** Although exophthalmos is considered by many HCPs to be the most characteristic sign of hyperthyroidism, it is actually absent in many patients with the disease [5].

**Hyperthyroidism alert!** Most of the signs and symptoms of hyperthyroidism are due to an increased metabolic rate, excessive heat production, increased cardiovascular and neuromuscular activity, and sympathetic nervous system hyperactivity [6].

Hyperthyroidism affects every system of the body. Therefore, a multitude of signs and symptoms may be apparent. The following is a review of additional signs and symptoms according to body systems.

- Cardiovascular system: Cardiovascular system effects are seen most often in elderly patients. Cardiac effects include arrhythmias (usually atrial fibrillation), tachycardia with a full, bounding pulse, cardiac insufficiency, visible point of maximal impulse, cardiac decompensation, and resistance to the prescribed therapeutic dose of digoxin in patients who are taking the drug [5,6,11].
- CNS: CNS signs and symptoms are most commonly seen in younger patients. For example, patients may complain of having difficulty concentrating. This is because an increased production of T4 accelerates cerebral functioning. An increase in basal metabolic rate can lead to anxiety, nervousness, mood swings, and emotional instability. Some patients may even develop overt psychosis. Increased activity in the area of the spinal cord that controls muscle tone can lead to tremors, shaky handwriting, and clumsiness [5,11].
- GI system: Anorexia may develop. Patients may complain of nausea and vomiting because of increased GI motility and peristalsis. Patients may notice an increase in the number of stools, soft stools, and/or diarrhea. The liver may become enlarged.
- Integumentary system: Skin is warm, smooth, moist, thick, flushed, and has a velvet-like texture. There is evidence of hyperpigmentation and loss of skin color in blotches (vitiligo). Plaque-like or nodular skin lesions may be noted. The hair is fine and soft and begins to gray prematurely. Hair loss is evident in both men and women. Nails are fragile and there is separation of the distal portion of the nail from the nail bed [11].
- Musculoskeletal system: There is muscle weakness accompanied by muscle atrophy. Osteoporosis and acropachy are also possibilities [11].
- Reproductive system: Women may experience menstrual abnormalities such as oligomenorrhea (abnormally light or infrequent menstrual periods) and amenorrhea (absence of menstruation), impaired fertility, decreased libido, and a higher incidence of spontaneous abortions. Men may develop gynecomastia (abnormal development of mammary glands) due to an increase in estrogen levels. They may also experience a decrease in libido [5,11].
- Senses: Patients blink infrequently as a result of exophthalmos. This leads to dry eyes, reddened conjunctiva and cornea, and corneal ulcers. Patients have difficulty looking upward and strabismus (the eyes do not “line up” at the same time and therefore cannot look at the same object at the same time) [5,11].

Diagnosis

Diagnosis is made based on the presenting clinical picture, a thorough history and physical examination, and evaluation of blood hormone levels [5,6].

**Diagnostic alert!** Although many of the signs and symptoms of hyperthyroidism are deemed to be “characteristic” of the disease, it is important for nurses and other HCPs to be alert to their development. Early recognition and prompt treatment are important.

In hyperthyroidism palpation of the thyroid gland may reveal that the gland is asymmetrical and lobular. It may actually be enlarged to as much as 3 to 4 times its normal size. Enlargement of the liver may also be noted [5,11].

A full, bounding pulse may be palpated along with a heart rate indicative of tachycardia [11].

Evaluation of reflexes may show hyperreflexia [11].

When auscultating the heart, the examiner may detect a rapidly accelerating heart beat that may be confirmed on ECG as paroxysmal supraventricular tachycardia or atrial fibrillation. Other findings may include [11]:

- Systolic murmur.
- Wide pulse pressure.
- Audible bruit over the thyroid gland (may indicate toxicity).

**Diagnostic alert!** Remember that cardiovascular signs and complications are especially likely in elderly patients [5,11].

The following tests are used to confirm a diagnosis of hyperthyroidism [5,6,11]:

- Radioimmunoassay shows elevated serum T4 and T3.
- TSH levels are decreased.
- Thyroid scan shows an increased uptake of radioactive iodine.

**Diagnostic alert!** Thyroid scan is contraindicated if the patient is pregnant [5].

- Ultrasound confirms the presence of subclinical ophthalmopathy.
- In Graves’ disease the thyroid stimulating immunoglobulin is positive.
Treatment and nursing considerations

Treatment initiatives depend on any underlying causes, the size of the goiter, patient age, severity of the disease, and any complications that are present [6].

Anti-thyroid medications

Anti-thyroid medications are used for children, young adults, pregnant women, and for those patients who are unable to tolerate or who refuse other types of treatment [5,6,11,16]. Examples of such medications include propylthiouracil (PTU) and methimazole (Tapazole). They act by depressing the synthesis of thyroid hormone by inhibiting thyroid peroxidase. PTU is given in daily divided doses. Tapazole is given in a single daily dose [6].

Anti-thyroid medications may also be used before or after radioiodine therapy as a supplemental treatment [16]. Side effects of both PTU and Tapazole include rash, joint pain, liver failure, or a decreased white blood cell (WBC) count. Use of Tapazole is associated with a slight risk of birth defects, thus PTU is the preferred drug for use in pregnant women [16].

Treatment continues until the patient becomes clinically euthyroid (having normal thyroid function). This can take from 3 months to 2 years. If normal thyroid function cannot be maintained without therapy, radiation or surgical intervention is recommended [6]. Medications are discontinued gradually to prevent exacerbation [6].

Beta blockers

Beta blockers do not prevent or inhibit thyroid hormone production. However, they do limit the effects of excessive amounts of thyroid hormone on the body. Beta blockers can provide fairly quick relief of some signs and symptoms such as arrhythmias, tremors, anxiety, irritability, diaphoresis, diarrhea, muscle weakness, and heat intolerance [6,16].

Radioactive iodine therapy

Radioactive iodine (radioiodine) acts by limiting secretion of thyroid hormone by destroying thyroid tissue. Given by mouth, dosage of the drug is controlled so that hypothyroidism does not occur. As the thyroid gland shrinks, signs and symptoms decrease gradually over a period of several weeks to several months. Radioactive therapy may increase Graves’ ophthalmopathy. This is usually temporary and mild. However, if the patient is already affected with moderate to severe eye problems, this type of therapy may be contraindicated [6,16].

Radioactive iodine therapy alert! The primary advantage of radioactive iodine therapy is that it can result in a lasting remission of hyperthyroidism. However, use of radioactive iodine therapy can cause the patient to become permanently hypothyroid [6].

While patients are receiving iodine therapy they must be observed for signs and symptoms of iodine toxicity such as swelling of the buccal mucosa, excessive salivation, skin eruptions, and/or coryza (inflammation of the nasal mucous membranes). If side effects occur the use of iodides is discontinued [6].

Surgery

If other therapeutic interventions are not effective surgery (subtotal thyroidectomy) may be necessary. Surgery is also used for patients who have large goiters. Most of the thyroid gland is removed, necessitating life-long thyroid hormone replacement. Risks associated with subtotal thyroidectomy include damage to vocal cords and the parathyroid glands [16].

Treatment of graves’ ophthalmopathy

Mild cases of Graves’ ophthalmopathy are managed by using OTC artificial tears during the day and lubricating gels at night [16]. For severe cases the following interventions may be prescribed [16].

- Administration of corticosteroids: Corticosteroids such as prednisone are given to reduce swelling behind the eyes.
- Eye muscle surgery: Inflammation may shorten the muscles of the eyes, making them too short for the eyes to properly align. During surgery, the surgeon cuts the eye muscles and reattaches them further back in the eye to facilitate alignment. More than one surgical procedure may be needed.
- Orbital decompression surgery: The surgeon removes the bone between the eye socket and the sinuses. This allows the eyes to move back to their normal position. This procedure is indicated if vision loss is possible due to pressure on the optic nerve.
- Orbital radiotherapy: Targeted x-rays, administered over a period of several days, are used to destroy some of the tissue behind the patients’ eyes. This procedure was once quite common. Recent studies, however, suggest that this procedure provides no benefit for patients with mild to moderately severe Graves’ ophthalmopathy.
- Prisms: Prisms in eyeglasses are used to correct double vision because of Graves’ disease. Prisms work for some, but not all, patients affected with double vision.

Emergency treatment of thyroid storm

Thyroid storm is a medical emergency requiring prompt treatment. Treatment initiatives include [5,6]:

- Prevention of new thyroid hormone synthesis with thioamides such as PTU.
- Prevention of thyroid hormone release using iodine (Lugol’s solution).
- Inhibition or control of the side effects of thyroid hormones with corticosteroids and beta blockers such as Inderal.
- Initiatives targeted at the systemic effects of thyroid hormones include the use of a cooling blanket and acetaminophen (Tylenol) for excessive body heat, administration of intravenous fluids and electrolytes to correct dehydration and electrolyte imbalance, and treatment of the trigger event (e.g., heart attack and other physical and emotional stressors).

Nursing actions focus on actions that help patients and families deal with the effects of the disease, compliance with treatment, and knowledge acquisition to help them lead normal lives.
### Environmental considerations

The following interventions are appropriate for both home and hospital environments [5,6]:

- Provide a calm quiet environment to combat anxiety and promote rest.
- Teach patients and families relaxation techniques such as meditation and deep breathing exercises.
- Refer patients and families to community resources for counseling to help deal with emotional stressors.
- Promote sleep and relaxation as much as possible.

### Nutritional needs

Nutritional needs focus on fluid and electrolyte replacement and promotion of a healthy diet [5,6]. Nurses should:

- Monitor intake and output and weight.
- Provide examples of a healthy, yet high calorie diet since weight loss is usually an issue.
- Monitor intravenous infusions and the patency of the intravenous site.
- Monitor lab results pertaining not only to hormone levels but electrolyte levels as well.

### Skin care

Maintaining skin integrity is a priority. Nurses should take, and teach patients and families to take, the following steps to maintain skin integrity [5,6]:

- Monitor skin turgor.
- Monitor for diaphoresis and body odor due to excessive sweating.
- Encourage the patient to bathe frequently with cool water and to change clothing and bed linens when they become damp.
- Avoid soaps that are drying to the skin such as perfumed soaps and shower gels.
- Apply lotion and lubricants to skin, especially boney prominences.
- Monitor skin for reddened or open areas. Teach patients to use a long-handled mirror to check areas of the skin on the back, the buttocks, and behind the legs.

### Adherence to medication regimen

- Caution patients to wear a medical identification bracelet.
- Explain how to take medications. Have patients and families verbalize knowledge of their medication regimens by asking them to state what medication(s) they must take, dose, route, time, action, side effects and what to do if adverse effects occur. Be especially careful to explain signs of hypothyroidism, which may indicate that the doses of their anti-thyroid medications are too high.
- As always warn patients not to discontinue medications unless told to do so by their HCPs and to tell their HCPs providers about any medications they are taking including prescription, OTC, herbs, vitamins, minerals, and weight loss products.
- Most patients affected by hyperthyroidism can lead normal lives. However, they need ongoing, life-long monitoring and adherence to any treatment regimens prescribed. They also need support and contact information for persons and resources that can help them deal with the disease and its effects.

### Thyroiditis

Margaret gave birth to her first child 3 months ago. She has become anxious and irritable and complains of fatigue and, a “racing” heart. Margaret has begun to lose weight even though her appetite is good and she is eating more than she usually does. Her mother and friends laugh at Margaret’s concerns and regale her with stories about how tired and nervous they were after the birth of their first children. “Wait until you have three like me, then you can complain!” one friend tells her. Margaret becomes more and more distraught until one day, she breaks down in tears while attempting to place the baby in his car seat for a visit to his pediatrician. Margaret’s next door neighbor, a retired RN, notices her distress and comes to help. Margaret tells her neighbor about her symptoms and says, “Everyone tells me I’m just over-reacting to being a new mother but I think something is really wrong!” The neighbor volunteers to babysit the next day so that Margaret can visit her family physician. When she returns, Margaret thanks her neighbor profusely. “The doctor says I have an inflamed thyroid gland. She says it doesn’t happen often after giving birth, but that it’s happened to me. Wait until I tell my mother and friends! This will shut them up!”

Thyroiditis is inflammation of the thyroid gland. It is most prevalent in people between the ages of 30 and 50, and is more common in women than in men. The highest incidence is in the Appalachian region of the United States [5].

### Types of thyroiditis

There are several forms of thyroiditis, which usually have three phases: overactive thyroid (hyperthyroidism), underactive thyroid (hypothyroidism), and return to normal [17]. However, not all forms allow for the return of normal thyroid functioning. Some patients need life-long follow-up and thyroid hormone replacement [5,19].

### Postpartum thyroiditis

Postpartum thyroiditis is an uncommon disorder characterized by inflammation of the thyroid gland within the first year following childbirth [18]. Its exact etiology is unknown, but it is associated with an immune system reaction/underlying autoimmune thyroid condition [18,19].

Women at increased risk for postpartum thyroiditis are those who have [18]:

- A history of postpartum thyroiditis.
- A family history of thyroid problems.
- High concentrations of anti-thyroid antibodies.

Most women who develop postpartum thyroiditis experience a return to normal thyroid function within 12 to 18 months of symptom onset. However, some women experience lingering signs and symptoms and can develop permanent complications [18,19].
There are generally two phases of postpartum thyroiditis. The first phase usually occurs within 1 to 4 months after giving birth and lasts for 1 to 3 months [18]. Signs and symptoms of the first phase are caused by inflammation and release of thyroid hormone and include [18]:

- Anxiety.
- Fatigue.
- Increased sensitivity to heat and heat intolerance.
- Irritability.
- Insomnia.
- Palpitations.
- Rapid heartbeat.
- Tremors.

Later in the disease process, thyroid cells become impaired and signs and symptoms of hypothyroidism might become evident such as [18]:

- Aches and pains.
- Constipation.
- Dry skin.
- Fatigue and lack of energy.
- Increased sensitivity to cold and cold intolerance.
- Trouble concentrating.

Postpartum thyroiditis alert! Some women with the disease develop symptoms only of hyperthyroidism or only of hypothyroidism, but not both [18].

Hashimoto’s thyroiditis

Hashimoto’s thyroiditis is a chronic progressive disease of the thyroid gland. It is an autoimmune disorder characterized by thyroid infiltration of lymphocytes. This causes progressive destruction of the parenchyma and hypothyroidism if left untreated. As the immune system “attacks” thyroid gland it gradually swells, and damage is sustained [6,19].

Hashimoto’s thyroiditis is generally thought to be the most common cause of adult hypothyroidism. Its exact etiology is unknown, but it is believed to be genetically transmitted and perhaps related to Graves’ disease. Ninety-five percent of Hashimoto’s thyroiditis cases occur in women in their 40s or 50s, and incidence of the disease is increasing [6].

Clinically, the disease progresses very slowly, taking months or even years to be identified [19]. Clinical manifestations of Hashimoto’s thyroiditis include [6,19]:

- Slow development of a firm, enlarged thyroid gland.
- Low basal metabolic rate.

Subacute thyroiditis

Subacute thyroiditis is a self-limiting, painful inflammation of the thyroid gland that usually occurs following a viral infection [5,6]. The disease is associated with a three-phase clinical course of hyperthyroidism, hypothyroidism, and return to normal thyroid gland functioning. It is estimated that sub acute thyroiditis may be responsible for 15% to 20% of patients presenting with hyperthyroidism and 10% of patients presenting with hypothyroidism [20].

There are three forms of subacute thyroiditis [20]:

- Subacute granulomatous thyroiditis: Also known as subacute painful or deQuervain thyroiditis.
- Lymphocytic thyroiditis: Also known as subacute painless thyroiditis.
- Subacute postpartum thyroiditis: See section on postpartum thyroiditis.

Subacute thyroiditis predominantly affects younger women. Its clinical course is characterized by four stages [20]:

- Stage 1: The disease goes into remission and thyroid hormone levels return to normal.
- Stage 3: At this stage, the thyroid is depleted of colloid and cannot produce adequate amounts of thyroid hormone, which leads to hypothyroidism. This stage may last up to 2 months. The hypothyroidism is usually mild and no thyroid hormone therapy is required unless the patient presents with significant signs and symptoms of hypothyroidism.
- Stage 4: The disease resolves itself and normal thyroid functioning is restored as thyroid follicles regenerate. Ninety percent to 95% of patients experience a return to normal thyroid function.

Subacute thyroiditis alert! About 10% of patients experience permanent hypothyroidism, necessitating long-term or life-long thyroid hormone replacement [6].

General signs and symptoms of sub acute thyroiditis include [6]:

- Pain, swelling, and tenderness of the thyroid gland that lasts for several weeks or months and then disappears.
- Fever, sore throat, referred ear pain.
- Fever, malaise, and chills.
Depending on the stage of the disease, patients may present with symptoms of hyper- or hypothyroidism. Hyperthyroidism symptoms include anxiety, nervousness, irritability, insomnia, weight loss, and heat intolerance. Hypothyroidism symptoms include lethargy, cold intolerance, weight gain, and constipation [5,6].

**Riedel thyroiditis**

Riedel thyroiditis (also known as Riedel’s thyroiditis) is a rare, chronic inflammatory disease of the thyroid gland. The disease is characterized by a “dense fibrosis that replaces normal thyroid parenchyma [21].” The fibrotic process extends to nearby structures of the neck and reaches beyond the thyroid capsule. Function of the thyroid depends on the amount of normal thyroid tissue that has been replaced with fibrotic tissue. Although most patients retain normal thyroid functioning, about 30% of them become hypothyroid [21].

*Riedel thyroiditis alert!* Some experts believe that Riedel thyroiditis is not a disorder of the thyroid gland, but rather is a symptom of the systemic disorder multifocal fibrosclerosis. It is estimated that about 33% of Riedel thyroiditis cases are linked to findings of multifocal fibrosclerosis when diagnosed [21].

Riedel thyroiditis is usually self-limiting, and patients have a favorable prognosis. However, there are some potential complications of the disease including airway obstruction, dysphagia (difficult, painful swallowing), dysphonia (hoarseness), hypothyroidism, hypoparathyroidism, and stridor because of compression of the trachea by the thyroid gland [21].

**Miscellaneous types of thyroiditis**

There are several other types of thyroiditis. These include [19]:

- Acute or infectious thyroiditis: This type of thyroiditis is usually due to a bacterial infection. Symptoms include sore throat, feeling generally sick, enlargement of the thyroid gland, and, occasionally, symptoms of hyperthyroidism or hypothyroidism. Symptoms usually resolve as the infection is treated with appropriate antibiotics.
- Drug-induced thyroiditis: Drug-induced thyroiditis is triggered by various drugs such as interferon (antiviral or immune response modifier), amiodarone (antiarrhythmic), and some anticancer drugs (e.g., sunitinib). Symptoms of hyperthyroidism or hypothyroidism may occur, but these usually resolve when drugs causing them are discontinued.

- Painless thyroiditis: The signs and symptoms of painless thyroiditis are similar to those of postpartum thyroiditis. However, painless thyroiditis can occur in both men and women and is not associated with childbirth. Painless thyroiditis usually causes a stage of high thyroid hormone levels, followed by a phase of low thyroid hormone levels, and, ultimately, a return to normal in about 12 to 18 months.
- Radiation induced thyroiditis: Radiation induced thyroiditis is triggered when radiation iodine treatment is used to treat overactive thyroid glands or for certain cancers. Resulting damage to the thyroid can cause symptoms of high or low levels of thyroid hormone. Hypothyroidism after treatment with radioactive iodine is usually permanent, and life-long thyroid hormone replacement therapy is needed.

**Nontoxic goiter**

Melanie is a 58-year-old partner in a prestigious law firm. After menopause, Melanie gained a bit of weight and has been rigidly dieting and vigorously exercising to maintain a trim figure. She has begun to notice some difficulty swallowing and a slight swelling in the front of her neck. Melanie dismisses these symptoms as annoying and continues with her busy lifestyle. During her annual physical exam her nurse practitioner notices the swelling and recommends some diagnostic laboratory tests. After reviewing the results of the tests and physical exam findings, Melanie is diagnosed with a “simple” goiter.

Goiter is an abnormal enlargement of the thyroid gland. A simple, or nontoxic goiter is enlargement of the thyroid gland that is not due to inflammation or cancer and is not due to abnormal thyroid function [5,22].

Nontoxic goiter is most common in females, particularly during adolescence, pregnancy, and menopause. During these periods of a female’s life, the demand for thyroid hormone increases [11].

*Goiter alert!* Toxic goiter, as compared to nontoxic goiter, stems from long-standing nontoxic goiter and is found in elderly people. Toxic goiter manifests itself as an enlarged thyroid gland that develops small rounded masses and secretes excessive amounts of thyroid hormone [11].

**Pathophysiology**

Nontoxic goiter occurs when the thyroid gland is unable to secrete sufficient thyroid hormone to meet the needs of the body. In an attempt to compensate for this insufficiency, the thyroid gland enlarges. Enlargement usually overcomes mild to moderate hormonal deficiencies [11].

Steps involved in the production of a nontoxic goiter are [5,11]:

- Impaired thyroid hormone synthesis and depletion of glandular organic iodine increase the thyroid glands response to normal levels of TSH.
- Increased response to normal TSH levels is accompanied by increases in thyroid gland mass and cellular activity, which compensate for mild deficiencies in the synthesis of thyroid hormone. Thus, metabolic function is normal even in the presence of a goiter.
- If an underlying disorder does exist or develop, and is severe, both a goiter and hypothyroidism may develop.

Depending on the size of the goiter, dysphagia and even respiratory distress may develop [5].

**Types of nontoxic goiter**

Nontoxic goiter is categorized as either endemic or sporadic.

Endemic goiter affects more than 10% of a population [22]. Its development is usually due to a diet that is inadequate in iodine, which, in turn, leads to inadequate synthesis of thyroid hormone. In Japan, however, goiter due to excessive intake of seaweed-containing iodine has been identified [11].

In the United States, some geographic locations have actually been dubbed “goiter belts” since they have a high incidence of endemic
goiter due to a lack of iodine in soil and water. These areas include
the Midwest, Northwest, and Great Lakes region [11]. However, the
introduction of iodized salt in the United States has drastically reduced
the incidence of the disorder [5].

Sporadic goiter is the most common cause of goiter in the United
States [22]. Sporadic goiter is usually the result of ingestion of large
amounts of goitrogenic foods or drugs. Goitrogenic foods and drugs
are those that contain substances that decrease the production of T4.

Diagnosis of nontoxic goiter

Diagnosis is based on patient history and physical exam. It is
important to rule out other disorders that can cause goiter and have
similar effects but can range from mild to serious and even life-
threatening such as thyroid carcinoma, Graves’ disease, and various
types of thyroiditis [5].

Diagnosis is based on patient history and physical exam. It is
important to rule out other disorders that can cause goiter and have
similar effects but can range from mild to serious and even life-
threatening such as thyroid carcinoma, Graves’ disease, and various
types of thyroiditis [5].

Diagnostic alert! HCPs must always be alert to the influence
of medications and diet on health. As in the case of nontoxic goiter, foods
and medications can be a major influence on its development [5,11].

Since nontoxic goiter does not adversely alter the patient’s metabolic
state, clinical manifestations of the disease develop solely due to thyroid
gland enlargement [5]. Symptoms of nontoxic goiter include [5,11]:
- Dysphagia.
- Respiratory distress.
- Stridor.

There is swelling and neck distention and, if the goiter is large enough,
obstruction of venous return which causes venous engorgement. Rarely,
large goiters may prompt the development of collateral venous
circulation in the chest. Venous obstruction may make the patient dizzy
or trigger syncope when the arms are raised above the head [5,11].

Diagnostic tests show [5,11]:
- Normal or elevated levels of TSH.
- Normal levels of thyroid hormones.

Diagnostic alert! Abnormalities in T3, T4, and TSH rule out a

- Thyroid antibody titers are generally normal. Increases in thyroid
antibody titers suggest chronic thyroiditis.
- Radioactive iodine uptake is usually normal. However, results may
increase if the patient has an iodine deficiency or a biosynthetic
defect.
- Urinalysis results may show low urinary excretion of iodine.

Ultrasound of the thyroid or radioisotope scanning may be used to
identify malignancies or nodules that need to be biopsied [5,11].

Treatment and nursing considerations

Treatment focuses on reduction of thyroid hyperplasia. The treatment
of choice for nontoxic goiter is thyroid hormone replacement therapy
with levothyroxine desiccated thyroid or lyophilene. Such treatment
inhibits secretion of TSH and allows the thyroid gland to rest [5,11].

Small doses of iodine in the form of Lugol’s solution or potassium
solution are given to patients whose goiter is caused by iodine
deficiency [5,11].

Patients must be cautioned to take medication exactly as prescribed.
Patients’ and families’ knowledge of how to administer medication,
dose, route, action, and side effects must be assessed. Patients must
inform their HCPs of any and all medications they are taking in
conjunction with drugs used to treat goiter including prescription,
OTC, herbs, vitamins, minerals, and any other supplements [5,14].

Other treatment initiatives include [5,11]:
- Diet: Patients who have sporadic goiters must be taught to avoid
goitrogenic foods and medications. Patients who are taking such
medications should be cautioned not to discontinue such drugs
without approval of the prescribing physician or nurse practitioner.
Patients must inform these providers about goiter development and
what medications have been prescribed to treat the goiter. Patients
with endemic goiters should be instructed to use iodized salt to
include necessary amounts of iodine in their diet.
- Radiation: Radioiodine ablation therapy to the thyroid gland
is administered to destroy cells that concentrate iodine for the
production of thyroid hormone.
- Surgery: Patients who have large goiters that do not respond to
other treatment measures may need surgery. Partial removal of the
thyroid gland (subtotal thyroidectomy) may relieve pressure on
adjacent structures.

Patients with large goiters may experience embarrassment because
of their appearance and fear permanent disfigurement. Nurses and
other HCPs should provide emotional support and the importance of
adhering to prescribed treatment initiatives.

Hypoparathyroidism

Jane has recently undergone a thyroidectomy as part of the treatment
for thyroid cancer. Following surgery Jane develops tremors triggered
by voluntary movement, paresthesia, headaches, and severe anxiety.
Diagnostic evaluation shows electrolyte imbalances indicative of
hypoparathyroidism. It seems that during surgery to remove the
thyroid gland, parathyroid tissue has also been removed.

Hypoparathyroidism

Hypoparathyroidism is an uncommon condition caused by a deficiency
of PTH. Since PTH is essential to the regulation and maintenance
of calcium and phosphorus, hypoparathyroidism is characterized by
hypocalcemia and neuromuscular hyper-excitability [6,23].

Etiology and pathophysiology

Hypoparathyroidism can be acute or chronic, and is categorized as
idiopathic or acquired [5,23].

The most common cause of hypoparathyroidism is due to accidental
removal or destruction of parathyroid tissue or circulation to such
tissue during thyroidectomy or radical neck dissection [5,6].

Rarely, acquired hypoparathyroidism is caused by massive thyroid
irradiation, ischemic infarction of the parathyroid glands during
surgery, or from tuberculosis, neoplasms, trauma, sarcoidosis, or
hemochromatosis [5].

Acquired hypoparathyroidism may be reversible if the cause is
hypomagnesemia (causing impairment of hormone synthesis),
suppression of normal gland functioning because of hypercalcemia, or from delayed maturation of parathyroid functioning [5].

Idiopathic hypoparathyroidism may be linked to autoimmune disease or the congenital absence of the parathyroid glands [5,23].

Research shows the following statistics as they relate to hypoparathyroidism [5]:
- The incidence is 4 out of 100,000 people.
- Incidence of idiopathic and reversible forms is highest in children.
- Incidence of the irreversible acquired form is highest in older patients who have undergone surgery for hyperthyroidism or other pathology of the head and neck.

Hypoparathyroidism can cause a myriad of effects that lead to severe hypocalcemia and hyperphosphatemia. Recall that PTH is not regulated by either the pituitary or hypothalamus. This hormone maintains blood calcium levels by increasing bone resorption and GI absorption of calcium. PTH maintains an inverse relationship between serum calcium and phosphate levels [5].

Insufficient PTH secretion leads to decreased resorption of calcium from the renal tubules, decreased absorption of calcium in the GI tract, and decreased resorption of calcium from bone. Serum calcium falls to below normal levels triggering signs and symptoms of hypocalcemia such as neuromuscular irritability, increased deep tendon reflexes, and tremors [6].

Since calcium and phosphate have an inverse relationship serum phosphate levels increase, and excretion of phosphate by the kidneys decreases [5,6].

A number of complications are associated with hypoparathyroidism. These include [5]:
- Arrhythmias.
- Cataracts.
- Delayed mental development in children.
- Loss of consciousness.
- Osteoporosis.
- Tetany.

Several of these preceding complications are irreversible. Irreversible complications include [23]:
- Mental retardation in children.
- Stunted growth.
- Cataracts.
- Deposits of calcium in the brain that causes problems with equilibrium and seizures.

### Signs and symptoms

Mild hypothyroidism may be asymptomatic. However, the disorder usually produces hypocalcemia and elevated phosphate levels that affect the central nervous system in particular and other body systems as well [5].

Characteristic signs of hypoparathyroidism as manifested by hypocalcemia are [5,6]:
- Tetany: Manifested by muscle hypertonia and tremors and spasmodic or uncoordinated movements triggered by attempts at voluntary movements.
- Chvostek’s Sign: Hyperirritability of the facial nerve manifested by a spasm of facial muscles, which occurs when muscles or branches of the facial nerve are tapped.
- Trousseau’s Sign: Carpopedal spasm (spasmodic contractions of the muscles of the hands and feet) triggered within three minutes after a blood pressure cuff is applied to the arm and inflated to 20 mmHg above patient’s systolic pressure.
- Laryngeal spasm.

Additional clinical manifestations of hypoparathyroidism include [5,6,23]:
- Abdominal pain.
- Anxiety.
- Arrhythmias.
- Brittle nails.
- Cataracts.
- Depression.
- Dry, coarse skin.
- Dry, dull hair.
- Fatigue.
- Headaches.
- Memory problems.
- Mood swings.
- Muscles aches and cramps.
- Painful menstruation.
- Paresthesia (tingling or burning sensations in fingers, toes, and lips).
- Patchy loss of hair.
- Renal colic is there is a history of calculi.
- Weakness of tooth enamel causing decay and tooth loss.
- Weakness.

**Hypoparathyroidism alert!** CNS signs and symptoms are exaggerated during pregnancy, infection, thyroid hormone withdrawal, before menstruation, hyperventilation, and right before menstruation [5].

### Diagnosis

Diagnosis is made on the basis of the patient’s history and physical, presenting signs and symptoms, and the results of specific diagnostic tests. These tests include [5,6]:
- Serum phosphorous level: Elevated.
- Serum calcium level: Hypocalcemia indicated by a serum calcium level of 7.5 mg/100 ml or less.
- Serum magnesium level: Decreased.
- Electrocardiogram (ECG): As a result of hypocalcemia ECG shows prolonged QT and ST intervals.

- Bone density: If hypoparathyroidism is chronic bone density may be increased.

**Diagnostic alert!** Monitor patient for signs of heart block and decreased cardiac output due to prolongation of QT and ST intervals. Also monitor patients for signs of digoxin toxicity such as arrhythmias, nausea, fatigue, and vision changes since the reversal of hypocalcemia may quickly lead to digoxin toxicity [5,6].

### Treatment and nursing considerations

Early detection and treatment are essential if complications such as cataracts and brain calcifications are to be prevented [5].

**Treatment alert!** Cimetidine (Tagamet) interferes with normal parathyroid function, especially if renal failure is also a problem.

Any interference with parathyroid function increases the risk of hypocalcemia [6].

Calcium absorption requires the presence of vitamin D. Therefore, treatment of hypoparathyroidism must include vitamin D along with the administration of supplemental calcium [5].

Intravenous calcium administration is needed in the presence of acute life-threatening tetany. The most effective calcium solution is ionized calcium chloride (10%). All intravenous calcium preparations are
given slowly since it is a highly irritating solution that stings and causes thrombosis. The patient experiences burning flushing feelings of the skin and tongue. However, the intravenous calcium solution also seems to rapidly relieve feelings of anxiety [6].

Additional treatment measures include [5,6,23]:
- Vitamin D to promote calcium absorption. If patients are unable to tolerate the pure forms of vitamin D alternatives such as dihydrocholesterol (if liver and kidney functions are adequate) or calcitriol (if liver and kidney functions are compromised).
- Thiazide diuretic therapy. Thiazide diuretics can increase blood calcium levels. If patients do not respond to calcium administration thiazide diuretics may be added to the treatment regimen. Be sure that loop diuretics are not prescribed since these can actually decrease calcium levels.
- Correction of preexisting hypomagnesemia.
- Provision of a high-calcium, low-phosphorus diet.
- Sedatives and anticonvulsants may be administered to control spasms and tremors until calcium levels return to normal.

Treatment alert! Chronic tetany requires life-long treatment with oral calcium and vitamin D supplements unless it is of a reversible form [5].

Patients with a history of tetany who are awaiting a diagnosis of hypoparathyroidism need to have a patent intravenous line. Intravenous calcium preparations, a tracheotomy tray, and endotracheal tube should be kept at the bedside of hospitalized patients so that swift intervention is possible in the event of laryngospasm [5]. Be alert for the onset of minor muscle twitching, which may signal the onset of tetany [5].

Parents should be taught how to plan a diet that is rich in calcium and low in phosphorus. High calcium foods include dairy products, green leafy vegetables, broccoli, kale, and fortified orange juice and breakfast cereals. Phosphorus-rich foods to avoid include carbonated soft drinks, meats, and eggs [23].

Additional patient education measures to be implemented include [5,6,23]:
- Always provide written as well as verbal instructions. Make sure that information is written in terms that the patients and families can understand and in a language with which they are comfortable.
- Teach patients and families to stay alert for development of even minor muscle twitching and laryngospasm. These can signal tetany onset and HCPs should be notified immediately.
- Teach patients and families signs and symptoms of hypercalcemia and hypocalcemia and what to do if they occur. Assess their knowledge by having them describe these signs and symptoms. Patient education alert! When providing patient/family education never simply hand out written instructions or assess knowledge by asking, “Do you understand how to take your medication?” or “Do you know what the symptoms of low calcium are?” These kinds of questions require only a “yes” or “no” answer. Many patients and families will simply answer “yes” rather than admit they don’t understanding something. Assess knowledge by having them describe or list things such as signs and symptoms or side effects of medication. Have them demonstrate how to take medication to assess accuracy and knowledge.
- Teach safe and accurate medication administration. Have patients and families demonstrate correct medication administration and actually describe how and when to take it as well as any possible side effects. Do not forget to teach patients and families what to do if side effects occur. Stress the importance of having them keep their primary HCP informed of all medications (including OTC drugs and supplements such as herbs and vitamins).
- Warn patients not to substitute OTC preparations for their prescribed medication without approval of their HCPs. Some patients may try to do this in an attempt to save money because of the cost of prescription drugs. If this is an issue, refer patients and families to appropriate financial resources. Since calcium and vitamin D may be prescribed, patients may assume that less expensive OTC preparations will “work” just as well as prescription medications. Teach patients that OTC preparations may not have the same ingredients and/or the same strength as those in prescription medications, and, therefore, will not have the desired therapeutic effect.
- Caution patients and families to have their serum calcium level checked according to HCP orders (usually at least 3 times a year).
- Advise patients to wear medical alert bracelets.
- Provide information about skin care. Patients may have dry, scaly skin. Advise them not to use drying or irritating soaps or shower gels, especially those that are heavily perfumed. Encourage the use of therapeutic creams or lotions to moisten skin.
- Encourage patients to take good care of their nails, which may become brittle and dry. Instruct patients to keep their nails clean and well-trimmed to keep them from splitting.

Hyperparathyroidism

Grace is a 52-year-old women’s college basketball coach at a prestigious university. She leads a busy life and travels frequently. She has been suffering from low back pain for many months, which she attributes to the strain of travel and physical activity related to her coaching responsibilities. Lately she has begun to notice some weakness in her legs accompanied by significant loss of appetite and nausea. She is losing sleep because of the onset of polyuria, which necessitates many trips to the bathroom at night. Because of her hectic travel schedule she has not made time to have these symptoms evaluated by a physician. Finally, at the conclusion of another successful basketball season, Grace consults her family physician about her ongoing symptoms. Her physician performs a thorough physical examination including evaluation of electrolyte levels, which show elevated blood calcium levels. Further diagnostic work-up shows a high concentration of serum PTH. Grace’s physician diagnoses hyperparathyroidism.

Incidence

Hyperparathyroidism is the unregulated, hypersecretion of PTH [6,24]. The disease can occur at any age but is most common among women older than 50 years of age [6]. Hyperparathyroidism is a common disorder, although its prevalence is slowly decreasing [24]. It affects one in 1,000 people and is two to three times more common in females than in males [5].

Etiology and pathophysiology

There are two types of hyperparathyroidism: primary and secondary.
- Primary hyperparathyroidism: In primary hyperparathyroidism, one or more of the parathyroid glands enlarge, increasing PTH secretion, and promoting the elevation of serum calcium levels. The most common cause of primary hyperparathyroidism (in about 80% of cases) is single parathyroid adenoma (benign tumor of epithelial tissue). Parathyroid hyperplasia (enlargement of the parathyroid glands) is responsible for about 20% of cases. Note that parathyroid malignancy accounts for less than 1% of all cases of hyperparathyroidism [5,6,24].
- Secondary hyperparathyroidism: Secondary hyperparathyroidism is the overproduction of PTH due to a chronic abnormal stimulus.
This is usually due to chronic renal failure. Other causes include vitamin D deficiency or osteomalacia (softening of bone) [5,6,24]. Chronic overproduction of PTH causes in increased levels of serum calcium [6]. The normal negative feedback mechanism does not function, and chronic excessive resorption of calcium from bone due to excessive parathyroid hormone can lead to osteopenia (loss of some bone density). Other symptoms of hyperparathyroidism are due to hypercalcemia specifically but are not specific to hyperparathyroidism [24].

In secondary hyperparathyroidism overproduction of PTH in patients with renal failure add to the pathophysiology of bone disease found in patients on dialysis [24]. The abnormality that causes hyperparathyroidism causes hypocalcemia rather than the hypercalcemia caused by primary hyperparathyroidism [5].

Signs and symptoms

Clinical manifestations of primary hyperparathyroidism are due to hypercalcemia and are evident is several body systems, including [5]:
- Cardiac system: Arrhythmias, hypertension, and cardiac standstill (cessation of cardiac output) [6].
- CNS: Hyperparathyroidism causes depression of neuromuscular function as evidenced by emotional instability, alterations in levels of consciousness, general fatigue, personality changes, depression, stupor, and, possibly coma [5,6].
- GI system: Pancreatitis, ongoing, severe epigastric pain that radiates to the back, peptic ulcers, abdominal pain, anorexia, nausea, and vomiting [5].
- Musculoskeletal system: Significant muscle weakness and atrophy, especially in the legs. Chronic low back pain and bones that easily fracture because of bone degeneration, bone pain, chondrocalcinosis, and occasional severe osteopenia [5].
- Renal system: Elevated calcium levels cause nephrocalcinosis, possible recurring nephrolithiasis that may lead to renal insufficiency. Various renal signs and symptoms such as polyuria, are among the most common effects of hyperthyroidism [5].
- Miscellaneous effects: Skin necrosis, cataracts, anemia, and subcutaneous calcification [5].

Secondary hyperparathyroidism decreased serum calcium levels cause symptoms of hypocalcemia with skeletal deformities accompanied by signs and symptoms of the underlying disease [5]. Secondary hyperparathyroidism may be prevented by ensuring a diet that contains adequate amounts of calcium or by taking calcium and vitamin D supplements [5].

Diagnosis

Diagnosis is based on history, clinical manifestations, identification of underlying disorders, and the results of diagnostic tests.

Primary hyperparathyroidism diagnosis

The following findings are indicative of primary hyperparathyroidism [5,6]:
- Radioimmunoassay shows high serum PTH accompanied by hypercalcemia. The hypercalcemia must be noted on at least two separate tests to validate consistency of results.
- Elevated chloride and alkaline phosphate levels and a decreased serum phosphorus level.
- Elevated uric acid and creatinine levels.
- Increased basal acid secretion.
- Skeletal changes are revealed on x-ray.

Early diagnosis of hyperparathyroidism can be difficult and complications may be evident before diagnosis is confirmed. CT scan can identify parathyroid tumors more quickly than traditional X-rays. Sestamibi scan can help to assess tumor location [6].

Secondary hypoparathyroidism diagnosis

Laboratory findings in the presence of secondary hyperparathyroidism show [5]:
- Normal or slightly decreased serum calcium levels.
- Significantly elevated phosphorus levels.
- Diagnostic work-up is performed to identify the underlying cause of the disease [5].

Treatment and nursing considerations

Treatment of primary hyperparathyroidism may include surgical removal of abnormal parathyroid tissue and initial management of hypercalcemia [5,6].

Treatment of hypercalcemia includes [6]:
- Administration of intravenous normal saline solution and diuretics such as Lasix and Edecrin to increase urinary excretion of calcium for those patients who are not in renal failure.
- Administration of agents to inhibit bone resorption of calcium. These include Aredia, Cibacalcin, or Didronel.
- Administration of oral phosphate as an anti-hypercalcemia agent.
- Restriction of dietary calcium and discontinuation of drugs that might facilitate hypercalcemia such as thiazides, and vitamin D.
- Dialysis for patients in renal failure or for those whose hypercalcemia does not respond to other treatments.
- Reduced dosage of digoxin since hypercalcemic patients are more vulnerable to the toxic effects of this drug.
- Monitoring of calcium (daily), blood urea nitrogen (BUN), potassium, and magnesium levels.

Surgical removal of parathyroid tissue may relieve bone pain within 3 days of the surgery. Unfortunately, renal damage may be irreversible [5].

Surgical alert! All but half of one remaining parathyroid gland is needed to maintain normal PTH levels [5].
Following surgery nursing interventions include [5,6]:
- Monitor intake and output.
- Provide adequate fluid and electrolyte replacement.
- Strain all urine for renal calculi.
- Limit dietary intake of calcium.
- Monitor for urinary tract infections, hematuria, and renal colic.
- Take safety precautions to prevent pathologic fractures, to which the patient is prone.
- Monitor for signs of tetany.
- Monitor fluid and electrolyte levels.

Pathology of hyperparathyroidism involves significant effects of hypercalcemia. Now, postoperatively, the patient must be monitored for hypocalcemia. Such signs and symptoms include [6]:
- Paresthesia.
- Positive Chvostek’s sign: Tapping the check over the facial nerve causes a twitch of the lip or facial muscles.
- Positive Trousseau’s sign: Carpopedal spasm induced by applying a blood pressure cuff and occluding circulation in the arm.
- Take safety precautions if the patient is prone to seizures.

Treatment of secondary hyperparathyroidism focuses on correction and treatment of the underlying cause. Remember that secondary hyperparathyroidism causes hypocalcemia as opposed to primary hypothyroidism, which causes hypercalcemia. Generalized treatment initiatives include [5]:
- Vitamin D therapy.
- Administration of oral calcium preparation in the presence of renal disease.
- Administration of a new classification of drugs (calcimimetics) approved for the treatment of secondary hyperparathyroidism. These drugs act by stopping the secretion of PTH.

Patients with chronic secondary hyperparathyroidism may find that the parathyroid glands do not revert to normal function even after calcium levels have been returned to normal [5].

### Adrenal insufficiency

Jackie is 55-year-old account executive with a major industrial company in a small urban area. She complains of not being able to get over the “flu” that she has had for nearly a month. She feels week, tired, has lost weight, and has periods of nausea, vomiting, and diarrhea. Her colleagues tease her that she has not been taking sick time from work but has actually been vacationing at the beach. They make these comments because Jackie appears to be deeply suntanned, especially in the creases of her hands, elbows, and knees. She also notices that several scars over her knees appear to be darker. Jackie discusses her symptoms with the company’s nurse. The nurse is disturbed, believing that these symptoms may indicate an endocrine disorder. After consulting several reference books the nurse wonders if Jackie may be suffering from Addison’s disease.

### Incidence

Adrenal hypofunction affects males and females in equal numbers and can occur at any age [5,11]. Hypofunction of the adrenal glands affects one in 16,000 neonates congenitally. In adults, 8 in 100,000 people are affected [5].

### Etiology

Primary adrenal hypofunction is defined as occurring when more than 90% of both adrenal glands are destroyed [5,25]. The majority of cases (up to 80% of Addison’s disease cases) are caused by an autoimmune process in which circulating antibodies specifically “attack” adrenal tissue [5,25]. Autoimmune Addison’s disease occurs primarily in middle-aged females and gradually destroys the adrenal cortex, the outer layer of the adrenal glands [25].

In primary adrenal hypofunction, the adrenal glands may be the only glands affected. However, other endocrine glands may be affected as well, something that is referred to as polyendocrine deficiency syndrome [25].

Polyendocrine deficiency syndrome appears in two forms: type 1 and type 2 [25]. Type 1 is inherited and occurs in children who, in addition to adrenal insufficiency, may also have [25]:
- Underactive parathyroid glands.
- Slowed sexual development.
- Pernicious anemia.
- Chronic fungal infections.
- Chronic hepatitis.

Type 2 polyendocrine deficiency syndrome, sometimes referred to as Schmidt’s syndrome, is also inherited but usually affects young adults and includes [25]:
- Underactive thyroid gland.
- Slowed sexual development.

Adrenal insufficiency, also known as Addison’s disease and adrenal hypofunction, is the result of hypofunction of the adrenal glands [5,25]. The disease occurs in two forms: primary and secondary [25].
- Primary adrenal insufficiency: The primary form (commonly referred to as Addison’s disease) originates within the adrenal glands and is characterized by a decrease in mineralocorticoid, glucocorticoids, and androgen secretion [11,25].
- Secondary adrenal insufficiency: The secondary form of the disease occurs secondary to a disorder (e.g., pituitary tumor) outside the adrenal glands. In this form, aldosterone secretion may not be affected [5,11]. Secondary adrenal insufficiency is more common than Addison’s disease [25].

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Type 2 polyendocrine deficiency syndrome, sometimes referred to as Schmidt’s syndrome, is also inherited but usually affects young adults and includes [25]:
- Underactive thyroid gland.
- Slowed sexual development.
Secondary adrenal hypofunction alert! Prescription corticosteroids should always be discontinued gradually over a period of time ranging from weeks to months to reduce the chances of adrenal insufficiency [25].

- Surgical removal of pituitary tumors.
- Infections of the pituitary.

Adrenal crisis

Adrenal insufficiency can sometimes lead to adrenal crisis, a critical deficiency of mineralocorticoids and glucocorticoids. Adrenal crisis is the most serious complication related to adrenal hypofunction. It can develop gradually or abruptly [5,11,25].

Adrenal crisis is most likely to develop in people who [5,11]:
- Fail to respond to hormone replacement therapy.
- Abruptly stop hormone or prescribed steroid therapy.
- Experience trauma, surgery, or other types of physiologic stress that exhaust the body’s provisions of glucocorticoids in someone who has adrenal hypofunction.
- Undergo bilateral adrenalectomy.
- Develop adrenal gland thrombosis following a severe infection (referred to as Waterhouse-Friderichsen syndrome).

During adrenal crisis, there is a swift decline in the steroid hormones cortisol and aldosterone. This decline impacts the liver, stomach, and kidneys [11]. Adrenal crisis produces [5,25]:
- Significant weakness and fatigue.
- Abrupt severe pain in the lower back, abdomen, or legs.
- Severe nausea and vomiting.
- Dehydration.

Pathophysiology of adrenal insufficiency

The adrenal glands, located just above the kidneys, produce cortisol and aldosterone. These hormones help regulate blood pressure, metabolism, and the way the body responds to stress. Adrenal hormones also help to produce androgens and estrogens [25].

The adrenal hormone cortisol, a glucocorticoid, affects almost every tissue and organ in the body. Cortisol helps maintain blood pressure, slow the immune system’s inflammatory response, and regulate metabolism.11,25

In the event of adrenal insufficiency, decreased levels of cortisol can have the following effects on specific organs [11,25]:

- Liver: Reduced hepatic glucose output leading to hypoglycemia which can progress to dangerous levels.
- Stomach: Reduced levels of digestive enzymes leading to nausea, vomiting, cramps, and diarrhea.

Decreased levels of aldosterone can have the following effects [11,25]:
- Kidneys: Sodium and water loss accompanied by potassium retention. Electrolyte imbalances can lead to hypoglycemia and adverse cardiac effects.
- Heart: Arrhythmias, decreased output, hypotension.

Untreated the effects of adrenal hypofunction can progress to adrenal crisis causing shock, coma, and death [11].

Signs and symptoms

The most common signs and symptoms of adrenal hypofunction include [5,25]:
- Abdominal pain.
- Anorexia.
- Craving salty foods.
- Depression.
- Decreased libido in women.
- Diarrhea.
- Diaphoresis.
- Fatigue (chronic or long-lasting).
- Headache.
- Hypoglycemia.
- Hypotension (especially orthostatic hypotension).
- Irritability.
- Menstrual abnormalities.
- Nausea.
- Vomiting.
- Weakness.
- Weight loss.

Addison’s disease usually causes a characteristic, conspicuous bronze coloration of the skin. Patients appear to be deeply suntanned especially in the creases of the hands, over the metacarpophalangeal joints, elbows, and knees. Scars may darken, areas of vitiligo appear, and increased pigmentation of the mucous membranes, particularly of the gingival mucosa [5].

Adrenal hypofunction alert! Abnormalities in skin and mucous membrane coloration are due to decreased secretion of cortisol, which makes the pituitary gland secrete excessive amounts of corticotropin and melanocyte-stimulating hormone [5].

As the disease progresses, additional cardiovascular effects may become evident such as decreased cardiac output, decrease in heart size, and a weak, irregular pulse [5,11]. Other clinical manifestations include [5,11,25]:
- Decreased ability to tolerate even the smallest amount of stress.
- Poor coordination.
- Hypoglycemia.
- Retardation of pubic and axillary hair growth.
- Amenorrhea.
Secondary adrenal hypofunction produces similar clinical manifestations to those of primary adrenal hypofunction but without hyperpigmentation because corticotropin and melanocyte stimulating hormone levels are low. Aldosterone secretions may continue to be fairly normal in the secondary type so electrolyte levels may also be normal and hypotension may not occur [5,11].

**Diagnosis**

After a thorough history and physical and evaluation of signs and symptoms various lab studies are used to confirm a diagnosis of adrenal hypofunction and to categorize the disease as primary or secondary [5,11]. Analysis of plasma and urine shows decreased levels of corticosteroid concentrations. A high level of corticotropin suggests primary adrenal hypofunction. A low level of corticotropin suggests secondary adrenal hypofunction [5,11].

A rapid corticotropin test (ACTH stimulation test) is used to evaluate plasma cortisol response to corticotropin. First, plasma cortisol samples are obtained. Then an intravenous infusion of cosyntropin is administered. Plasma samples are obtained at 30 and 60 minutes after cosyntropin infusion. If plasma cortisol levels do not increase, adrenal insufficiency is suspected [11].

In patients who have characteristic signs and symptoms of Addison’s disease the following laboratory tests indicate acute or crisis level adrenal hypofunction [5,11]:
- Increased potassium serum calcium, and blood urea nitrogen levels (BUN).
- Decreased serum sodium levels.
- Elevated hematocrit, lymphocyte, and eosinophils counts.
- X-rays show decreased heart size and adrenal calcification.
- Decreased plasma cortisol levels in plasma. Levels are less than 10 mcg/dL in the morning. Levels are lower at night.

**Treatment and nursing considerations**

All patients affected by primary or secondary adrenal hypofunction need life-long corticosteroid replacement therapy. Cortisone or hydrocortisone is administered because these agents have a mineralocorticoid effect [5,11]. To minimize or prevent dehydration and hypotension, a synthetic drug that acts as a mineralocorticoid (oral fludrocortisones) may be given. Testosterone injections may be given to women who experience a decrease in libido and muscle weakness. However, testosterone injection may cause masculinizing effects [11].

Special nursing considerations include [5,25]:
- Monitor for signs of adrenal crisis. Teach patients and families how to recognize adrenal crisis and to seek immediate emergency medical attention if it occurs.
- Explain that corticosteroid therapy must be taken for the rest of the patients’ lives. Teach patients how to take their medication and have them demonstrate knowledge of these medications by demonstrating how to take them and being able to state the name of the drug(s), route, dose, action, and side effects. As always, keep HCPs aware of any medications and supplements they are taking.
- Advise patients to wear medical alert bracelets that contain the name of the drugs they are taking and the doses of these drugs.
- Teach patients (and their families) who are diabetic that steroid replacement therapy may require insulin dosage adjustments and to monitor their blood glucose levels with particular care. Tell them to consult with the HCP who helps manage their diabetes that they are on life-long corticosteroid therapy so that he/she can adjust treatment plans accordingly.
- Advise patients to use caution in their daily activities because steroid therapy can make it easier for some patients to bruise.
- Advise patients who are anorectic to try eating 6 small meals a day instead of 3 large ones. Explain that a late-morning snack may help prevent hypoglycemia.
- Advise patients and families that the dose of corticosteroid therapy may need to be increased during times of stress such as physical illness or emotional trauma.
- Advise patients and families that infection, trauma, injury, or profuse diaphoresis may trigger an adrenal crisis.
- Teach patients that they should keep an emergency kit containing hydrocortisone in a prefilled syringe for use in times of stress or adrenal crisis. Have patients and families demonstrate their ability to give themselves hydrocortisone injections.

**Hyeraldosteronism**

Emma is a 40-year-old chemistry professor at a private university. She has a family history of diabetes mellitus and has recently been diagnosed with the disease. Her family physician, however, is concerned that there is something “more” than diabetes causing additional signs and symptoms such as muscle weakness, increased neuromuscular irritability, and irregular heart rate. Lab studies show low levels of potassium, which could account for the preceding “additional” symptoms. Emma does not take diuretics nor has she had any recent illness that would contribute to GI losses such as vomiting or diarrhea. Searching for a cause of the hypokalemia (abnormally low potassium), the physician does a more detailed diagnostic work-up. After extensive diagnostic testing, Emma is found to have a benign aldosterone-producing adrenal adenoma, which is triggering hypersecretion of aldosterone.

**Adrenal hypofunction diagnostic alert!** Note that testing cortisol levels takes considerable amounts of time. Therefore, adrenal crisis treatment should not be delayed while waiting for the results of this particular test [11].

After a diagnosis of Addison’s disease is made, the following tests may help HCPs determine if the disease is related to tuberculosis or to antibodies associated with autoimmune Addison’s disease [25]:
- Abdominal ultrasound: Performed to identify adrenal gland abnormalities such as an increase or decrease in size, nodules, or the presence of calcium deposits that may suggest bleeding.
- Tuberculin skin test: A positive test suggests adrenal insufficiency related to tuberculosis.
- Antibody blood test: The presence of antibodies associated with autoimmune Addison’s disease helps to confirm diagnosis.

After a diagnosis of secondary adrenal insufficiency is made, the following tests may be performed to assess pituitary gland functioning [25]:
- CT scan: A CT scan can show the size and shape of the pituitary gland and abnormalities such as nodules or tumors.
- MRI: An MRI provides three dimensional images of the hypothalamus and the pituitary gland to detect abnormalities in size and the presence of tumors, nodules, or other abnormalities.
- Hormonal blood tests: Hormonal blood tests are used to evaluate pituitary functioning.
Hyperaldosteronism, also referred to as Conn’s syndrome or aldosteronism, is the hypersecretion of the mineralocorticoid aldosterone by the adrenal cortex. Such extreme secretion causes excessive reabsorption of sodium and water, and excessive renal excretion of potassium [5].

Etiology and incidence

Benign aldosterone-producing adrenal adenoma is the cause of hyperaldosteronism in 70% of patients. The cause is unknown in 15% to 30% of patients. Rare cases of hyperaldosteronism are bilateral adrenocortical hyperplasia (affecting children) and cancer. Incidence is 3 times greater in females compared to males and occurs most often in persons between the ages of 30 and 50 [5].

Pathophysiology

Hyperaldosteronism may be classified as primary or secondary. Primary hyperaldosteronism refers to a chronic excess of aldosterone that is independent of the renin-angiotensin system. This disorder actually causes a suppression of plasma renin activity [5,26].

Primary hyperaldosteronism primarily affects adults. Incidence peaks in the fourth to sixth decades of life. About 60% of cases of primary hyperaldosteronism are due to an idiopathic hyperaldosteronism (IHA). An estimated 40% of cases are due to an aldosterone-producing adenoma (APA). Only about 1% of cases are classified as inherited and are most likely to occur during childhood [26].

In primary hyperaldosteronism, excess aldosterone facilitates sodium reabsorption by the kidneys, which leads to mild hypokalemia, hypokalemia, and increased extracellular fluid (ECF) volume. Intravascular fluid volume also increases and causes volume-dependent hypertension and increased cardiac output [5].

Hyperaldosteronism alert! Eating large amounts of English black licorice or licorice-like substances can cause a syndrome that mimics primary hyperaldosteronism. This is because glycyrrhizic acid, a substance found in licorice, has a mineralocorticoid action [5].

Diagnosis

Diagnosis depends on history and physical, signs and symptoms, and specific diagnostic tests.

Most of the presenting clinical manifestations of hyperaldosteronism are due to hypokalemia. Signs and symptoms related to decreased potassium include [5,26]:

- Fatigue.
- Headaches.
- Intermittent, flaccid paralysis.
- Muscle weakness.
- Paresthesia.

Metabolic alkalosis may also occur, leading to hypokalemia. If hypokalemia occurs, the patient may also exhibit tetany [5].

Diabetes mellitus is frequently found in conjunction with hyperaldosteronism because hypokalemia, which causes the majority of clinical effects of hyperaldosteronism, can interfere with proper secretion of insulin. Hypertension, vision problems, polyuria, and polydipsia may also occur [5].

Diabetic alert! Ongoing low levels of serum potassium in patients who do not have edema, are not taking diuretics, have not had GI tract losses due to vomiting or diarrhea, and who have a normal sodium intake strongly suggest hyperaldosteronism [5].

In hyperaldosteronism, serum bicarbonate levels are often elevated accompanied by alkalosis. Other tests show significantly increased urinary aldosterone levels, increased plasma aldosterone levels, and in secondary hyperaldosteronism, increased levels of plasma renin [5].

Typically the plasma aldosterone concentration (PAC) to plasma renin activity (PRA) ratio test is used as a screening tool for hyperaldosteronism. This test measures the plasma PAC to PRA ratio. A high ratio of PAC to PRA suggests primary hyperaldosteronism, but additional testing is usually performed to confirm diagnosis [27].

Tests used to confirm primary hyperaldosteronism include [27]:

- Captopril suppression test: Patients are given a single dose of the antihypertensive drug captopril after which plasma aldosterone and renin are measured. In patients with primary hyperaldosteronism blood levels of aldosterone remain high and renin levels are low.
- 24-hour urinary excretion of aldosterone test: Patients ingest a high-sodium diet for 5 days after which the amount of aldosterone in the urine is measured. In patients with primary hyperaldosteronism, aldosteronism will not be suppressed by the salt load, and the level of aldosterone in the urine will be high [27].
- Saline suppression test: Patients are given intravenous salt solutions after which blood levels of aldosterone and renin are measured. In patients with primary hyperaldosteronism the level of aldosterone in the blood is still high, and the level of renin is low even after this salt loading [27].

A suppression test is also helpful in differentiating between primary and secondary hyperaldosteronism. Patients receive oral deoxycorticosterone for 3 days while plasma aldosterone levels and urinary metabolites are continuously measured. In secondary hyperaldosteronism, levels decrease but levels remain the same in primary hyperaldosteronism [5].

Treatment and nursing considerations

Treatment measures for unilateral hyperaldosteronism (only one adrenal gland is affected) include surgical adrenalectomy of the affected gland, administration of a potassium sparing diuretic, and restriction of sodium [5]. In the presence of bilateral adrenal hyperplasia, administration of spironolactone (the drug of choice) is recommended for the management of primary hyperaldosteronism.
Cushing’s syndrome

Brenda is a 30-year-old financial counselor. She suffers from rheumatoid arthritis and has taken prednisone for a significant period of time in an attempt to control the increasingly severe effects of the disease. Lately, Brenda has begun to notice some troubling new symptoms. She complains about gaining weight, and that this excess weight is especially noticeable over the trunk of her body and on her face, which she says has gotten “round.” She feels weak, and minor cuts and scratches “take forever” to heal. Brenda also notices an increase in facial hair over her lip and chin. Brenda attributes these signs and symptoms to the effects of rheumatoid arthritis, which she says has “ruined” her life. Brenda is in no hurry to report these new problems, believing that nothing can be done to resolve them. “I’ll just wait until my next regular doctor’s appointment next month.” When Brenda next sees her physician these new signs and symptoms have arisen. Based on Brenda’s history and presenting clinical picture the physician initiates a diagnostic work-up to confirm her suspicion that Brenda has Cushing’s syndrome.

Cushing’s syndrome is a hormonal disorder caused by prolonged exposure of the body’s tissues to excessive levels of adrenocortical hormones, especially cortisol, related corticosteroids, and, to a lesser extent, androgens and aldosterone [5,28].

Cushing’s syndrome produces a characteristic clinical picture that includes fat deposits of the face, neck, and trunk and purple striae on the skin. Prognosis depends on the underlying cause of the syndrome. Prognosis is poor in persons who do not receive treatment and in people with untreatable ectopic corticotropin producing cancer [5].

Cushing’s syndrome alert! If excess of glucocorticoids is due to a pituitary dependent condition, it is called Cushing’s disease [11].

Review of the role of cortisol

The hypothalamus sends corticotropin-releasing hormone (CRH) to the pituitary gland. CRH triggers the pituitary to secrete adrenocorticotropin hormone (ACTH), which stimulates the adrenal glands to release adrenocortical hormones such as cortisol and, to a lesser extent, androgens and aldosterone [5,28].

Cortisol is essential to many critical body functions. Cortisol [28]:

- Helps maintain blood pressure and cardiovascular function.
- Reduces the inflammatory response of the immune system.
- Balances the effects of insulin.
- Regulates the metabolism of proteins, carbohydrates, and fats.
- Helps the body respond to stress.

Incidence and etiology

Cushing’s syndrome is 10 times more common in women than in men and is most often diagnosed in persons between the ages of 25 and 40. It affects 13 out of every one million people [5,6].

Cushing’s syndrome can be categorized as three types [11]:

- Primary: Primary Cushing’s syndrome is due to disease of the adrenal cortex.
- Secondary: Secondary Cushing’s syndrome is caused by hyperfunction of cells that secrete corticotropin in the anterior pituitary gland.
- Tertiary: Tertiary Cushing’s syndrome is due to dysfunction or injury of the hypothalamus.

The majority of cases of Cushing’s syndrome (70%) are caused by excess production of corticotropin. This leads to hyperplasia (excessive cell proliferation) of the adrenal cortex [5,11]. Causes of corticotropin overproduction include [5,11,28]:

- Monitoring for signs of tetany and hypokalemia such as cardiac arrhythmias, weakness, and paresthesia. Teach patients to recognize these signs and to report them to their HCPs promptly.
- Monitoring for signs of rising serum potassium levels and signs of adrenal hypofunction (especially hypertension) after adrenalectomy.
- Collaborating with the dietician, patients, and families, to develop a low sodium, high potassium diet.
- Teach patients who are taking the potassium-sparing diuretic spironolactone to be alert to the development of signs of hyperkalemia. Patients should be informed that long-term use of this drug may lead to impotence and gynecomastia.
- Advise patients who are taking steroid hormone replacement therapy to wear a medical identification bracelet.

Etiology alert! Rarely, Cushing’s syndrome may be due to an inherited tendency to develop tumors of one or more of the endocrine glands [28].
Complications

There are a number of complications associated with Cushing’s syndrome. These complications are related to pathological effects of the disorder [5,6,11].
- Lipidosis, a disorder of fat metabolism, may occur.
- Increased gastric secretion, pepsin production, and decreased amounts of gastric mucous can lead to the development of peptic ulcers.
- Increased hepatic gluconeogenesis and insulin resistance may lead to impaired glucose tolerance.
- Increased calcium resorption from bone can cause osteoporosis and pathological fractures.
- Decreased lymphocyte production, hyperglycemia, and inhibited antibody formation can lead to frequent infections and/or slow healing of wounds.
- Sodium and water retention contribute to the development of hypertension, which is quite common in persons with Cushing’s syndrome. Ischemic heart disease and heart failure may develop.
- Increased adrenal androgen production can cause menstrual problems and disturbances in sexual function.
- A decreased ability to cope with physical or psychological stress can lead to mental health disturbances that can range in severity from mood swings to psychosis.

Signs and symptoms

Cushing’s syndrome can have adverse effects on multiple body systems. Effects are directly related to the adrenocortical hormone involved [5].

Differentiation between Cushing’s syndrome and cushingoid syndrome! Differentiating between Cushing’s syndrome and cushingoid syndrome can be challenging. Chronic depression, alcoholism, and long-term treatment with corticosteroids can combine to produce cushingoid syndrome, an adverse consequence characterized by fat deposits between the shoulders and around the waist and many systemic abnormalities. Cushing’s syndrome has similar signs, but can be differentiated from cushingoid syndrome by the additional presence of hypertension, renal problems, hyperglycemia, muscle weakness, tissue wasting, and frequently changing emotional states (emotional lability) [5].

Signs and symptoms of Cushing’s syndrome can be grouped according the body system affected.
- Cardiovascular system: Sodium and water retention leads to hypertension, left ventricular hypertrophy, expanded blood volume, edema, weight gain, fatigue, capillary weakness stemming from protein loss, bleeding, petechiae, and ecchymosis [5,6].
- Endocrine and metabolic systems: Diabetes mellitus, decreased glucose tolerance, fasting hyperglycemia, and glycosuria [5].
- Gastrointestinal system: Increased gastric secretion, pepsin production, and decreased gastric mucous can cause peptic ulcer [5,11].
- Immune system: Excessive levels of adrenocortical hormones can cause decreased lymphocyte production and suppressed antibody formation. This increases the likelihood of infection, slows the wound healing process, and decreases the body’s ability to withstand stress [5,6].

Cushing’s syndrome alert! Immune system suppression can mask infection, even severe infections [5]. It is important for HCPs to recognize, and to teach patients and families to recognize, even the slightest signs of infection.
- Musculoskeletal system: Muscle weakness because of low potassium levels is evident. Loss of muscle mass may occur. Decreased bone mineral often leads to pathologic fractures and, in children, skeletal growth retardation [5,6,11].
- Renal/urologic system: Sodium and fluid retention occurs. There is increased excretion of potassium, inhibited antidiuretic hormone secretion, and formation of ureteral calculi [5].
- Reproductive system: Increased androgen leads to hypertrophy of the clitoris and amenorrhea or oligomenorrhea in women. Sexual dysfunction and loss of libido may also occur [5,6].

Diagnosis

Diagnosis depends on the patient’s clinical presentation and the results of various diagnostic tests. The first step in the diagnostic process is the review of signs and symptoms, especially notable being the characteristic moon face, buffalo hump, muscle weakness, and purple striae [5]. A clinical picture that suggests Cushing’s syndrome requires determination of plasma steroid levels. Plasma cortisol levels should be obtained in the morning since levels are higher in the morning and decrease gradually throughout the day. In the presence of Cushing’s syndrome cortisol levels do not fluctuate. They remain consistently elevated throughout the day. Analysis of a 24-hour urinary cortisol collection provides evidence of consistently elevated cortisol levels [5,11].

If morning plasma cortisol levels are elevated, and 24-hour urinary cortisol collection show consistent elevation, Cushing’s syndrome is considered to be likely and should be confirmed by additional testing [5,11]. Test results indicative of Cushing’s syndrome include [6,11]:
- Significantly elevated plasma cortisol levels.
- Increased blood glucose levels.
- Glucose intolerance.
- Reduced eosinophils.
- Elevated urinary 17-hydroxycorticoids and 17-ketogenic steroids.
- In the presence of an adrenal tumor, ACTH plasma levels are elevated. ACTH levels that are higher in the petrosal sinuses than in a vein in the forearm suggest the presence of a pituitary adenoma.
- Elevated salivary cortisol levels are considered significant.
- Elevated WBC count.
- CT scan, MRI, and/or ultrasound are used to detect the presence and location of a tumor in the pituitary or adrenal glands.

High dose dexamethasone suppression test is used to determine if Cushing’s syndrome is due to pituitary dysfunction. If dexamethasone suppresses plasma cortisol levels, the test is considered positive. Failure to suppress plasma cortisol levels indicates the presence of an adrenal tumor or non-endocrine, corticotropin-secreting tumor. HCPs should be aware that this test can produce false positive results [4,11].
Donna is nearly 14 years old and has not yet begun to menstruate. She is starting to develop a faint mustache, which has made her the target of ridicule by her peers. Concerned, Donna’s mother, Shirley, decides to take her to be evaluated by a gynecologist. Physical examination shows excessive growth of axillary hair, failure to menstruate, and an enlarged clitoris. The physician orders a battery of diagnostic tests including serum electrolyte, aldosterone, renin, and cortisol levels. Test results in conjunction with history and physical findings indicate adrenogenital syndrome.

Adrenogenital syndrome

Donna is nearly 14 years old and has not yet begun to menstruate. She is starting to develop a faint mustache, which has made her the target of ridicule by her peers. Concerned, Donna’s mother, Shirley, decides to take her to be evaluated by a gynecologist. Physical examination shows excessive growth of axillary hair, failure to menstruate, and an enlarged clitoris. The physician orders a battery of diagnostic tests including serum electrolyte, aldosterone, renin, and cortisol levels. Test results in conjunction with history and physical findings indicate adrenogenital syndrome.

Surgical intervention

Surgery is performed to remove adrenal or pituitary (hypophysectomy) tumors. Pituitary tumors may be removed via the transsphenectomy approach, during which the pituitary is removed through the nasal cavity, sphenoid sinus, and into the sella turcica [6]. Transsphenoidal hypophysectomy is extremely delicate surgery, and patients are usually referred to medical facilities that specialize in this type of surgery. The success rate when performed by a surgeon experienced in this procedure is more than 80%. If the surgery is not successful, or provides only a temporary cure, it can be repeated, often with good outcomes [28].

If a tumor has grown beyond the sella turcica a transfrontal craniotomy may need to be performed. If there is hyperplasia of both adrenal glands, bilateral adrenalectomy may be needed [6].

Before surgery, the patient must undergo treatment to control edema, diabetes, hypertension, and other cardiovascular effects caused by Cushing’s syndrome. Patients must be especially careful to avoid infection prior to and after surgery [5,11].

Immediately prior to surgery, the administration of glucocorticoids can help prevent acute adrenal hypofunction during the surgery itself. During and after surgery, cortisol therapy should be administered to help the patient deal with the physiologic stress caused by the removal of the pituitary or adrenal gland(s) [5, 11].

In the event that normal cortisol production resumes, steroid therapy may be gradually tapered and ultimately discontinued, usually within a period of 12 to 18 months [5,6,11]. However, if both adrenal glands have been removed (bilateral adrenalectomy), or if the entire pituitary has been removed (total hypophysectomy), life-long steroid replacement therapy is necessary [5,11].

Radiation therapy

If surgical approaches fail, or if a patient is not a candidate for surgery, radiation therapy is a possible alternative treatment. Radiation treatment to the pituitary gland is generally administered over a 6-week period. Improvement is noted in 40% to 50% of adults and up to 85% of children [28].

Another option is stereotactic radiosurgery or gamma knife radiation. This allows for the delivery of radiation in a single high-dose treatment [28].

Radiation alert! It may take months or even years for patients to feel better after receiving radiation treatment alone. Radiation in conjunction with cortisol-inhibiting drugs can help speed up the recovery process [28].

Medications

Patients with non-endocrine corticotropin-producing tumors require excision of the tumor followed by drug therapy. Drug therapy is also administered if the patient cannot undergo surgery. Medications prescribed include [5,6,11]:

- Mitotane: Mitotane (Lysodren) is toxic to the adrenal cortex. Its administration is referred to as medical adrenalectomy. Side effects of this drug include nausea, vomiting, diarrhea, somnolence, and depression.
- Meryrapone: Meryrapone (Metopirone) is given to control hypersecretion of steroids in those who fail to respond to mitotane.

A combination of aminoglutethimide, cyproheptadine, and ketoconazole may be prescribed in an effort to decrease levels of cortisol.

Aminoglutethimide may be given alone or along with metyrapone as part of the treatment for metastatic adrenal cancer.

Nursing considerations

Nursing considerations of particular interest include [5,6]:

- Monitor patients carefully for signs of infection. Patients with Cushing’s syndrome are especially prone to infection.
- Facilitate physical and emotional rest. Cushing’s syndrome can trigger periods of emotional lability. Adequate rest is essential to help relieve some of this instability.
- Monitor weight, intake and output, electrolyte levels, hormone levels, and glucose levels.
- Caution the patient to wear a medical identification bracelet.
- Instruct patients to inform their HCPs immediately if they develop infections, physical illness, and/or significant emotional stress, which may trigger the need for increased dosage of hormone therapy.
- Teach patients to take replacement steroid therapy with food or with antacids to reduce gastric irritation. It is often recommended that two-thirds of the dosage be taken in the morning and one-third in the early afternoon. This should mimic natural rates of adrenal secretion.

Adrenal gland cancer

Adrenal gland cancer is a rare disease that can affect people of all ages. It is most common in older adults and is more likely to occur in women than in men. Symptoms may include:

- Fatigue
- Weakness
- High blood pressure
- Abdominal pain
- Jaundice

Treatment options for adrenal gland cancer may include surgery, chemotherapy, and/or radiation therapy. The goal of treatment is to remove the tumor and stop the growth of cancer cells. If surgery is not possible, chemotherapy and/or radiation therapy may be used to shrink the tumor and relieve symptoms.

Surgery is the most common treatment for adrenal gland cancer. It may involve removing one or both adrenal glands. If the tumor is malignant, the surrounding tissue may also need to be removed. Surgery may be performed through a small incision in the side of the body or through a laparoscopic procedure.

Chemotherapy is used to treat adrenal gland cancer when surgery is not possible or when the cancer has spread to other parts of the body. Chemotherapy is a type of drug that is given to stop the growth of cancer cells.

Radiation therapy is used to treat adrenal gland cancer when surgery is not possible or when the cancer has spread to other parts of the body. Radiation therapy uses high-energy rays to kill cancer cells.

Nursing considerations

Nursing considerations of particular interest include [5,6]:

- Monitor patients carefully for signs of infection. Patients with Cushing’s syndrome are especially prone to infection.
- Facilitate physical and emotional rest. Cushing’s syndrome can trigger periods of emotional lability. Adequate rest is essential to help relieve some of this instability.
- Monitor weight, intake and output, electrolyte levels, hormone levels, and glucose levels.

After bilateral adrenalectomy and/or pituitary surgery it is important that nurses [5,6]:

- Caution the patient to wear a medical identification bracelet.
- Instruct patients to inform their HCPs immediately if they develop infections, physical illness, and/or significant emotional stress, which may trigger the need for increased dosage of hormone therapy.
- Teach patients to take replacement steroid therapy with food or with antacids to reduce gastric irritation. It is often recommended that two-thirds of the dosage be taken in the morning and one-third in the early afternoon. This should mimic natural rates of adrenal secretion.
Adrenogenital syndrome, perhaps more commonly known as congenital adrenal hyperplasia, is a syndrome caused by disorders of adrenocortical steroid biosynthesis. Most cases of the syndrome are due to the failure of the adrenal glands to produce enough cortisol [5,29].

**Incidence**

- If adrenogenital syndrome is inherited, it is referred to as congenital adrenal hyperplasia (CAH). The syndrome may also be caused by an adrenal tumor (adrenal virilism) [5,29].

*Adrenogenital syndrome alert! A salt-losing form of CAH in neonates may cause a fatal adrenal crisis [5].*

Some experts describe CAH as having two major types [29]:
- Classic CAH is the more severe form of the disease. It is usually diagnosed in infancy or early childhood.
- Non-classic CAH is the less severe form, which is usually recognized in late childhood or early adulthood.

CAH is “the most prevalent adrenal disorder in infants and children” with simple virilizing CAH and salt-losing CAH being the most common forms [5]. Acquired adrenal virilism is a rare form of the disorder, affecting twice as many females as males [5]. About one in 10,000 to 18,000 infants are born with CAH [5,30].

**Pathophysiology**

CAH is an inherited autosomal recessive trait. It is usually due to insufficient production of cortisol. Production of mineralocorticoids such as aldosterone and androgens such as testosterone may also be affected [29].

Various compensatory mechanisms are enacted to combat inadequate production of cortisol. For example:
- Simple virilizing CAH: There is a deficiency of 21-hydroxylase, which leads to cortisol deficiency. This deficiency triggers an increase in corticotropin secretion as a compensatory mechanism. The corticotropin increase causes the production of large amounts of cortisol precursors and androgens that do not need 21-hydroxylase for synthesis. Excess androgens cause male characteristics to appear early in males or inappropriately in females [5,30].
- Salt-losing CAH: In this form of CAH, 21-hydroxylase is almost completely absent. This leads to an increase in corticotropin secretion, which leads to excessive production of cortisol precursors including those that are salt-wasting. At the same time cortisol and aldosterone levels that are dependent on 21-hydroxylase fall sharply. This abrupt decrease combined with excess amounts of salt-wasting compounds can trigger an acute adrenal crisis. Adrenal androgen production increases, and masculinization occurs [5].

**Complications**

A number of complications are associated with CAH including [5]:
- Adrenal tumor.
- Altered growth patterns and abnormalities in external genitalia and sexual maturity.
- Cardiovascular collapse and cardiac arrest in neonates.
- Hyperkalemia.
- Hypertension.
- Infertility.

**Acquired adrenal virilism**

Although the focus of this education program is CAH, it is important to also describe acquired adrenal virilism and its effects. Acquired adrenal virilism occurs in the presence of adrenal tumors, malignancies, or adenomas. This disorder is rare, can occur at any age, and is twice as common in females as in males [5].

Symptoms vary with age and include [5]:
- Prepubescent females: Pubic hair, enlarged clitoris, delayed development of breasts, and delayed or absent menses.
- Females, especially those who are middle-aged: Appearance of dark hair on face, legs, arms, chest and back, oily skin, menstrual irregularities, development of masculine muscle mass, breast and uterine atrophy, and public hair that extends toward the naval.
- Prepubescent males: Significant enlargement of genitalia with penis and prostate development equal to that of an adult male, failure of testicular maturation, and hirsutism.
- Males: There are no obvious signs in males. The identification of a tumor is usually accidental.

The following diagnostic results indicate acquired adrenal virilism [5]:
- Elevated urinary total 17-ketosteroids.
- Significantly elevated dehydroepiandrosterone levels.
- Normal serum electrolyte levels.
- Kidney x-rays may show kidney displacement caused by a tumor.

Treatment involves surgical excision of the tumor and/or radiation and chemotherapy as needed. Prognosis is very good for patients who have slow-growing and non-recurring tumors [5].

**Signs and symptoms of CAH**

Signs and symptoms of CAH depend on the severity of the disease and the age of the patient [30].

**Female with simple virilizing CAH**

Females have ambiguous genitalia such as an enlarged clitoris with a urethral opening at the base. There may be some labioscrotal fusion but the genital tract and gonads are normal [5].

As she ages and reaches puberty she develops [5,29,30]:
- Facial hair.
- Deep voice.
- Acne.
- Early appearance of pubic and axillary hair.
- Failure to menstruate.
Male with simple virilizing CAH:
The neonate male does not have obvious abnormalities. But at puberty he has accentuated masculine characteristics including a deep voice and an enlarged penis with frequent erections [5].

CAH alert! Both males and females may be taller than other children their age because they experience rapid bone and muscle growth. However, excessive androgen levels cause early epiphyseal closure, which leads to short adult height [5].

Females and males with salt-losing CAH
The salt-losing form of CAH is more severe than the simple form and causes more complete virilization in females. Male external genitalia (but without testes) develop [5,29,30].

Males with salt-losing CAH have no abnormalities in external genitalia. Thus, diagnosis immediately after birth is difficult and usually delayed until severe signs and symptoms develop.

In severe cases, signs of salt-losing CAH infants may develop as soon as 2 to 3 weeks after birth in both males and females. These signs include vomiting, diarrhea, dehydration, low potassium and sodium levels, and abnormal heart rhythms [29,30]. Infants are apathetic and fail to eat. These signs indicate the onset of adrenal crisis, which, unless treated promptly, may lead to cardiovascular collapse and cardiac arrest [5].

Diagnosis
Physical examination shows ambiguous genitalia in females or, in severe forms of the disease, females may have overt male external genitalia. Precocious puberty (onset of puberty before the age of 9) in both females and males is also indicative of CAH [5].

Laboratory findings that help confirm the diagnosis of CAH include [5,30]:
- Elevated urinary levels of hormone metabolites.
- Elevated plasma 17-hydroxyprogesterone level.
- Normal or decreased urinary levels of 17-hydroxycorticosteroids.

Diagnosis alert! Adrenal crisis or evidence of adrenal hypofunction in the first week of life suggests salt-losing CAH [5].

Treatment
Treatment focuses on return of hormone levels to normal or to near normal [5,30]. This involves administering a large, intramuscular dose of cortisone or hydrocortisone. Dosage is adjusted according to urinary 17-ketosteroid levels. Infants receive hormonal therapy intramuscularly until they reach the age of 18 months after which they can be given the hormones orally [5].

Patients with ambiguous external genitalia undergo sex chromatin and karyotype studies to determine their genetic sex. Females who have male external genitalia undergo reconstructive surgery between the ages of 1 and 3 years following evaluation of the impact of cortisone therapy [5].

Instruct patients’ parents that the child should wear a medical identification bracelet explaining that they are on long-term steroid therapy. Counseling and emotional support should be provided as parents deal with the psychological impact of CAH [5,30].

Hermaphroditism
Hermaphroditism is a condition appropriately mentioned as part of a discussion of CAH. Hermaphroditism is a rare condition in which children have both ovarian and testicular tissues. External genitalia are usually ambiguous, but may also be completely male or female, which effectively “hides” hermaphroditism until puberty. The child with hermaphroditism almost always has a uterus and ambiguous gonads. Fertility, however, is rare [5].

Lab studies similar to those of CAH are performed to rule out congenital adrenal hyperplasia [5].

Sexual assignment is based on the anatomy of the external genitalia. Reconstructive surgery, during which inappropriate reproductive organs are removed, is performed as early as possible to prevent the development of incongruous secondary sex characteristics at puberty. Hormonal replacement may be needed [5].

Parents need emotional support and counseling as they deal with their choice of sexual assignment of their children [5].

Pheochromocytoma
Pheochromocytoma is a rare, catecholamine-secreting tumor associated with hyperfunction of the adrenal medulla leading to an excessive secretion of epinephrine and norepinephrine. It may trigger life-threatening hypertension as well as an increase in metabolism and hyperglycemia [5,6,31].

Although this disease is potentially fatal, prognosis is good with appropriate treatment. Kidney damage associated with the disease, however, is irreversible [5].

Incidence and etiology
Pheochromocytoma affects all races and both men and women. It can occur at any age, but is most common between the ages of 30 and 60 and rare in people over the age of 65 [5,6].

The majority of pheochromocytoma tumors are benign, but 10% are malignant with associated metastasis [6]. Ninety-five percent of pheochromocytomas are located in the abdomen, and may occur as the result of an inherited autosomal dominant trait [5].

Pheochromocytoma alert! Research indicates that about 0.5% of patients newly diagnosed with hypertension have pheochromocytoma [5].
Complications

Complications associated with pheochromocytoma include [5, 6]:
- Heart failure.
- Irreversible damage to the kidneys.
- Retinopathy.
- Stroke.

Signs and symptoms

Pheochromocytoma causes episodes typically characterized by [31]:
- Headaches.
- Palpitations.
- Diaphoresis.
- Severe, possibly life-threatening, hypertension.

Occurrence of these episodes can vary from once every 2/months to as often as 25 times a day, and they may last from seconds to hours. As time goes by, these episodes usually occur more often and become more severe as the tumor grows in size [5,31]. Episodes can occur spontaneously or follow specific triggering events such as exercise, smoking, urination, or a change in environmental or body temperature [5].

Additional clinical manifestations that may also be part of pheochromocytoma episodes include [5,31]:
- Abdominal pain.
- Anxiety.
- Constipation.
- Fever.
- Flank pain.
- Pallor.
- Paresthesia.
- Sense of impending doom.
- Tachycardia.
- Tremors.
- Warmth or flushing.
- Weight gain or weight loss.

Pheochromocytoma is often diagnosed during pregnancy when the expanding uterus puts pressure on the tumor, thus, triggering more frequent attacks. These attacks can lead to stroke, cardiac arrhythmias, acute pulmonary edema, or hypoxia, any of which can be fatal to mother and/or fetus. The risk of spontaneous abortion is significant, but most infant deaths take place during labor or immediately after birth [5].

Diagnosis

The most common indicator for pheochromocytoma is continuous hypertension and a history of episodes characteristic of the disease [5,31]. The tumor itself is rarely palpable, and findings from diagnostic laboratory tests are necessary to confirm diagnosis [5,31].

The following tests are used to diagnosis pheochromocytoma [5,6,31]:
- Urine plasma catecholamine levels: A baseline specimen is obtained and another obtained during an episode of hypertension. Levels are elevated during a hypertensive episode.
- Total plasma catecholamines: Levels are 10 to 50 times above normal.
- Clonidine suppression test: In normal patients, results show decreased plasma catecholamine levels. However, in those persons with pheochromocytoma, levels remain unchanged.

Additional postoperative nursing considerations include [5,31]:
- CT scans or MRIs: Imaging tests are used to identify tumor location.

Analysis of a 24-hour urine specimen is used to confirm a diagnosis of pheochromocytoma [5]. “Increased urinary excretion of total free catecholamines and their metabolites, VMA and metanephrine, as measured by analysis of 24-hour urine specimen, confirms pheochromocytoma [5].”

Diagnostic alert! To makes sure that urine catecholamine measurements are reliable, have patients avoid foods high in vanillin for 2 days before urine collection of VMA. Examples of such foods include coffee, nuts, chocolate, and bananas [5].

Treatment

The treatment of choice for pheochromocytoma is surgical resection of the tumor, which usually cures the hypertension [31]. It is important that specific preoperative measures be taken beginning 1 to 2 weeks prior to surgery to control blood pressure and prevent intraoperative hypertensive crisis [5,31].

Measures to achieve preoperative medial stabilization include [31]:
- Administration of an alpha-adrenergic blocker or metyrosine.
- Volume expansion with isotonic sodium chloride solution.
- Facilitation of liberal salt intake.
- Administration of a beta blocker only after sufficient alpha blockade to “avoid precipitating a hypertensive crisis from unopposed alpha stimulation [31].”
- Administration of the last doses of oral alpha and beta blockers on the morning of surgery.

Postoperatively, the following measures are taken [5]:
- Administration of intravenous fluids.
- Administration of plasma volume expanders.
- Administration of vasopressors.

Persistent hypertension is possible in the period immediately following surgery as are acute episodes of headaches, palpitations, anxiety, and diaphoresis. Management of hypertensive crisis or acute episodes includes administration of push or drip intravenous phenolamine or nitroprusside to control and normalize blood pressure [5].

For persons who are not able to withstand surgery, alpha-adrenergic blockers and beta-adrenergic blockers are administered to help control the effects of catecholamine and to prevent acute episodes [5].

Treatment alert! Rarely, pheochromocytoma is malignant. If this is the case, surgery to excise the tumor is followed by radiation and/or chemotherapy as needed [5,31].

Additional postoperative nursing considerations include [5,31]:
- Monitor blood pressure meticulously. If hypertensive crisis occurs, blood pressure and heart rate should be monitored every 2 to 5 minutes until the patient stabilizes.
- Monitor glucose levels.
- Provide a quiet, calm environment since anxiety, noise, and excitement can trigger a hypertensive episode.
- Keep the room cool since postoperative adrenal gland secretions cause profuse diaphoresis. Change clothing and bed linens frequently.

Treatment alert! Blood pressure may also drop drastically in the postoperative period, especially during the first 24 to 48 hours after surgery. Monitor for signs of severe hypotension [5].

If the occurrence of pheochromocytoma is due to a suspected autosomal dominant transmission, family members should also be tested for this problem [5].
Multiple endocrine neoplasia

Multiple endocrine neoplasia (MEN) is an inherited disorder in which 2 or more of the endocrine glands develop hyperplasia, an adenoma, or a malignancy. These pathologies can occur at the same time or consecutively [5].

There are 2 types of MEN that are well recorded [5]:
● MEN I, also called Werner’s syndrome, occurs because of a defect in a gene that carries the code for the protein menin [32]. This defect leads to hyperplasia and tumors of the pituitary and parathyroid glands, islet cells of the pancreas, and, rarely, the thyroid and adrenal glands [5].
● MEN II, also called Sipple’s syndrome, is a rare familial malignancy caused by genetic mutation [33]. It usually involves medullary cancer of the thyroid and hyperplasia and tumor growth of the adrenal medulla and parathyroid glands [5,33].

MEN I is the more common form [5].

Incidence and etiology

MEN is usually due to autosomal dominant inheritance. It affects twice as many females as males and can occur at any time from adolescence through old age. However, it is rare in children [5].

Signs and symptoms

Clinical manifestations of MEN depend on the glands involved. The most common signs and symptoms of MEN I are those of hyperparathyroidism, including hypercalcemia, followed by ulcer development because of increased production of gastrin from non-beta islet cell tumors of the pancreas (Zollinger-Ellison syndrome). Hypoglycemia may occur as a result of pancreatic beta cell tumors that lead to increased production of insulin [5].

Here is a list of possible signs and symptoms related to MEN I [5,32]:
● Abdominal pain.
● Amenorrhea.
● Anxiety.
● Black, tarry stools.
● Confusion.
● Decreased appetite.
● Decreased libido.
● Epigastric pain relieved by eating or taking antacids.
● Fatigue.

MEN II signs and symptoms are related to the gland(s) affected by the malignancy. For example, an affected thyroid gland causes an enlarged thyroid mass, elevated calcitonin, and sometimes, evidence of Cushing’s syndrome. Adrenal medulla tumors cause headache tachycardia-related arrhythmias, and elevated blood pressure. If the parathyroid glands are affected, signs and symptoms are caused by the development of renal calculi [5].

Diagnosis

Clinical manifestations will indicate the type of diagnostic tests needed. Signs and symptoms that suggest particular gland involvement indicate the type of testing to be done. For example, upper gastric pain and ulcers due to Zollinger-Ellison syndrome indicate the need for pancreatic evaluation. In fact, 50% of patients with Zollinger-Ellison syndrome are ultimately diagnosed with MEN [5].

CT scans, MRIs, and x-rays may be used to identify tumor location [5,32]. Examples of additional tests, based on the presenting clinical picture include [32]:
● Fasting blood sugar.
● Cortisol levels.
● Serum electrolyte levels.
● Serum levels of various hormones depending on specific signs and symptoms.
● Tumor biopsies.

Since MEN is predominantly a hereditary disorder family members may undergo genetic testing [5,32].

Treatment

Treatment focuses on tumor removal and therapy to control any residual symptoms [5]. Treatment of malignant tumors may include, in addition to surgical removal, radiation therapy and chemotherapy depending on the size of the tumor, the surgeon’s ability to remove all of the tumor, and if there is evidence of metastasis [5,32,33].

Side effects of particular tumors such as hypertension with adrenal medullary tumor or treatment of peptic ulceration with MEN I must be dealt with in conjunction with tumor removal. If significant amount of specific glandular tissue is removed, hormonal replacement therapy is necessary [5].

Diabetes mellitus

Diabetes mellitus (DM) is a chronic disease of glucose intolerance. It is caused by a complete or relative deficiency of insulin or by a resistance to insulin characterized by disturbances in protein, fat, and carbohydrate metabolism [5,6].

In the United States, DM is [5,11]:
● The fifth leading cause of death.
● A contributing factor in approximately 50% of heart attacks.

● A contributing factor in about 75% of strokes.
● A contributing factor in renal failure.
● A contributing factor in peripheral vascular disease.
● The leading cause of new blindness.
Types of diabetes, incidence, and etiology

DM affects approximately 6.3% of the U.S. population or 18.2 million people. About 5.2 million people are not even aware that they have the disease, and incidence increases with age [11].

There are three types of DM.

- **Type 1:** Type 1 diabetes occurs when the beta cells in the pancreas are destroyed or suppressed. Formerly referred to as juvenile diabetes or insulin-dependent diabetes, type 1 diabetes is subdivided into idiopathic and immune-mediated types. In idiopathic diabetes there is permanent deficiency of insulin and no evidence of autoimmunity. In immune-mediated diabetes the body produces an autoimmune attack on pancreatic beta cells, and the pancreas becomes inflamed. By the time signs and symptoms appear, 80% of the beta cells are destroyed. Some experts, however, believe that beta cells are not destroyed but disabled and may later be reactivated [5,6,11].
- **Type 2:** Type 2 diabetes, formerly referred to as adult-onset diabetes or non-insulin dependent diabetes, may be attributed to insulin resistance in target tissues, abnormal insulin secretion, or overproduction of glucose (inappropriate hepatic gluconeogenesis) [6,11].

Risk factors for type 2 diabetes

Risk factors for type 2 diabetes include [5,6]:

- Black, Hispanic, Pacific Islander, Asian-American, or Native Americans.
- Family history of diabetes.
- High density lipoprotein cholesterol of less than 35 mg/dl or triglyceride of greater than 250 mg/dl.

Complications

Patients with DM have a risk of numerous complications that can affect every system of the body. Possible complications include [5,6,11]:

- Cardiovascular disease.
- Gastrointestinal (delayed gastric emptying and feelings of fullness after eating).
- Impaired ability to fight infection.
- Nephropathy.
- Nocturnal diarrhea.
- Orthostatic hypotension.
- Peripheral and autonomic neuropathy.

Complications of hyperglycemic crisis

Acute complications of hyperglycemic crisis may occur with diabetes. Failure to treat these complications appropriately can lead to coma or even death. These two complications are diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic nonketotic syndrome (HHNS) [11].

DKA is seen most often in patients who have type 1 diabetes. It may actually be the first sign of the disease. HHNS is seen most often in patients who have type 2 diabetes, but it can occur in any patient whose insulin tolerance is stressed or who has undergone procedures such as peritoneal dialysis, Hemodialysis, tube feedings, or total parenteral nutrition [11].

These complications occur when inadequate levels of insulin cause interferes with the body cells’ ability to take in glucose and convert it to energy. Thus, glucose accumulates in the blood, and the cells lack the energy needed to function. This triggers the liver to convert glycogen to glucose and still more glucose is released into the blood. But no matter how much glucose is manufactured and released into the bloodstream, the cells are not able to utilize it because of insulin deficiency [5,11,6].

Blood glucose levels become grossly elevated, serum osmolarity increases, and high amounts of glucose are present in the urine (glycosuria). This triggers osmotic diuresis and massive fluid loss, which, in turn, causes electrolyte loss. Water loss is greater than glucose and electrolyte loss and dehydration continues along with a decreased glomerular filtration rate and an eventual reduction of the amount of glucose excreted in the urine. As glucose excretion decreases, blood glucose levels continue to increase. This cycle continues, and if not stopped, leads to shock, coma, and death [11].

**DM complication alert!** Research now shows that glucose readings do not need to be as elevated as once believed for complications to occur. This means that exact glucose control is more important than ever [11].

**Type 2 diabetes alert!** Type 2 diabetes may also develop as a consequence of obesity. In fact, most patients with type 2 diabetes are obese [5].

- Secondary Diabetes: Secondary diabetes is so-called because this type occurs “secondarily” to another condition or event. The factors that trigger secondary diabetes include [11]:
  - Physical or emotional stress that can cause prolonged elevation of cortisol, epinephrine, glucagon, and GH. Such elevations increase blood glucose levels and demands on the pancreas.
  - Pregnancy, which causes weight gain, high levels of estrogen, and high levels of placental hormones [11]. This type of diabetes is referred to as gestational diabetes mellitus (GDM). Glucose levels usually return to normal after the women gives birth. However, women who have had GDM have a 40% to 60% chance of developing type 2 diabetes within 5 to 10 years [5].
  - Use of specific medications such as adrenal corticosteroids, hormonal contraceptives, and other drugs that oppose the desired effects of insulin [11].

Hypertension.
- Obesity.
- Older than 45 years of age.
- Sedentary lifestyle.
- Significantly impaired glucose tolerance.

- Peripheral vascular disease.
- Retinopathy.
- Skin disease (diabetic dermopathy).
- Urinary tract infections (UTIs).
- Vaginitis.

**DM alert!** DKA also leads to the conversion of fats into glycerol and fatty acids, which cannot be quickly metabolized and accumulate in the liver. There they are converted into ketones. Ketones accumulate in the blood and urine, causing acidosis [11].

DKA and HHNS are medical emergencies and require immediate treatment to correct fluid loss, electrolyte imbalances, and acid-base imbalances. Insulin is administered to correct hyperglycemia [5,11,6].
Signs and symptoms

DM may develop gradually or abruptly [5,11]. The most common symptom is generalized fatigue. Hyperglycemia “pulls” fluid from the tissues of the body, which causes characteristic symptoms (in both type 1 or type 2 diabetes) of polyuria (excessive urination), excessive thirst (polydipsia), and excessive eating (polyphagia) [5,6,11].

Other signs and symptoms include [5,6,11]:
- Dehydration.
- Dry, itchy skin.
- Frequent infections of the skin.
- Poor skin turgor.
- Unexplained weight loss.
- Vision changes.
- Weakness.

Type 1 diabetes usually causes a rapid development of symptoms including effects of muscle wasting and loss of subcutaneous fat [11]. Persons affected by type 2 diabetes generally have a symptom onset that is vague and gradual [5,11].

Diagnosis

According to American Diabetes Association (ADA) guidelines, DM can be diagnosed if patients manifest any of the following [5]:
- Symptoms of DM plus a random, nonfasting blood glucose level equal to or greater than 200 mg/dl.
- Fasting blood glucose equal to or greater than 126 mg/dl.
- Oral glucose tolerance test (2-hour sample) results equal to or greater than 200 mg/dl.

Diagnostic alert! Questionable results require that diagnosis be confirmed by repeat testing on a different day [5].

The ADA recommends the following testing guidelines [5]:
- Test people aged 45 and older who have no symptoms every 3 years.
- People with characteristic signs and symptoms should be tested immediately.
- High risk groups should be tested frequently.

An ophthalmologic exam may reveal diabetic retinopathy. Acetone is present in urine, and blood tests for glycosylated hemoglobin show recent glucose cortisol [5].

Blood glucose levels are classified by the ADA as [5]:
- Normal: <100 mg/dl.
- Prediabetes: 100 to 125 mg/dl.
- Diabetes: >126 mg/dl.

Treatment and nursing considerations

Treatment goals are to optimize blood glucose levels and decrease complications [11].

Medications

Many types of drugs are used to treat DM. Treatment of type 1 DM includes insulin replacement. Current forms of insulin replacement therapy include single-dose, mixed-dose, split-mixed dose, and multiple-dose regimens, which may be administered via an insulin pump. Insulin may be rapid, intermediate, or long-acting or a combination of rapid- and intermediate-acting [5,11].

Persons with type 2 DM may require oral antidiabetic medications that stimulate insulin production, increase cellular sensitivity to insulin, and suppress hepatic gluconeogenesis [5].

A variety of drugs have proven helpful in treating DM such as [5,14]:
- Sulfonylureas, which stimulate pancreatic insulin release.
- Meglitinides, which cause immediate, brief release of insulin and are given before meals.
- Biguanides, which decrease hepatic glucose production.
- Alpha-glucosidase inhibitors, which slow glucose breakdown and decrease postprandial glucose peaks.
- Thiazolidinediones, which enhance the action of insulin.
- Synthetic analogue of human amylin, which helps control glucose and is used with insulin.

Diet

Patients require in-depth dietary instruction. Each patient’s diet is planned specifically for him/her and should take into consideration dietary preferences to facilitate compliance. Patients with type 2 diabetes often need to lose weight. If this is the case, weight loss strategies should be incorporated into the diet plan [5,6].

Exercise

Exercise is encouraged as part of a healthy lifestyle and is especially helpful in the management of type 2 diabetes. Exercise facilitates weight loss, improves glucose tolerance, and increases insulin sensitivity [11].

Additional nursing considerations

In addition to diet, exercise, and medication administration, nurses must teach patients how to monitor their blood glucose levels and to care for equipment used for such monitoring. Blood pressure control and smoking cessation reduces complication onset and progression [5,6,11].

Patients must be taught to take meticulous care of their skin and seeking prompt treatment for infection. Special attention should be paid to care of the feet. Teach patients to wash feet daily and dry them carefully, especially between the toes. Inspect the feet for corns, calluses, redness, bruising, and any breaks in the skin. Abnormalities should be reported to the physician promptly. Advise patients never to walk barefoot and to wear non-constricting shoes [5].

Stress the need to report and numbness or pain in the hands and feet and any changes in voiding patterns (e.g., incontinence), which may indicate diabetic neuropathy. All blisters, cuts, and scrapes should be carefully treated. Teach patients signs and symptoms of UTIs and to report these signs and symptoms to their HCPs promptly [5,11].

Explain the importance of keeping all scheduled medical appointments and encourage annual eye examinations. Caution patients to contact
their HCPs in the event of illness, injury, and/or infection since they may necessitate a medication dose adjustment [5].

Teach patients and families the signs of acute complications of diabetic therapy, particularly the signs and symptoms of hypoglycemia. Signs and symptoms of hypoglycemia include anxiety, mental changes, dizziness, weakness, pallor, tachycardia, diaphoresis, seizures, confusion, and loss of consciousness that may progress to coma. Teach patients and families that if these signs and symptoms occur, patients should immediately be given carbohydrates such as glucose tablets, honey, or fruit juice. If patients are unconscious, they should be given glucagon or dextrose IV [5,6,11].

Patients and families must also be taught to recognize ketoacidosis. Signs and symptoms of ketoacidosis include acetone breath (fruity-smelling breath), weak, rapid pulse, polyuria, thirst, deep, rapid respirations (Kussmaul’s respirations), changes in level of consciousness, and stupor. Prompt treatment with IV fluids, insulin, and, often, potassium replacement is necessary [5].

### Elder considerations

Several important issues involve elderly patients. These include [5,6,11]:

- Cells become more resistant to insulin with aging. This decreases the older adult’s ability to metabolize glucose. Additionally, insulin release from the pancreas is delayed, and sudden concentrations of glucose occur. Such concentrations cause more prolonged hyperglycemia in elders.

- The thirst mechanism is less efficient in the elderly than in younger adults and children. Thus older adults may not experience the polydipsia that is characteristic of DM in younger adults.

- Healing is often slower in elders than in younger patients. DM compounds this delay in healing.

### CASE STUDY SCENARIOS AND STUDY QUESTIONS

The endocrine system and its pathologies are complex topics. Compounding the complexity is the fact that many endocrine disorders are uncommon, and many HCPs have not had experience in working with patients who are dealing with endocrine pathologies. Recognition and treatment initiation, as well as providing appropriate nursing interventions, depend on a thorough knowledge of the glands of the endocrine system, hormonal production and actions, and clinical manifestations of pathologies. Education offerings must provide opportunities for review. Therefore, the following case study scenarios and study questions have been developed to reinforce the learner’s ability to recognize the various endocrine disorders and their management.

**The endocrine and nervous systems work together to regulate and integrate metabolic activities of the body: Differentiate the ways endocrine hormones and nerve impulses regulate such activities.**

The hormones that are secreted by the glands of the endocrine system are chemical messengers that transfer information from one set of cells to another to coordinate bodily functions [3]. Hormones cause changes in the metabolic activities in specific cells while nerve impulses cause gland secretion and muscle contraction. Hormonal action is rather slow, but of prolonged duration. The action of nerve impulses, on the other hand, is rapid but of short duration [2].

_Andrea is preparing an oral presentation on the pituitary gland as part of her graduate work in nursing. When discussing this gland she must differentiate between the anterior and posterior regions of the pituitary. How can she do this?_

Recall that the pituitary is the “master gland.” It is divided into two regions: the anterior lobe or adenohypophysis and the posterior lobe or neurohypophysis [1,2,3]. The larger anterior lobe produces 6 hormones that are regulated by the hypothalamus: growth hormone (GH), thyroid stimulating hormone (TSH), adrenocorticotrophic hormone (ACTH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), and prolactin (PRL) [1,2,3]. The posterior pituitary is responsible for the secretion of antidiuretic hormone (ADH) and oxytocin [2,3].

Another important differentiation between the actions of the anterior and posterior pituitary is that the anterior pituitary actually produces 6 hormones. Production is regulated by the hypothalamus, which responds to negative and positive feedback mechanisms. The posterior pituitary stores ADH and oxytocin, which are actually produced by the hypothalamus [1,2,3].

_Andrea (and other HCPs) must be aware of the pituitary’s role in endocrine functioning. They must know what hormones are secreted by the pituitary and how these hormones act on various body systems._

Jennifer is a professor of nursing. She is developing an exam that deals with endocrine function. Several of the questions are designed to assess students’ knowledge of what glands produce specific hormones. Here are some samples of questions that Jennifer has written.

- Which gland is responsible for manufacturing a hormone that is the major metabolic hormone of the body? What is the hormone called?

- The correct answer would be the thyroid gland, which produces thyroid hormone, the major metabolic hormone of the body. Thyroid hormone increases metabolic rate, oxygen consumption, glucose absorption, and body temperature. It also affects growth and development and improves the effects of the sympathetic nervous system [3].

- What pancreatic cells produce glucagon? What pancreatic cells produce insulin?

- The alpha cells produce glucagon, which raises the blood glucose level by causing the breakdown of glycogen to glucose. The beta cells secrete insulin, which lowers blood glucose levels by facilitating movement of blood glucose across cells, converting glucose to glycogen [1,3].

- Researchers are investigating the role of which gland in regulation of the sleep-wake cycle?

- The pineal gland, which is located in the middle of the brain, produces melatonin, which is believed to regulate circadian rhythms as part of the sleep-wake cycle [1,2].

- What are the primary sources of sex hormones?

- The gonads are the primary source of sex hormones. In female, the ovaries produce eggs and the steroidal hormones estrogen and progesterone. In male, the testes, located in the scrotum, produce spermatozoa and the male sex hormone testosterone [1,2].

- Give an example of a hormone that is controlled by positive feedback. What is the function of this hormone?

- Oxytocin is controlled by positive feedback. It targets the uterus and mammary glands, causing uterine contractions during childbirth and milk production for lactation. Oxytocin is produced by the hypothalamus and transported via nerves to the pituitary gland, where it is stored and then secreted when needed [1,2,3].

- What pituitary hormone is secreted in response to a state of dehydration?

- ADH (antidiuretic hormone) release is stimulated by dehydration and increased plasma osmolarity. ADH controls water loss by the kidneys. It facilitates water reabsorption in the distal convoluted tubules and collecting ducts of the kidneys. Controlled by negative feedback, ADH release is stimulated by dehydration and increased plasma osmolarity [3].
**What is the major role of the thymus?**
The major role of the thymus seems to be related to the immune system since it produces T cells, which are critical to cell-mediated immunity [1].

**The endocrine system depends on both negative and positive feedback. Differentiate between negative and positive feedback.**
The endocrine system depends on both negative and positive feedback for its regulation. Negative feedback takes place when the rate of production of a particular product decreases as the concentration of that product increases. Negative feedback manages the rate of production to avoid accumulation of a particular product. For example, as the amount of some hormones reach the desired level, the body stops or reduces the rate of their production to avoid excessive accumulation [1,3].

Positive feedback occurs when the rate of production of a particular product increases as the concentration of that product increases. Positive feedback is less common in the body than negative feedback. An example of positive feedback is the secretion of oxytocin that stimulates uterine muscle contraction during labor. As labor progresses, pressure on the cervix continues to stimulate oxytocin release, which continues to stimulate uterine muscle contraction [3].

**Hypopituitarism is a rare disorder that usually presents as a rather slow, predictable pattern of hormonal failures. What are the clinical manifestations of this disease? Why do they occur?**
The pituitary gland secretes TSH, FSH, LH, GH, ACTH, prolactin, ADH, and oxytocin [1,4]. Clinical manifestations depend on the severity of the disease and on the number of hormones that are deficient. Presenting signs and symptoms usually begin with hypogonadism because of decreased FSH and LH levels. In adults, this causes females to stop menstruating, and decreases libido in males. Signs and symptoms of GH deficiency usually follow including osteoporosis, lethargy, subtle emotional changes, adverse lipid changes, and decreased lean-to-fat body mass [4,5].

In children, lack of GH leads to short stature, delayed growth, delayed puberty, and, possibly, dwarfism. If hypopituitarism occurs before puberty, the development of secondary sex characteristics is prevented. In males, this means lack of facial and body hair, undersized penis, testes, and prostate gland, and failure to initiate and maintain an erection. In females, there is a lack of pubic and axillary hair, failure to develop mature breasts, and primary amenorrhea [4,5].

It is important to note that clinical signs and symptoms of hypopituitarism do not usually become apparent until 75% of the gland is destroyed [5].

**Hypopituitarism that involves all of the pituitary hormones is called Panhypopituitarism. It is characterized by involvement of all pituitary hormones. However, it is more likely that only one or more pituitary hormones are involved. This leads to only isolated or partial hypopituitarism [4].**

Hypopituitarism does not usually produce signs and symptoms until..............

Clinical signs and symptoms of hypopituitarism do not usually become apparent until 75% of the gland is destroyed [5].

**What causes dwarfism in children? What signs and symptoms first indicate dwarfism and when do they appear?**
In children, lack of GH causes short stature, delayed growth, delayed puberty, and, possibly, dwarfism [4,5]. Dwarfism is not often evident at birth but initial signs and symptoms appear in the first few months of life, and by six months growth retardation is apparent [5].

Danielle has been diagnosed with acromegaly. What endocrine gland disorder causes this disorder? What clinical manifestations are associated with this disease? How is it treated?
Acromegaly is formally known as hyperpituitarism and is a chronic, progressive disease characterized by hormonal dysfunction and disturbing skeletal overgrowth. Hyperpituitarism also causes gigantism. The difference between acromegaly and gigantism is the age of the person affected. Acromegaly affects adults and has a slow, gradual progression. Overgrowth of cartilage and connective tissue, a big nose, enlarged hands and feet, thickened lips, tongue, gingers, and ear, and changes in the shape of the face are characteristic features. The voice deepens, the skin becomes coarse, oily, and thick, and there is extreme diaphoresis. A variety of mood changes and mental health disturbances may occur. Acromegaly affects males and females equally, usually between the ages of 30 and 50 [5,8].

Gigantism can affect infants and children and is a very rare disorder, affecting only 100 persons to date. Affected children may reach as much as three times the normal height for their age [5].

The most common cause of excessive GH production is a tumor, usually a benign adenoma of the pituitary gland. Treatment involves surgical removal of the tumor and reducing GH production. Removal of a pituitary is complex and should be performed only by surgeons who are experienced in this type of surgery. If the entire tumor cannot be removed via surgery, radiation may be needed to destroy remaining tumor cells and reduce GH levels. Various medications may be prescribed to lower production or block the action of GH.

Since acromegaly progresses so slowly, what is one way that patients and families actually notice changes in the patient's physical appearance?
Since the disease develops so slowly some people may notice physical changes in appearance only by comparing old and current photographs [8].

Several endocrine disorders interfere with normal levels of GH. Therefore, it is important to remember what important point when obtaining blood samples for GH suppression test?
Patients should not be emotionally or physically stressed when obtaining blood samples for the GH suppression test since stress can elevate GH levels [9].

**What is stereotactic radiosurgery? Why is it performed? Who should perform it?**
Stereotactic radiosurgery is also known as Gamma Knife radiosurgery. It is performed in order to direct radiation at tumor cells. For example, in cases of acromegaly when it is necessary to destroy pituitary tumor cells and to gradually reduce GH levels, stereotactic radiosurgery may be used. A high dose of radiation directed at the tumor cells is administered in a single dose while limiting radiation exposure to surrounding normal cells. This type of radiation therapy may bring GH levels to within normal limits within three to five years. Administration of stereotactic radiosurgery requires an extremely high level of technical skill and is available at only a few United States health care facilities.

Mark is a healthy 30-year-old sales manager at a luxury car dealership. While accompanying a potential buyer on a test-drive, a motorcycle runs a red light and crashes into Mark’s car. The airbag deploys and strikes Mark’s head with considerable force. Mark is evaluated at the hospital and diagnosed with a mild concussion. Three days later Mark returns to work feeling fine and resumes his busy schedule. However, later in the afternoon, Mark complains of feeling dizzy and weak. He is extremely thirsty and begins to void large quantities of urine. Mark leaves work early, telling his boss that he is too tired even to drive himself home. His wife arrives to take him home. Alarmed she insists that he see their family physician. Upon arrival at the doctor’s office Mark heads straight for the nearest bathroom, overwhelmed by an urgent need to void. His doctor arranges for Mark to be transported to the hospital for

Angela is inactive, seldom cries, and sleeps excessively. Her face anticipates finding on physical examination? hypothyroidism? What additional signs and symptoms would you anticipate finding on physical examination?

When and why does gestational diabetes insipidus occur?

Gestational diabetes insipidus occurs during pregnancy when an enzyme produced by the placenta destroys the mother’s ADH [5].

Hilary and Nathan have two children. Steven, an active three year old who is “into everything” is a “real handful” his parents say. Even as a baby, Steven was “a challenge.” He awoke frequently during the night and seldom seemed to need to nap. As a toddler Steven constantly “explores” and seldom sits still. His favorite word, of course, is “no.” Hilary gave birth to a baby girl three months ago. Her ecstatic parents tell everyone what a “good” baby little Angela is. She seldom cries, sleeps through the night and naps during the day, and, as her parents describe her, is very “calm.” Nathan’s mother is concerned, however. “This baby is way too “good,” she thinks. The baby has a puffy face and droopy eyelids. Her parents attribute this to “baby fat” and “sleepiness.” Nathan’s mother believes that something is really wrong.

Is Nathan’s mother right or wrong? Is little Angela simply a “good” baby or is there something more ominous going on?

Little Angela is displaying behaviors that are characteristic of hypothyroidism in children. This is a common disorder that affects one in every 2,500 to 3,000 babies [12]. Timing of recognition and treatment is critical. If treatment begins before the age of three months the infant usually experiences normal growth and development. However, if the baby remains untreated beyond the age of two, irreversible mental retardation occurs. Skeletal abnormalities, however, are reversible with treatment [5].

Infant hypothyroidism (cretinism) is usually due to defective embryonic development of the thyroid gland. The next most common cause is related to an inherited enzymatic defect in the synthesis of thyroxine [5].

What aspects of Angel’s clinical manifestations indicate childhood hypothyroidism? What additional signs and symptoms would you anticipate finding on physical examination?

Angela is inactive, seldom cries, and sleeps excessively. Her face is puffy and her eyelids droop. Additional signs and symptoms of childhood hypothyroidism include [5,12]:

- Hoarse cry.
- Abnormally deep tendon reflexes and hypotonic abdominal muscles.
- Short forehead, puffy, wide-set eyes, broad, upturned nose, and a vacant, dull facial expression.
- Protruding abdomen.
- Feeding problems.
- Cold, coarse, thick, and dry skin.
- Dry, brittle hair.
- Slow pulse.
- Below normal body temperature.
- Jaundice.
- Large, protruding tongue.

Angela will require life-long thyroid hormone replacements. Doses in children are higher than in adults because children metabolize thyroid hormone much more rapidly. Initial doses are of moderate strength, but are gradually increased to attain levels adequate for life-long maintenance [5].

A word of caution about thyroid hormone replacements! Too rapid increase in dosage can lead to thyrotoxicity. Signs and symptoms include tachycardia, vomiting, hypotension, tremor, weakness, shortness of breath, cough, swollen extremities, and coma. Immediate emergency treatment is needed [5,11].

Parents should be taught to differentiate between signs and symptoms of a “good” baby and pathology.

Differentiate among the various types of hypothyroidism that affect children.

- Congenital Hypothyroidism (CH): Occurs when the thyroid gland fails to develop or function normally before birth [12].
- Acquired Hypothyroidism-Autoimmune Hypothyroidism: Occurs as the result of an autoimmune disorder called chronic lymphocytic thyroiditis (CLT). In CLT the child’s immune system “attacks” the thyroid gland, causing damage and reduced functioning. Patients who have other types of autoimmune diseases (most often insulin-dependent diabetes) are at higher risk for developing CLT. It is estimated that 20% to 30% of diabetics will develop CLT. Thus, annual screening for CLT is often a routine part of diabetic care [12].
- Acquired Hypothyroidism-Latrogenic Hypothyroidism: Occurs in people who have had their thyroid glands surgically removed or medically destroyed [12].

Louise is preparing for oral comprehensive exams as part of her doctor of nursing science degree. One of the areas of focus is endocrinology. Her study group members are taking turns asking each other to present information as part of their preparation for these exams. Louise is asked:

What is myxedema coma? What causes it? How is it treated?

How should Louise respond?

Myxedema coma is the most serious and dramatic complication of hypothyroidism. It often leads to death. This complication usually has a slow progression. However, stressors such as infection, trauma, exposure to cold, or heart attack can intensify hypothyroidism, causing myxedema coma to develop abruptly [5,6]. Since initial signs and symptoms of hypothyroidism can be vague and nonspecific patients may go undiagnosed for long periods of time. This can increase the possibility that a gradual development of myxedema coma may be the dramatic event that triggers diagnosis.

Signs and symptoms of myxedema coma are significantly depressed respirations, decreased cardiac output, and progressively worsening cerebral hypoxia. Heart rate slows, and blood pressure drops. Patients become hypothermic and stuporous [6].

Treatment involves I.V. administration of thyroid hormones and correction of electrolyte and volume imbalances [5,6]. In order to save the patient’s life, treatment must be swift, and the patient’s condition monitored in the hospital setting. It is therefore imperative that HCPs be taught to recognize myxedema coma and to initiate immediate treatment.

The day arrives for Louise to take her oral comprehensive examination. The examiners ask Louise to:

- Define and describe the clinical manifestations of Graves’ disease.
- Identify risk factors that increase the risk for its development.
- What treatment initiatives are appropriate for Graves’ disease?
- How do you think Louise should respond to these questions?

Graves’ disease, or hyperthyroidism, is a metabolic imbalance characterized by excessive amounts of thyroid hormone in the bloodstream [5,6]. Risk factors for its development include [5,11,16]:

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Anti-thyroid medications. Medication administration continues
General treatment initiatives include [5,6,11,16]:

- Coexistence of other autoimmune disorders.
- Smoking
- Pregnancy.
- Stress.
- Excessive dietary intake of iodine.

Characteristic signs and symptoms of hyperthyroidism include [5,6,11]:
- Enlarged thyroid gland (also referred to as goiter).
- Exophthalmos (abnormally protruding eyes and a characteristic staring gaze).
- Heat intolerance.
- Nervousness.
- Inability to sit still.
- Weight loss even though appetite is increased.
- Diaphoresis.
- Diarrhea.
- Tremors.
- Palpitations.

Hyperthyroidism affects every system of the body. Additional signs and symptoms as they relate to body systems include [5,6,11]:
- Cardiovascular system: Arrhythmias, tachycardia, full bounding pulse, cardiac insufficiency, and resistance to the prescribed therapeutic dose of digoxin. Cardiovascular effects are seen most often in elderly patients.
- CNS: These effects are most often seen in younger patients. They include trouble concentrating, nervousness, anxiety, mood swings, emotional instability that may progress to psychosis, tremors, and clumsiness.
- Gastrointestinal system: Anorexia, nausea, vomiting, diarrhea, and enlarged liver.
- Musculoskeletal system: Muscle weakness and atrophy.
- Reproductive system: Menstrual abnormalities, impaired fertility, decreased libido, and higher incidence of spontaneous abortions in women. Gynecomastia and decreased libido may be found in men.
- Senses: Exophthalmos causes patients to blink less frequently. This leads to dry eyes, reddened conjunctiva and cornea, and corneal ulcers. Patients also have trouble looking upward and strabismus [5,11].

Treatment depends on any underlying causes, the presence and size of goiter, the age of the patient, disease severity, and any current complications [6].

General treatment initiatives include [5,6,11,16]:
- Anti-thyroid medications. Medication administration continues until normal thyroid levels are reached. This can take from three months to two years. If normal levels cannot be reached, radiation or surgical intervention is recommended. Medications must be discontinued gradually to prevent exacerbation.
- Beta blockers, although they do not prevent or inhibit thyroid hormone production, do limit the effects of excessive amounts of thyroid hormone on the body.
- Radioactive iodine therapy limits secretion of thyroid tissue by destroying tissue. It can cause a permanent remission of hyperthyroidism, but may also cause the patient to become permanently hypothyroid.
- Surgery to remove part of the thyroid gland is performed to reduce secretions. If most of the thyroid gland is removed life-long thyroid hormone replacement therapy is necessary.
- Ophthalmopathy is managed by OTC artificial tears during the day and lubricating gels at night.

What is thyroid storm? What are its clinical manifestations?
Thyroid storm is the most serious complication of hyperthyroidism. Also referred to as thyrotoxic crisis, thyroid storm usually occurs in patients with preexisting, though often undiagnosed, thyrotoxicosis. Untreated, it is usually fatal [5,11]. When excessive amounts of T3 and T4 are produced systemic adrenergic activity increases, which leads to overproduction of epinephrine. Excessive amounts of epinephrine cause significant hypermetabolism that, in turn, leads to rapid cardiac, gastrointestinal, and sympathetic nervous system decompensation. Hypertension, tachycardia, vomiting, extreme irritability, and temperature up to 106 °F can occur. Thyroid storm can progress to delirium, coma, and death. The onset of thyroid storm is abrupt and triggered by stressors such as trauma, surgery, infection, or serious events such as stroke, myocardial infarction, preeclampsia, or pulmonary embolism [11].

Differentiate among the various types of thyroiditis.
Thyroiditis is defined as inflammation of the thyroid gland. It is most prevalent in people between the ages of 30 and 50 and is more common in women than in men [5]. The disease usually occurs in three phases: overactive thyroid, underactive thyroid, and return to normal [17]. For patients who do not experience a return to normal thyroid functioning, life-long follow-up and thyroid hormone replacement [5,19].

There are five types of thyroiditis:
- Postpartum Thyroiditis: This is an uncommon disorder characterized by inflammation of the thyroid gland within the first year following childbirth. It is associated with an immune system reaction/underlying autoimmune thyroid condition [18,19]. Some women may experience signs and symptoms only of hyperthyroidism or only of hypothyroidism, but not both [18]. Most women who develop postpartum thyroiditis experience a return to normal thyroid function within 12 to 18 months [18].
- Hashimoto’s Thyroiditis: Hashimoto’s thyroiditis is a chronic progressive disease of the thyroid gland. It is an autoimmune disorder characterized by thyroid infiltration of lymphocytes. As the immune system “attacks” its own thyroid, the gland gradually swells, and damage is sustained [6,19]. Thyroid hormone levels are abnormally low. This type of thyroiditis cannot be cured, and low thyroid hormone levels are usually permanent. Thus, life-long thyroid hormone replacement is usually necessary [19].
- Subacute Thyroiditis: Subacute thyroiditis is a self-limiting, painful inflammation of the thyroid gland that usually occurs following a viral infection [5,6]. Ninety to 95% of patients experience a return to normal thyroid function. The remaining 10% experience permanent hypothyroidism and require life-long thyroid hormone replacement [6].
- Riedel Thyroiditis: Riedel thyroiditis is a rare, chronic inflammatory disease of the thyroid gland. The thyroid undergoes dense fibrosis that replaces normal thyroid parenchyma. Most patients retain normal thyroid functioning, but about 30% become hypothyroid [21].
- Miscellaneous Types: These include acute or infectious thyroiditis due to a bacterial infection; drug induced thyroiditis caused by various drugs (e.g. amiodarone); painless thyroiditis, which produces signs and symptoms similar to postpartum thyroiditis but is not associated with childbirth; and radiation induced thyroiditis that is triggered when radiation iodine treatment is used to treat overactive thyroid glands or for certain cancers [19].

Carla is an RN who works in a busy pediatric private office practice in the Great Lakes region. Mrs. Reynolds brings her three year old daughter in for a wellness checkup. Carla notices that Mrs. Reynolds has a slight swelling in the front of her neck and slight neck distention. Mrs. Reynolds seems to be breathing rather heavily even though she
is sitting down. Carla offers her a cup of water, which Mrs. Reynolds refuses saying, “I must be getting a sore throat. I seem to be having trouble swallowing lately.” Although Carla’s clinical experience as a HCP has been primarily in pediatrics she begins to wonder if Carla has a thyroid problem. Carla encourages Mrs. Reynolds to see a physician. Mrs. Reynolds does so and later calls Carla to thank her. It turns out that Mrs. Reynolds does have a thyroid problem, but has normal levels of thyroid hormones. What type of thyroid problem does Mrs. Reynolds have?

Mrs. Reynolds has been diagnosed as having a nontoxic goiter. It occurs when the thyroid gland is unable to secrete sufficient thyroid hormone to meet the needs of the body. The thyroid gland enlarges as a compensatory mechanism, which usually overcomes mild to moderate hormonal deficiencies. Hence, thyroid hormone levels are normal [5,11,22].

Treatment focuses on reduction of thyroid hyperplasia. The treatment of choice is thyroid hormone replacement therapy with levothyroxine desiccated thyroid or liothyronine, which inhibits secretion of TSH and allows the thyroid gland to rest [5,11].

Small doses of iodine in the form of Lugol’s solution or potassium solutions are given to patients whose goiter is caused by iodine deficiency [5,11].

Other treatment initiatives include [5,11]:
- Diet: Avoid foods such as cabbage, soybeans, peanuts, peaches, peas, spinach, strawberries, and radishes, which decrease the production of T4 [11,22].
- Radiation: Radiation ablation therapy may be used to destroy cells that concentrate iodine for thyroid hormone production.
- Surgery: Large goiters that do not respond to other treatments may require partial removal of the thyroid gland.

Accidental removal of parathyroid tissue during thyroidectomy may cause symptoms related to what electrolyte imbalance? Hypoparathyroidism is an uncommon condition caused by a deficiency of parathyroid hormone (PTH). Since PTH is essential to the regulation and maintenance of calcium and phosphorus, hypoparathyroidism is characterized by hypocalcemia and neuromuscular hyper-excitability [6, 23].

Characteristic signs of hypoparathyroidism include [5,6]:
- Tetany: Manifested by muscle hypertonia and tremors and spasmodic or uncoordinated movements triggered by attempts at voluntary movements.
- Chvostek’s Sign: Hyperirritability of the facial nerve manifested by a spasm of facial muscles, which occurs when muscles or branches of the facial nerve are tapped.
- Trousseau’s Sign: Carpopedal spasm (spasmodic contractions of the muscles of the hands and feet) triggered within three minutes after a blood pressure cuff is applied to the arm and inflated to 20 mm Hg above patient’s systolic pressure.
- Laryngeal spasm.

Central nervous signs and symptoms of hypoparathyroidism are exaggerated during what conditions? Central nervous system signs and symptoms are exaggerated during pregnancy, infection, thyroid hormone withdrawal, before menstruation, hyperventilation, and right before menstruation [5].

What drug interferes with normal parathyroid function especially if renal failure is also a problem? Cimetidine (Tagamet) interferes with normal parathyroid function. Remember that any interference with parathyroid function increases the risk of hypocalcemia.

In the event of acute, life-threatening tetany, what treatment measures must be initiated immediately? Intravenous calcium administration is needed in the presence of acute life-threatening tetany. The most effective calcium solution is ionized calcium chloride (10%). All intravenous calcium preparations are given slowly since it is a highly irritating solution that stings and causes thrombosis. The patient experiences burning flushing feelings of the skin and tongue. However, the intravenous calcium solution also seems to rapidly relieve feelings of anxiety [6].

Hyperparathyroidism is a fairly common disorder, affecting one in 1,000 people and is two to three times more common in females than in males [5]. It is defined as the unregulated, hypersecretion of parathyroid hormone (PTH) [6,24].

Differentiate between the two types of hyperparathyroidism. Explain the pathophysiology of hyperparathyroidism. Describe treatment initiatives for hyperparathyroidism. There are two types of hyperparathyroidism: primary and secondary.

- Primary Hyperparathyroidism: In primary hyperparathyroidism one or more of the parathyroid glands enlarge, increasing PTH secretion and causing elevated serum calcium levels. The most common cause of primary hyperparathyroidism is a single parathyroid adenoma, a benign tumor. Parathyroid hyperplasia is responsible for the remainder of cases [5,6,24].
- Secondary Hyperparathyroidism: Secondary hyperparathyroidism is due to a chronic abnormal stimulus, usually chronic renal failure, vitamin D deficiency, or osteomalacia [5,6,24].

Chronic overproduction of PTH causes increased serum calcium levels. Normal negative feedback mechanisms do not function, and chronic excessive resorption of calcium can lead to osteopenia (loss of some bone density). In secondary hyperparathyroidism overproduction of parathyroid hormone in patients with renal failure add to the pathophysiology of bone disease found in patients on dialysis [24]. Clinical signs and symptoms of primary hyperparathyroidism are due to hypercalcemia and include arrhythmias, hypertension, emotional instability, fatigue, personality changes, severe epigastric pain, peptic ulcers, chronic low back pain, muscle weakness and atrophy, polyuria, catarracts, anemia, and calcifications [5,6].

Secondary hyperparathyroidism causes signs and symptoms of hypocalcemia as well as the underlying disorder. Secondary hyperparathyroidism may be prevented by ensuring a diet that contains adequate amounts of calcium or by taking calcium and vitamin D supplements [5].

Treatment of hypercalcemia includes [6]:
- Administration of intravenous normal saline solution and diuretics such as Lasix and Edecrin to increase urinary excretion of calcium for those patients who are not in renal failure.
- Administration of agents to inhibit bone resorption of calcium. These include Aredia, Cibacalcin, or Didronel.
- Administration of oral phosphate as an anti-hypercalcemia agent.
- Restriction of dietary calcium and discontinuation of drugs that might facilitate hypercalcemia such as thiazides, and vitamin d.
- Dialysis for patients in renal failure or for those whose hypercalcemia does not respond to other treatments.
- Reduced dosage of digoxin since hypercalcemic patients are more vulnerable to the toxic effects of this drug.
- Monitoring of calcium (daily), blood urea nitrogen (BUN), potassium, and magnesium levels.

Surgical removal of parathyroid tissue may relieve bone pain within three days of the surgery. Unfortunately, renal damage may be irreversible [5].

Treatment of secondary hyperparathyroidism focuses on correction and treatment of the underlying cause. Remember that secondary hyperparathyroidism causes hypocalcemia as opposed to primary hypothyroidism, which causes hypercalcemia. Generalized treatment initiatives include [5]:
- Vitamin D therapy.
- Administration of oral calcium preparation in the presence of renal disease.
• Administration of a new classification of drugs (the calcimimetics) approved for the treatment of secondary hyperparathyroidism. These drugs act by stopping the secretion of PTH.

Patients with chronic secondary hyperparathyroidism may find that the parathyroid glands do not revert to normal function even after calcium levels have been returned to normal [5].

Andrea is a nurse practitioner. One of her patients has just been diagnosed with Addison's disease. The patient, who is also a nurse, is distraught, and has many questions about the disease. Andrea needs to do a great deal of patient education. She begins by explaining what Addison's disease is, what causes it, prognosis, and treatment options. What do you think Andrea will tell her patient?

Andrea's disease is also known as adrenal hypofunction or adrenal insufficiency. It occurs in two forms: primary and secondary. Primary adrenal insufficiency is the form commonly referred to as Addison's disease. It originates within the adrenal glands and is characterized by a decrease in mineralocorticoid, glucocorticoids, and androgen secretion [11,25].

Secondary adrenal insufficiency occurs secondary to a disorder outside of the adrenal glands such as a pituitary tumor [5,11].

Adrenal hypofunction affects about eight in 100,000 people [5].

Up to 80% of 80% of cases of Addison's disease cases are caused by an autoimmune process in which circulating antibodies specifically “attack” adrenal tissue. This disease is found primarily in middle-aged females and gradually destroys the adrenal cortex [25].

All patients affected by Addison's disease, in fact all patients affected by either primary or secondary adrenal hypofunction, need lifelong corticosteroid replacement therapy in the form of cortisone or hydrocortisone [5,11].

Andrea should tell her patient that she must take corticosteroid replacement therapy for the rest of her life. The patient should be instructed to wear a medical alert bracelet as well. She should also tell her patient [5,25]:

• To keep her HCP informed of any medications she takes including prescription, OTC, herbal preparations, vitamins, minerals, and other supplements.
• To take steroid therapy in the morning since taking steroids in the late afternoon or evening may stimulate the central nervous system and cause insomnia.
• Caution her that steroid therapy can make it easier to bruise.

Additional nursing concerns include advising patients who are diabetic that steroid replacement therapy may require insulin dose adjustments [5,25].

Patients must also be warned about the possibility of adrenal crisis.

Adrenal crisis is a critical deficiency of mineralocorticoids and glucocorticoids. It is the most serious complication of adrenal hypofunction and can develop gradually or abruptly. It is most likely to occur in patients who fail to respond to hormone replacement therapy, who abruptly stop hormone therapy, who experience physiologic stress, who undergo bilateral adrenalectomy, or who develop an adrenal gland thrombosis [5,11,25].

Signs and symptoms of adrenal crisis include [5,25]:

• Significant weakness and fatigue.
• Abrupt severe pain in the lower back, abdomen, or legs.
• Severe nausea and vomiting.
• Dehydration.
• Hypotension.
• Loss of consciousness.

Untreated adrenal crisis can lead to vascular collapse, renal shutdown, coma, and death. Patients need to receive an emergency bolus of hydrocortisone followed by fluid resuscitation. Patients should carry an emergency kit with a corticosteroid injection with them at all times. They and their families and friends should be taught how to administer the injection [25].

Mark has a real weakness for black licorice, and eats large quantities of it. Eating large amounts of black licorice can cause signs and symptoms that mimic what disease? Why does this occur?

Eating large amounts of English black licorice or licorice-like substances can cause a syndrome that mimics primary hyperaldosteronism. This is because glycyrrhizic acid, a substance found in licorice, has a mineralocorticoid action [5].

Why is diabetes mellitus often found in conjunction with hyperaldosteronism?

Diabetes mellitus is frequently found in conjunction with hyperaldosteronism because hypokalemia, which causes the majority of clinical effects of hyperaldosteronism, can interfere with proper secretion of insulin.

What electrolyte imbalance is strongly linked to a diagnosis of hyperaldosteronism?

Ongoing low levels of serum potassium in patients who do not have edema, are not taking diuretics, have not had GI tract losses due to vomiting or diarrhea, and who have a normal sodium intake strongly suggest hyperaldosteronism [5].

What saline suppression test results would you expect to find in patients who have hyperaldosteronism?

Patients are given intravenous salt solutions after which blood levels of aldosterone and renin are measured. In patients with primary hyperaldosteronism the level of aldosterone in the blood is still high, and the level of renin is low even after this salt loading [27].

In cases of primary hyperaldosteronism, what results would you expect the 24-hour urinary excretion of aldosterone test to show?

Patients ingest a high-sodium diet for five days after which the amount of aldosterone in the urine is measured. In patients with primary hyperaldosteronism, aldosteronism will not be suppressed by the salt load, and the level of aldosterone in the urine will be high [27].

Cushing's syndrome is a hormonal disorder caused by prolonged exposure of the body's tissues to excessive levels of adrenocortical hormones, especially cortisol, related corticosteroids, and, to a lesser extent, androgens and aldosterone [5, 28]. Differentiate between Cushing's syndrome and Cushing's disease. If excess of glucocorticoids is due to a pituitary dependent condition, it is called Cushing's disease [11].

What groups of people may have high levels of cortisol not related to pathological processes?

Since cortisol helps the body to respond to stress, pregnant women in the last three months of pregnancy and highly trained athletes have high levels of this hormone [28].

Differentiate among the three types of Cushing's syndrome.

Cushing's syndrome can be categorized as three types [11]:

• Primary: Primary Cushing’s syndrome is due to disease of the adrenal cortex.
• Secondary: Secondary Cushing’s syndrome is caused by hyperfunction of cells that secrete corticotropin in the anterior pituitary gland.
• Tertiary: Tertiary Cushing’s syndrome is due to dysfunction or injury of the hypothalamus.

Differentiate between Cushing's syndrome and cushingoid syndrome.

Differentiating between Cushing's syndrome and cushingoid syndrome can be challenging. Chronic depression, alcoholism, and long-term treatment with corticosteroids can combine to produce cushingoid syndrome, an adverse consequence characterized by fat deposits between the shoulders and around the waist and many systemic abnormalities. Cushing's syndrome has similar signs, but can be differentiated from cushingoid syndrome by the additional presence of hypertension,
renal problems, hyperglycemia, muscle weakness, tissue wasting, and frequently changing emotional states (emotional lability) [5].

Complete this sentence: In Cushing’s syndrome, immune system suppression can mask __________.

Immune system suppression can mask infection, even severe infections [5]. It is important for HCP to recognize, and to teach patients and families to recognize, even the slightest signs of infection.

Describe how radiation therapy is used to treat Cushing’s syndrome.

If surgical approaches fail, or if a patient is not a candidate for surgery, radiation therapy is a possible alternative treatment. Radiation treatment to the pituitary gland is generally administered over a six-week period. Improvement is noted in 40% to 50% of adults and up to 85% of children [28].

It may take months or even years for patients to feel better after receiving radiation treatment alone. Radiation in conjunction with cortisol-inhibiting drugs can help speed up the recovery process [28].

In Cushing’s syndrome, patients with non-endocrine corticotropin-producing tumors require excision of the tumor followed by drug therapy. Drug therapy is also administered in the event that patient cannot undergo surgery. One of the drugs used is Lysodren. How does Lysodren work?

Mitotane (Lysodren) is toxic to the adrenal cortex. Its administration is referred to as medical adrenalectomy. Side effects of this drug include nausea, vomiting, diarrhea, somnolence, and depression.

Adrenogenital syndrome, perhaps more commonly known as congenital adrenal hyperplasia, is a syndrome caused by disorders of adrenocortical steroid biosynthesis. Most cases of the syndrome are due to the failure of the adrenal glands to produce enough cortisol [5,29]. Inherited adrenogenital syndrome is referred to as congenital adrenal hyperplasia (CAH).

Which is the more severe form of CAH? What are its clinical manifestations?

The salt-losing form of CAH is more severe than the simple form and causes more complete virilization in females. Male external genitalia (but without testes) develop [5,29,30].

Males with salt-losing CAH have no abnormalities in external genitalia. Thus, diagnosis immediately after birth is difficult and usually delayed until severe signs and symptoms develop.

In severe cases signs of salt-losing CAH infants may develop as soon as two to three weeks after birth in both males and females. These signs include vomiting, diarrhea, dehydration, low potassium and sodium levels, and abnormal heart rhythms [29, 30]. Infants are apathetic and fail to eat. These signs indicate the onset of adrenal crisis, which, unless treated promptly, may lead to cardiovascular collapse and cardiac arrest [5].

What does adrenal crisis or evidence of adrenal hypofunction in the first week of life suggest?

Adrenal crisis or evidence of adrenal hypofunction in the first week of life suggests salt-losing CAH [5].

Define hermaphroditism.

Hermaphroditism is a condition appropriately mentioned as part of a discussion of CAH. Hermaphroditism is a rare condition in which children have both ovarian and testicular tissues. External genitalia are usually ambiguous, but may also be completely male or female, which effectively “hides” hermaphroditism until puberty. The child with hermaphroditism almost always has a uterus and ambiguous gonads. Fertility, however, is rare [5].

How is sexual assignment made in hermaphroditism?

Sexual assignment is based on the anatomy of the external genitalia. Reconstructive surgery, during which inappropriate reproductive organs are removed, is performed as early as possible to prevent the development of incongruous secondary sex characteristics at puberty. Hormonal replacement may be needed [5].

Complete this sentence: Research indicates that about ________ of patients newly diagnosed with hypertension have ________.

Research indicates that about 0.5% of patients newly diagnosed with hypertension have pheochromocytoma [5].

Pheochromocytoma causes episodes that are generally characterized by what four factors?

Pheochromocytoma causes episodes typically characterized by [31]:

- Headaches.
- Palpitations.
- Diaphoresis.
- Severe, possibly life-threatening, hypertension.

Urine collection of VMA (vanillylmandelic acid) is part of the diagnostic process for pheochromocytoma. What dietary instructions should the nurse provide prior to this test?

Instruct patients to avoid food that are high in vanillin (such as coffee, nuts, chocolate, and bananas) for two days prior to collection of urine [5].

Treatment of choice for pheochromocytoma is surgical resection of the tumor, which usually cures the associated hypertension. It is important that specific measures be taken prior to surgery to control blood pressure and prevent intraoperative crisis. What measures should be taken preoperatively?

Preoperative measures include [31]:

- Administration of an alpha-adrenergic blocker or metyrosine.
- Volume expansion with isotonic sodium chloride solution.
- Facilitation of liberal salt intake.
- Administration of a beta blocker only after sufficient alpha blockade to “avoid precipitating a hypertensive crisis from unopposed alpha stimulation [31, pg. 2].
- Administration of the last doses of oral alpha and beta blockers on the morning of surgery.

Frank is a registered nurse who is certified in critical care. His 45-year-old sister-in-law telephones him one evening in great distress. She has just been diagnosed with multiple endocrine neoplasia and has an appointment with an endocrinologist to discuss treatment options next week. She asks Frank to accompany her. She also tells Frank that “I have the kind that isn’t cancer.” Frank agrees to go with her, but realizes that he must do some research on the disorder in order to be helpful. He is not familiar with multiple endocrine neoplasia and wonders what his sister-in-law meant by “the kind that isn’t cancer.” What is some information that would be helpful for Frank to know?

There are two types of multiple endocrine neoplasia (MEN) that are well recorded. These are [5]:

- MEN I: MEN I, also called Werner’s syndrome, occurs because of a defect in a gene that carries the code for the protein menin [32]. This defect leads to hyperplasia and tumors of the pituitary and parathyroid glands, islet cells of the pancreas, and, rarely, the thyroid and adrenal glands. It is the more common form of MEN [5].
- MEN II: MEN II, also called Sipple’s syndrome, is a rare familial malignancy caused by genetic mutation [33]. It usually involves medullary cancer of the thyroid and hyperplasia and tumor growth of the adrenal medulla and parathyroid glands [5,33].

Autosomal dominant inheritance is the usual cause of MEN. It affects twice as many females as males, can occur at any age from adolescence through old age, but is rare in children [5].

Clinical manifestations of MEN depend on the glands that are affected. Frank needs to find out what signs and symptoms have affected his sister-in-law and if her physician has talked about specific gland involvement.

Treatment focuses on tumor removal and therapy to control any residual symptoms. Frank will need to discuss specific gland
involvement, tumor size and location, postoperative therapy, and recommendations for surgeons who have experience in the type of surgery that will be needed.

**Differentiate among the different types of diabetes.** There are three types of DM.

- **Type 1:** Type 1 diabetes occurs when the beta cells in the pancreas are destroyed or suppressed. Formerly referred to as juvenile diabetes or insulin dependent diabetes, type 1 diabetes is subdivided into idiopathic and immune-mediated types. In idiopathic diabetes there is permanent deficiency of insulin and no evidence of autoimmunity. In immune-mediated diabetes the body produces an autoimmune attack on pancreatic beta cells, and the pancreas becomes inflamed. By the time signs and symptoms appear, 80% of the beat cells are destroyed. Some experts, however, believe that beta cells are not destroyed but disabled and may later be reactivated [5,6,11].

- **Type 2:** Type 2 diabetes, formerly referred to as adult-onset diabetes or non-insulin dependent diabetes, may be attributed to insulin resistance in target tissues, abnormal insulin secretion, or overproduction of glucose (inappropriate hepatic gluconeogenesis) [6,11].

- **Secondary Diabetes:** Secondary diabetes is so-called because this type occurs “secondarily” to another condition or event. The factors that trigger secondary diabetes include [11]:
  - Physical or emotional stress that can cause prolonged elevation of cortisol, epinephrine, glucagon, and GH. Such elevations increase blood glucose levels and demands on the pancreas.
  - Pregnancy, which causes weight gain, high levels of estrogen, and high levels of placental hormones [11]. This type of diabetes is referred to as gestational diabetes mellitus (GDM). Glucose levels usually return to normal after the women gives birth. However, women who have had GDM have a 40% to 60% chance of developing type 2 diabetes within five to 10 years [5].
  - Use of specific medications such as adrenal corticosteroids, hormonal contraceptives, and other drugs that oppose the desired effects of insulin [11].

**Identify the diagnostic criteria for DM according to the American Diabetes Association (ADA) Guidelines.**

DM can be diagnosed if patients manifest any of the following [5].

- Symptoms of DM plus a random, nonfasting blood glucose level equal to or greater than 200 mg/dl.
- Fasting blood glucose equal to or greater than 126 mg/dl.
- Oral glucose tolerance test (2-our sample) results equal to or greater than 200 mg/dl.

**How are blood glucose levels classified according to the American Diabetes Association?**

- **Normal:** <100 mg/dl.
- **Prediabetes:** 100 to 125mg/dl.
- **Diabetes:** >=126 mg/dl.

**What are the ADA recommended testing guidelines for DM?**

The ADA recommends the following testing guidelines [5].

- Test people age 45 and older who have no symptoms every three years.
- People with characteristic signs and symptoms should be tested immediately.
- High risk groups should be tested “frequently.”

**There are several important issues regarding DM and elderly patients. What are they?**

**Implications for nursing continuing education**

Seldom does a day go by without media-grabbing headlines pertaining to health care. Whether it be about treatment breakthroughs, new means of prevention, or implications for safe and appropriate care, such news usually has significant implications for nurses.

Those implications, in part, require that nurses acquire new knowledge and/or psychomotor skills. In other words, the necessity for new knowledge acquisition is a almost a daily task. To add to the plethora...
of new knowledge requirements is the need to become familiar with diseases and disorders that are not particularly common.

Since many endocrine diseases and disorders are uncommon, and some are quite rare, how can nurses be expected to recognize the sometimes subtle signs and symptoms of what could be serious pathologies?

Nursing professional development specialists are generally responsible for planning, developing, implementing, and evaluating the continuing education endeavors of the nursing department. It is a challenge just to keep up with accrediting organization mandates and education offerings that help nurses to provide care to nurses in various health care specialties. Issues surrounding the recognition of these diseases and provision of nursing care to persons affected by these and other pathologies increase the need for nursing continuing education. How can administrators and managers facilitate continuing education efforts?

There is a need for creativity. Education is not solely delivered in a classroom setting. Various means of distance education can be used to offer brief "spurts" of education. E-mail, texts, and alerts can be sent to nurses' iPhones, computers, and other devices. A disease that is uncommon can be highlighted on a weekly basis and important highlights pertaining to signs and symptoms, pathophysiology, risk factors, and treatment can be provided.

Of course, nurses themselves are primarily responsible for their own education. They cannot rely exclusively on their employing organizations to provide all of the continuing education that is so critical to their professional endeavors. Nurses should suggest education topics for development by nursing professional development and for department-based and unit-based education.

Here are some sources for nurses who are interesting in expanding their knowledge of endocrine disorders and other topics pertinent to the practice of nursing.

- Professional associations: Nurses should join and become active in professional organizations including those pertaining to their particular nursing specialty. These organizations often provide continuing education using various modalities.
- Professional journals: Most journals are offered in hard copy and via electronic media. Keeping abreast of new developments in health care in general and nursing in particular can be facilitated by reading reliable professional nursing journals.
- Companies that specialize in continuing education for HCPs: These companies offer continuing education on a wide variety of topics. Many companies offer a variety of ways to obtain continuing education including in-person seminars, hard copy catalogues, and online programming. These companies can usually be relied upon to offer education offerings that provide contact hours for licensure renewal.
- Professional books: Many nurses opt to purchase professional references that can be downloaded onto their various electronic devices, thus making resources readily accessible.
- Professional libraries: Most health care facilities have their own resource centers. Nurses should be encouraged to find out about the resources within their own organizations.
- Organizational committees and task forces: In this age of shared governance staff nurses are assuming more and more responsibility for managing their own practice. Part of this responsibility often includes serving on various committees and task forces. Meetings of these groups often provide opportunities for continuing education.
- Colleagues: Nurses need to be able to rely on each other for support and as education resources. An atmosphere of open communication should be part of every health care organization. This atmosphere should encourage nurses to not only share their knowledge with each other but to be able to ask questions without fear of embarrassment.
- Internet resources: Internet resources such as the Joint Commission, the Centers for Disease Control (CDC), and other organizations that are known to be reliable sources of accurate information also used for education. Many such organizations have downloadable apps that can be accessed quickly for frequent, even daily updates on issues of importance to nurses and other HCPs. These apps can also be used to search for information specific to particular signs and symptoms and diseases and disorders.

Summary

The endocrine system is quite complex and works in conjunction with the nervous system to maintain the delicate balance that ensures homeostasis. Even slight variations in its functioning can cause significant body disturbances.

Several endocrine diseases and disorders are fairly common, such as thyroid disorder and diabetes mellitus. However, still more of them are uncommon and even rare, making recognition a challenge. To compound the complexity of caring for patients affected by endocrine pathologies, many of these diseases and disorders have insidious onsets, and initial signs and symptoms can be vague and mimic a variety of problems.

It is essential that nurses pursue continuing education from a variety of sources to acquire knowledge about the endocrine system and nursing considerations when caring for patients with diseases and disorders of this body system.

In addition to pursuing their own educational opportunities nurses must enhance their skills as patient/family educators. Many endocrine diseases require life-long follow-up including hormonal replacement. The abilities of patients and families to adhere to life-long treatment regimens depend, in large part, on the ability of nurses to effectively teach them how to do so. Nurses must assess patient/family knowledge acquisition objectively by having them demonstrate necessary psychomotor skills and verbally explain other important points such as a description of signs and symptoms, how to deal with complications, and medication side effects.

To conclude, pathologies of the endocrine system can prove to be challenging to nurses and other HCPs. They are complex, often uncommon diseases and disorders. Nurses may be among the first HCPs to recognize their existence. They may also be among the first to facilitate proper evaluation and treatment of these conditions.
1. The endocrine glands secrete specific hormones produced by the body to regulate cell and organ activity.
   - True
   - False

2. Insulin is secreted by the alpha cells of the pancreas and is responsible for elevating blood glucose levels during fasting or starvation.
   - True
   - False

3. The most common cause of primary hypopituitarism is traumatic brain injury or infection.
   - True
   - False

4. Assessing GH levels is important in hyperpituitarism. It is important to remember that patients should not be emotionally or physically stressed when obtaining blood samples for the GH suppression test since stress can elevate GH levels.
   - True
   - False

5. Characteristic signs and symptoms of diabetes insipidus include polyuria, extreme thirst, nocturia, poor skin turgor, and dehydration.
   - True
   - False

6. Clinical hyperthyroidism can be triggered by excessive dietary intake of iodine or by stress in patients who have latent hyperthyroidism.
   - True
   - False

7. Adrenal insufficiency is also referred to as Cushing’s syndrome.
   - True
   - False

8. Hermaphroditism is a rare condition in which children have both ovarian and testicular tissues.
   - True
   - False

9. Ketoacidosis requires prompt treatment including administration of glucose tablets, honey, or fruit juice.
   - True
   - False

10. A blood glucose level of 100 to 125 mg/dl is classified as prediabetes.
    - True
    - False