Basic Athletic Training
Course Pack D

CHAPTER

28

Endocrine Conditions

STUDENT OUTCOMES

1. Describe the signs and symptoms of hyperthyroidism and hypothyroidism and explain the management of thyroid disorders.
2. Describe the signs and symptoms of acute and chronic pancreatitis and explain the management of both conditions.
3. Explain the insulin regulation of blood glucose levels.
4. Explain the physiological basis of diabetes.
5. Describe the four types of diabetes mellitus.
6. Describe the circulatory and neural complications that can result from diabetes mellitus.

7. Describe the signs and symptoms of insulin shock and diabetic coma and explain the management of insulin shock and diabetic coma.
8. Explain the nutritional recommendations for physically active individuals with type 1 and type 2 diabetes.
9. Identify physical activities that are indicated and contraindicated for a physically active individual with diabetes.

INTRODUCTION

The endocrine system is one of the most critical systems for maintaining a healthy body. The system is a series of ductless organs that secrete hormones into the blood to be transported throughout the body (Fig. 28.1). Endocrine glands include the pituitary, thyroid, parathyroid, adrenal, pineal, and thymus glands. In addition, several organs contain discrete areas of endocrine tissue and produce hormones. Such organs include the pancreas, gonads (i.e., ovaries and testes), and hypothalamus. When hormones are not produced in the proper level, serious conditions may occur. In this chapter, thyroid disorders, such as hyperthyroidism and hypothyroidism, are discussed, as are pancreatitis and diabetes mellitus.

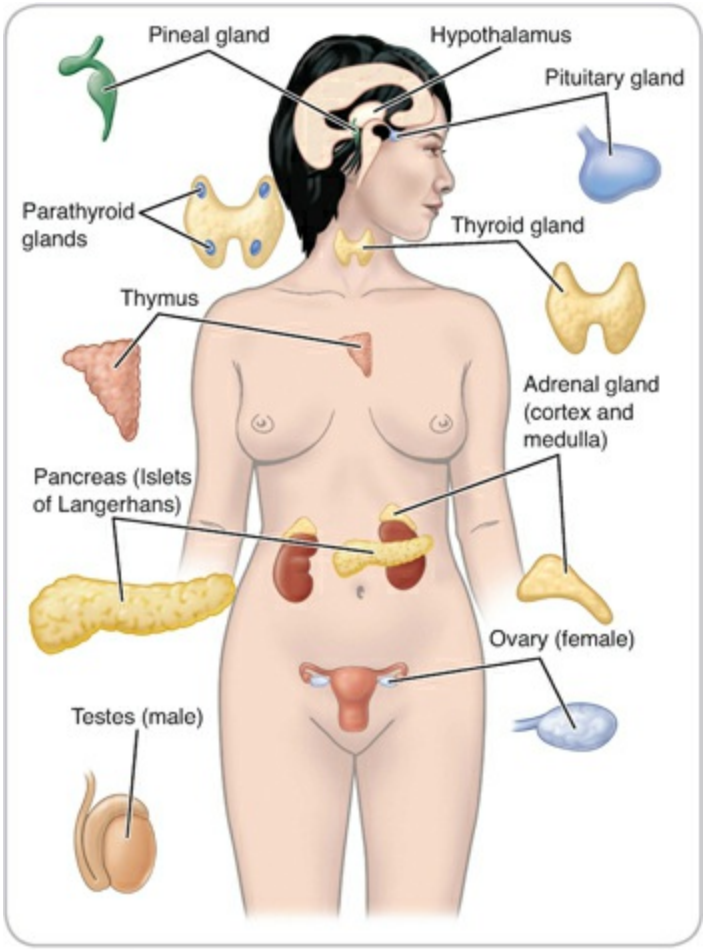


Figure 28.1. Location of the major endocrine glands.

THYROID DISORDERS

The thyroid is a butterfly-shaped gland located at the base of the neck, just below the Adam's apple. The thyroid gland produces two main hormones: thyroxine and triiodothyronine. These hormones maintain the rate at which the body uses fats and carbohydrates, regulate the body temperature, influence the heart rate, and help to regulate the production of protein. The thyroid gland also produces calcitonin, a hormone that regulates the amount of calcium in the blood. The rate at which these hormones are released is controlled by the pituitary gland and the hypothalamus, an area at the base of the brain that acts as a thermostat for the body.

As long as the thyroid produces the necessary amount of hormones,

metabolism functions normally. Occasionally, the thyroid gland produces too much thyroxine causing a condition known as hyperthyroidism (i.e., overactive thyroid disease). This condition can accelerate the body's metabolism by as much as 60% to 100%, causing sudden weight loss, rapid or irregular heartbeat, nervousness, or irritability. In contrast, if too little thyroxine is produced, hypothyroidism occurs.

Hyperthyroidism

Etiology

Hyperthyroidism is caused by the overproduction of thyroxine. This condition frequently produces a cluster of symptoms called thyrotoxicosis. Hyperthyroidism can result from a number of conditions and diseases, most commonly **Graves disease**. In turn, Graves disease may result from genetic and immunological factors and is marked by an enlarged thyroid gland (i.e., goiter), characteristic changes in the structure of the eyes (i.e., ophthalmopathy), and rarely, characteristic skin lesions (i.e., dermopathy).[1] Graves disease usually occurs in people younger than 40 years of age and affects women seven to eight times more than men, and is the most common cause of pediatric hyperthyroidism.[2,3]

Signs and Symptoms

The classic manifestations of hyperthyroidism are goiter, symptoms of thyrotoxicosis, ophthalmopathy, and dermopathy (**Table 28.1**). The symptoms of thyrotoxicosis include nervousness, increased heart rate, loss of sleep, fatigue during ordinary activities, excessive perspiration, and heat intolerance. As a result, sport and physical activity may be contraindicated. Ophthalmopathy results in the eyeball protruding beyond its normal protective orbit when the tissues and muscles behind the eyes swell and push the eyeball forward; this can cause the front surface of the eyeball to become very dry. Other symptoms include the following:

TABLE 28.1 Signs and Symptoms of Thyroid Disorders

HYPERTHYROIDISM	HYPOTHYROIDISM
Sudden weight loss, even when appetite and food intake remain normal or increase	Unexplained weight gain
Rapid or irregular heartbeat (arrhythmia) or pounding of the heart	Elevated blood cholesterol levels
Nervousness, irritability, tremor	Depression
Sweating	Pale, dry skin; puffy face
Changes in menstrual patterns	Heavier than normal menstrual periods
Increased sensitivity to heat	Increased sensitivity to cold
Changes in bowel patterns, especially more frequent bowel movements	Constipation
An enlarged thyroid (goiter), which may appear as a swelling at the base of the neck	Goiter
Fatigue, muscle weakness	Headache
Difficulty sleeping	Sleep apnea Brittle nails; coarse hair Hypertension Paresthesia

- Red or swollen eyes
- Widening of the space between the eyelids
- Excessive tearing or discomfort in one or both eyes
- Light sensitivity, blurry or double vision, and inflammation or reduced eye movement
- Inflammation of the muscles surrounding the eye leading to persistent blinking

Dermopathy is marked by the appearance of thickened patches of skin, usually on the feet or legs, having an "orange skin" texture and uneven pigmentation.[4]

Management

Several treatments are available for hyperthyroidism. The choice of a specific approach depends on the individual's age, sex, physical condition, and severity of the disorder. One approach involves the use of antithyroid medication (i.e., drugs that block hormone production within the thyroid gland). Symptoms usually improve in 6 to 8 weeks, but it may be necessary to continue the use of antithyroid medications for a year or more.[1]

Another approach is to alter the structure of the thyroid gland, either through surgery or with **radioactive iodine**. Radioactive iodine, which is the

most common treatment method in the United States, is taken orally. It is absorbed by the thyroid gland, causing the gland to shrink and the symptoms to subside. The process usually occurs within 2 to 3 months. Because this treatment causes thyroid activity to slow considerably, however, the individual eventually may need to take a medication every day to replace thyroxine.[1,2]

Hypothyroidism

Etiology

Hypothyroidism may be caused by an insufficient quantity of thyroid tissue or by the loss of functional thyroid tissue. An insufficient quantity of thyroid tissue may result from thyroid surgery, in which a portion of the gland is removed; radioactive iodine therapy for another thyroid disease, such as hyperthyroidism or Graves disease; or a congenital thyroid abnormality. The progressive loss of functional thyroid tissue generally is idiopathic, but it is thought to have strong ties to an autoimmune disorder, such as Hashimoto thyroiditis, which is the leading cause of hypothyroidism.[5] The principal symptom of Hashimoto thyroiditis is goiter. Other causes may include iodine deficiency, radiation therapy for cancers of the head and neck, a pituitary disorder whereby the pituitary gland fails to produce enough thyroid-stimulating hormone, or pregnancy. Some women develop hypothyroidism during or after pregnancy, often because they produce antibodies to their own thyroid gland. Left untreated, hypothyroidism increases the risk of miscarriage, premature delivery, and preeclampsia. It also can seriously affect the developing fetus.[4]

Hypothyroidism occurs mainly in women older than 40 years, and the risk of developing the disorder increases with age.[6,7] Others at risk for the condition include those with a close relative, such as a parent or grandparent, who has an autoimmune thyroid disorder; those with previous head and neck or thyroid irradiation or surgery; those with other autoimmune endocrine conditions, such as type 1 diabetes mellitus, adrenal insufficiency, and ovarian failure; and those with some other nonendocrine autoimmune disorders (e.g., celiac disease, vitiligo, pernicious anemia, and multiple sclerosis), primary pulmonary hypertension, and Down and Turner syndromes.[6]

Signs and Symptoms

The signs and symptoms of hypothyroidism vary widely, depending on the severity of the hormone deficiency and on the age of the patient (**Table 28.1**). In children, retarded growth, delayed emergence of secondary sexual characteristics, impaired intelligence, and one or more of the adult symptoms of hypothyroidism may be present. In adults, the initial onset of symptoms such as fatigue, constipation, intolerance to cold, muscle cramps, menorrhagia (prolonged or profuse menses), and sluggishness are barely noticeable and may be attributed simply to the individual getting older. Additional symptoms, however, develop as the metabolism continues to slow; these additional symptoms include mental clouding, diminished appetite, and weight gain. The skin may become dry, and the hair and nails may become brittle. During the advanced stages of the disease, the affected individual may have an expressionless face, sparse hair, and an enlarged thyroid (goiter).[5] Hypothyroidism also may be associated with increased risk of heart disease, primarily because high levels of low-density lipoprotein (LDL) cholesterol (considered to be the "bad" cholesterol) can occur in people with an underactive thyroid. Hypothyroidism also can lead to an enlarged heart, bouts of depression, dementia, and decreased sexual desire (libido).

Management

The treatment of choice for hypothyroidism involves hormone replacement therapy with the synthetic thyroxine. The medication restores adequate hormone levels, reduces fatigue, gradually lowers cholesterol levels elevated by the disease, and may reverse any weight gain.[5] Overtreatment of the synthetic thyroxine may cause subclinical hyperthyroidism, which increases the risk of osteoporosis and atrial fibrillation. Undertreatment causes subclinical hypothyroidism, which may increase cardiovascular risk.[7,8]

PANCREATITIS

The pancreas is a long, flat gland that lies horizontally behind the stomach. The

head of the pancreas rests against the upper part of the small intestine (duodenum), and its tail reaches toward the spleen. The pancreas has two main functions:

1. Produce digestive juices and enzymes to help break down fats, carbohydrates, and proteins (pancreatic exocrine function). Once produced, these juices and enzymes are then transported through a small duct that opens into the duodenum.
2. Secrete the hormones insulin and glucagon into the bloodstream, along with somatostatin, another hormone that helps to regulate their function. The primary role of insulin and glucagon is to regulate the metabolism of carbohydrates and to control the level of blood sugar.

When inflammation develops in the pancreas, these functions are disrupted. The inflammation can be acute or chronic. Symptoms in most cases are mild to moderate but can be severe causing permanent tissue damage.[9]

Acute Pancreatitis

Etiology

Gallstones are the leading cause of acute pancreatitis.[9] These stones migrate out of the gallbladder through the common bile duct, which merges with the pancreatic duct near the entrance to the duodenum. At this junction, gallstones can lodge in or near the pancreatic duct and block the flow of pancreatic juices into the duodenum thus causing inflammation. Digestive enzymes then become active in the pancreas instead of in the digestive tract, causing acute pancreatitis.

Alcohol abuse typically is cited as the second major cause of acute pancreatitis. Other causes of the disorder include drug reactions, viral infections, systemic immunological disorders, pancreatic cancer, or complications from a duodenal ulcer. In some cases, the cause is unknown.

Signs and Symptoms

The main symptom of acute pancreatitis is the sudden onset of persistent, mild

to severe abdominal pain centered over the epigastric region that may radiate to the back and, occasionally, the chest. It can persist for hours or days without relief. Drinking alcohol or eating worsens the pain. Many people with acute pancreatitis sit up and bend forward or curl up in a fetal position, because these positions seem to relieve the pain. Severe attacks also may cause abdominal distention or bruises from internal bleeding, nausea or vomiting, fever, and tachycardia. Vital signs show a rapid pulse, low blood pressure, and elevated temperature.

Management

Acute pancreatitis requires immediate medical care to avoid possibly fatal complications. Treatment largely is symptomatic. The aim is to maintain circulation and fluid volume, decrease pain and pancreatic secretions, antibiotics and control any complications. Analgesic drugs, intravenous administration of fluids, and fasting may be necessary. Acute pancreatitis usually is self-limiting, and pancreatic function eventually is restored.

Chronic Pancreatitis

Etiology

Unlike acute pancreatitis, which may improve spontaneously and without long-term complications, chronic pancreatitis is characterized by permanent damage in terms of structure, function, or both because of progressive inflammation. Alcohol abuse is thought to be the leading cause. Less common causes include autoimmune disease, systemic immunological disorders, obstruction of the pancreatic duct by tumor, and genetic abnormalities. The disease usually presents itself in adults between 30 and 40 years of age, but some patients present before the age of 30 years.[9]

As the inflammation persists, it slowly destroys the pancreas, and the organ becomes less able to secrete the enzymes and hormones that are needed for proper digestion. This leads to poor absorption (malabsorption) of nutrients, particularly fat, causing weight loss and passage of fat-containing stools that are loose, malodorous, and oily in appearance. Eventually, the cells that

produce insulin are impaired, causing diabetes. Chronic pancreatitis, especially the hereditary form, also is linked to increased risk for pancreatic cancer.[9]

Signs and Symptoms

Chronic pancreatitis differs from acute pancreatitis in that the inflammation is a slow, progressive destruction of the tissues, often over many years. This disease usually is less obvious, and during its early stages, the signs and symptoms can be difficult to recognize. Some people with chronic pancreatitis have no pain. Others have intermittent periods of mild to moderate abdominal pain linked to recurrent bouts of acute pancreatitis, often worsening after drinking alcohol or eating a meal. Other symptoms may include nausea, vomiting, fever, bloating, gas, weight loss, malabsorption, and hyperglycemia.

Management

Treatment of chronic pancreatitis is aimed at managing pain and correcting any nutritional disorders that result from malabsorption. A low-fat diet is recommended. Pain may be relieved through medication or various surgical procedures. Pancreatic enzyme replacement therapy may help to correct malabsorption problems and provide additional pain relief. If the condition is caused by alcoholism, the mortality rate is high.[1,9]

DIABETES MELLITUS

During a rehabilitation session, a patient becomes very dizzy, complains of a headache, and reports being very hungry. The patient is sweating profusely, the skin appears to be pale and clammy, and movement is somewhat clumsy. In addition, the person appears to be swallowing an excessive number of times. What condition should be suspected, and how should the condition be managed?

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by a

near or absolute lack of the hormone insulin, insulin resistance, or both. The disease affects approximately 30 million children and adults in the United States—9.3% of the population—and ranks fifth among the leading causes of death in the United States. The most life-threatening consequences of diabetes are heart disease and stroke, which strike people with diabetes more than twice as often as they do those without the disorder.[10] Several factors increase the risk and severity of diabetes, including heredity, increasing age, minority ethnicity, obesity, female gender, stress, infection, a sedentary lifestyle, and a diet high in carbohydrates and fat.

Physiological Basis of Diabetes

Carbohydrates in human nutrition supply the body's cells with glucose to deliver energy to the body's systems. Eating causes the blood glucose (BG) to rise, stimulating the pancreas to release insulin. Under normal conditions, BG ranges between 80 and 120 mg per dL. The main effect of insulin is lowered blood sugar levels, but it also stimulates amino acid uptake, fat metabolism, and protein synthesis in muscle tissue. Insulin lowers blood sugar by enhancing membrane transport of glucose and other simple sugars from the blood into body cells, especially the skeletal and cardiac muscles (**Fig. 28.2**). It does not accelerate the entry of glucose into liver, kidney, and brain tissue, all of which have easy access to BG regardless of insulin levels. After glucose enters the target cells, insulin

- Promotes the oxidation of glucose for adenosine triphosphate production.
- Joins glucose together to form glycogen.
- Converts glucose to fat for storage, particularly in adipose tissue.

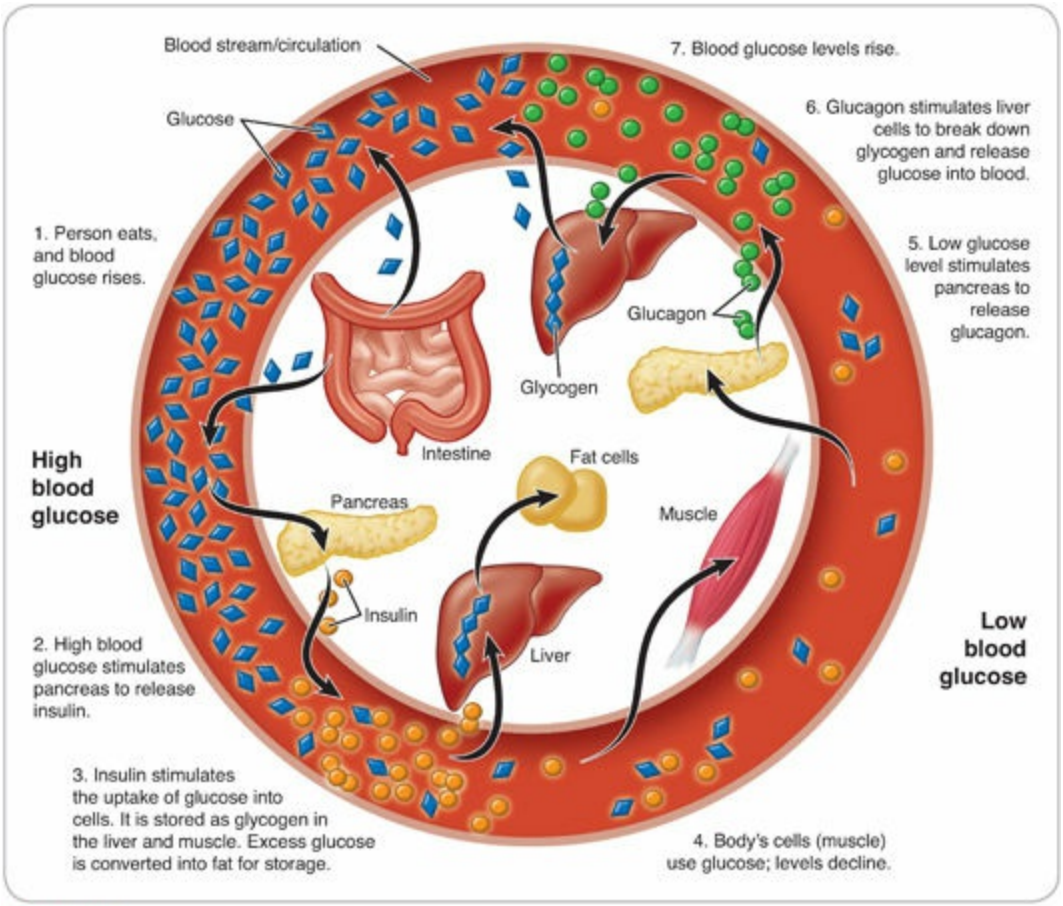


Figure 28.2. Maintaining a balance of blood glucose. Insulin must be available to stimulate uptake of BG into the body's cells. As the cells use glucose, blood levels decline, and the liver responds by releasing glucagon into the bloodstream. Glucagon stimulates liver cells to break down stored glycogen and releases glucose into the blood and, in doing so, raises BG to normal levels.

As a general rule, energy needs are met first, and then liver and muscle cells can assemble the excess single glucose cells into long, branching chains of glycogen for storage. The liver cells also are able to convert excess glucose to fat for export to other cells. High BG levels return to normal as excess glucose is stored as glycogen, which can be converted back to glucose, and as fat, which cannot be converted back to glucose.

When BG falls, as occurs between meals, other special cells of the pancreas respond by secreting glucagon into the blood. Glucagon raises BG by stimulating the liver to dismantle its glycogen stores and release glucose into the blood for use by the body's cells. Epinephrine, another hormone, also can stimulate the liver cells to return glucose to the blood from liver glycogen. This "fight-or-flight" response often is triggered when a person experiences stress.

When insulin activity is absent or deficient, as in those with diabetes, the level of blood sugar remains high after a meal, because glucose is unable to move into most tissue cells, causing BG levels to increase to abnormally high levels. Increased osmotic blood pressure drives fluid from the cells into the vascular system, leading to cell dehydration. The excess glucose is passed into the kidneys, resulting in **polyuria**, a huge urine output of water and electrolytes that leads to decreased blood volume and further dehydration. Serious electrolyte losses also occur as the body rids itself of excess ketones. Because ketones are negatively charged ions, they carry positive ions out with them; as a result, sodium and potassium ions are lost. An electrolyte imbalance leads to abdominal pains and possible vomiting, and the stress reaction spirals. Dehydration stimulates the hypothalamic thirst centers, causing **polydipsia**, or excessive thirst. In response, the body shifts from carbohydrate metabolism to fat metabolism for energy. The final cardinal sign, **polyphagia**, refers to excessive hunger and food consumption—a sign that the person is "starving." As such, although plenty of glucose is available, it cannot be used. In severe cases, blood levels of fatty acids and their metabolites rise dramatically, producing an excess of ketoacids that, in turn, results in acidosis. Acetone, a by-product of fat metabolism, is volatile and blown off during expiration, which gives the breath a sweet or fruity odor. If the condition is not rectified with insulin injection, further dehydration and ketoacidosis result as the ketones begin to spill into the urine (i.e., ketonuria). If untreated, ketoacidosis disrupts virtually all physiological processes, including heart activity and oxygen transport. Severe depression of the nervous system leads to confusion, drowsiness, coma, and finally, death.

Types of Diabetes

The National Diabetes Data Group and the World Health Organization recognize four types of DM:

1. Type 1
2. Type 2

3. Gestational DM
4. Other specific types of diabetes

Each group is characterized by high BG levels, or hyperglycemia. For individuals with symptoms of DM, such as excessive thirst and urination or unexplained weight loss, only an elevated fasting plasma glucose of greater than 126 mg per dL, or a random venous plasma glucose of greater than 200 mg per dL, is required to confirm the diagnosis. High blood pressure, LDL cholesterol, and glucose levels are linked to serious complications in the diabetic. Recently, the American College of Physicians, National Institutes of Health, and American Diabetes Association established new guidelines for monitoring those with diabetes. In addition to traditional glucose testing that measures blood sugar levels, a new gold standard is the A1C test, which measures levels of a substance in the blood called hemoglobin A1C. The A1C test reports control of glucose levels for the past 2 to 3 months. The U.S. Food and Drug Administration has approved several A1C monitors for home use, and the ideal parameters for diabetics are as follows[10,11]:

- A1C <7%
- Blood pressure <135/80 mm Hg
- LDL cholesterol <100 mg per dL

Because athletic trainers most likely encounter physically active individuals with type 1 or type 2 DM, these conditions receive more focus and are compared in **Table 28.2**.

TABLE 28.2 Comparison of Type 1 and Type 2 Diabetes Mellitus

	TYPE 1	TYPE 2
Former names	Juvenile-onset diabetes Insulin-dependent diabetes mellitus	Adult-onset diabetes Non–insulin-dependent diabetes mellitus
Age of onset	Usually before 30 years	Usually after 30 years
Type of onset	Abrupt (i.e., days to weeks)	Usually gradual (i.e., weeks to months)
Nutritional status	Almost always lean	Usually obese
Insulin production	Negligible to absent	Present but may be in excess and ineffective because of obesity
Insulin	Needed for all patients	Necessary in only 20%–30% of patients
Diet	Mandatory, along with insulin for control of blood glucose	Diet alone frequently is sufficient to control blood glucose.
High incidence	White population	Women with a history of gestational diabetes, blacks, Native Americans, Hispanics
Family history	Minor	Common link

Type 1 Diabetes Mellitus

In type 1 DM, the pancreas cannot synthesize insulin. As such, the individual must obtain insulin to assist the cells in taking up the needed fuels from the blood. This insulin must be injected. It cannot be taken orally, because insulin is a protein and the gastrointestinal enzymes would digest it. Type 1 DM (formerly called insulin-dependent diabetes or juvenile diabetes) is considered to be an autoimmune disorder and is one of the most frequent chronic childhood diseases. The onset usually is acute, developing over a period of a few days to weeks. Individuals who develop type 1 DM are usually younger than 25 years, with an equal incidence in both sexes and an increased prevalence in the white population.[11] A family history of type 1 DM or other endocrine disease is found in a small number of cases. The disease typically has an onset before 30 years of age in people who typically are not obese; however, it can begin at any age. Obese individuals generally have a more difficult time balancing glucose levels. In addition, the effects of exercise on the metabolic state are more pronounced in these individuals, and the management of exercise-related problems is more difficult.

Type 2 Diabetes Mellitus

Type 2 DM is the most common form of diabetes and is associated with a family history of diabetes, older age, obesity, and lack of exercise. It is more common in women with a history of gestational diabetes and in African

Americans, Latinos, Native Americans, Asian Americans/Pacific Islanders.[10] Although the exact cause of type 2 DM is unknown, high BG and insulin resistance are major contributing factors. The body cannot use insulin correctly and the resistance is related to an insulin secretory defect, which prohibits or limits the transfer of insulin across the cellular membrane. As in type 1 DM, BG rises to an inappropriate level (i.e., hyperglycemia). The high BG stimulates the pancreas to make insulin, exhausting the insulin secretory cellular defect and reducing the cell's ability to continue making insulin. Therefore, type 2 DM appears to be a self-aggravating condition.

Onset is typically after 40 years of age, but type 2 DM also is seen in obese children. Obesity, a major factor in adults, affects nearly 90% of adults with type 2 DM. Compared to normal-weight individuals, obese people require much more insulin to maintain a normal BG level. More insulin is produced, but as body fat increases, insulin receptors are reduced in number and ability to function. Consequently, insulin resistance increases, and adipose and muscle tissues become less able to take up glucose. At some point, the body cannot supply enough insulin to keep up, and type 2 DM develops.

Gestational Diabetes Mellitus

Gestational DM is an operational classification (rather than a pathophysiological condition) that identifies women who develop DM during gestation. The condition is associated with older age, obesity, and a family history of diabetes. Women who are diagnosed with DM before pregnancy are not included in this group. Women who develop type 1 DM during pregnancy and women with undiagnosed, asymptomatic, type 2 DM that is discovered during pregnancy are classified as having gestational DM. Most women classified with gestational DM have normal glucose homeostasis during the first half of the pregnancy, but the mother's BG rises because of hormones secreted during the latter half of the pregnancy. As a result, the mother cannot produce enough insulin to handle the higher BG, leading to hyperglycemia. The hyperglycemia usually resolves after delivery, but it places the woman at risk for developing type 2 DM later in life.

Other Specific Types of Diabetes

Types of DM from various known causes are grouped together to form the classification called "other specific types." This group includes those with genetic defects of β-cell function or with defects of insulin action and persons with pancreatic disease, hormonal disease, and drug or chemical exposure.[10]

Complications of Diabetes Mellitus

In both type 1 and type 2 DM, glucose fails to enter into the cells and accumulates in the blood, which can lead to both acute and chronic complications. **Figure 28.3** illustrates some of the metabolic consequences of untreated diabetes. Over the long term, these metabolic changes can lead to serious chronic complications. Chronically elevated BG levels can damage the blood vessels and nerves, leading to circulatory and neural damage. Failure to adequately balance nutrition, exercise, insulin injections, and BG levels also can cause a physically active individual to experience insulin shock or diabetic coma.

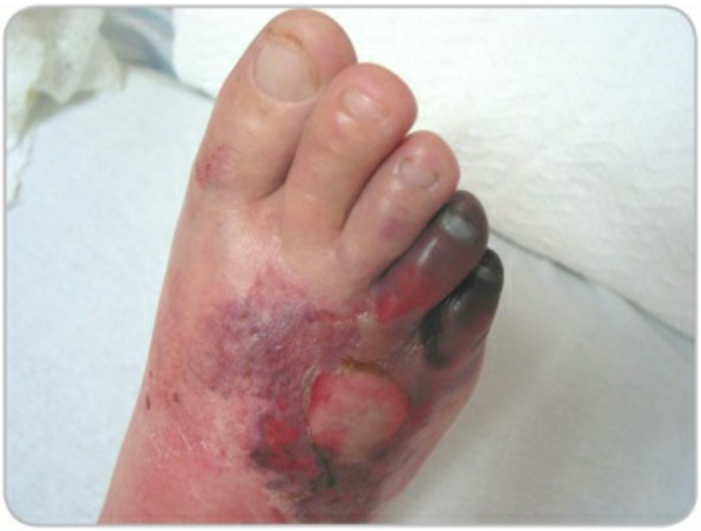


Figure 28-3. Metabolic consequences of untreated diabetes. The metabolic consequences of type 1 diabetes mellitus are more rapid and severe than those of type 2 diabetes mellitus. In type 1, no insulin is available to allow glucose to enter the cells, resulting in a cascade of metabolic changes. In type 2, some glucose enters the cells. Because the cells are not "starved" for glucose, the body does not shift into the metabolism of fasting (i.e., losing weight and producing ketones).

Circulatory Complications

Coronary heart disease, the most common form of cardiovascular disease, usually involves atherosclerosis and hypertension. Atherosclerosis is the accumulation of lipids and other materials in the arteries. The condition begins with the accumulation of soft, fatty deposits along the inner arterial wall, especially at branch points. These deposits eventually enlarge and become hardened with minerals, forming plaque, which in turn hardens and narrows the arteries. Blood platelets cause clots to form whenever an injury occurs. Under normal conditions, these clots form and dissolve in blood all the time, but with atherosclerosis, clots form faster than they are dissolved. In those with diabetes, atherosclerosis tends to develop early, progress rapidly, and be more severe. More than 84% of people with diabetes die as a result of cardiovascular diseases, especially heart attacks.[10] Complications in the capillaries also may lead to impaired kidney function and retinal degeneration with accompanying loss of vision. Approximately 75% of people with diabetes have impaired kidney function, loss of vision, or both.[10] Consequently, diabetes is the leading cause of kidney failure and blindness.

Nerve Complications

Diabetes causes nerves to deteriorate. The initial symptom often is a painful, prickling sensation in the arms and legs. Later, loss of sensation may occur in the hands and feet. Injuries to these areas may go unnoticed, and infections can progress rapidly. Undetected injuries and infection may lead to aseptic necrosis of tissue (i.e., **gangrene**), necessitating amputation of the involved limbs (most often the feet or legs). For this reason, it is critical that those with diabetes take very good care of their feet and visit a podiatrist regularly. As a preventive measure, silica gel shoe inserts should be used and cotton polyester socks worn to prevent blisters and keep the feet dry. All open wounds should be treated promptly, cleaned daily, and carefully checked for infection.

Nerve damage also can retard gastric emptying. When the stomach empties slowly after a meal, the person may experience a premature feeling of fullness. This can lead to bloating, nausea, vomiting, weight loss, and poor control of BG resulting from irregular nutrient absorption.

Hypoglycemia

Hypoglycemia, which is common in those with type 1 insulin-treated diabetes, can range from very mild, lower levels of glucose (60 to 70 mg per dL) with minimal or no symptoms to severe hypoglycemia with very low levels of glucose (< 40 mg per dL) and neurological impairment. Although hypoglycemia can occur with any individual, it is critical in a person with type 1 DM, because the ability to recover from it is limited. In a person with diabetes, hypoglycemia associated with insulin therapy may be related to errors in dosage, delayed or skipped meals, exercise, intensity of BG control, variation in absorption of insulin from subcutaneous injection sites, variability of insulin binding, and impairment of counterregulation. When left untreated, this condition can lead to insulin shock.

Insulin Shock

Etiology

Exercise lowers blood sugar; as such, any exercise must be counterbalanced with increased food intake or decreased amounts of insulin. Hypoglycemia results if BG falls below normal levels. Although any individual can experience hypoglycemia, it is particularly critical for the physically active person with diabetes to address the situation immediately.

Signs and Symptoms

Contrary to the slow onset of a diabetic coma, hypoglycemia has a rapid onset. Signs and symptoms include dizziness; headache; intense hunger; aggressive behavior; pale, cold, and clammy skin; profuse perspiration; salivation; drooling; and tingling in the face, tongue, and lips. Other observable signs may include a staggering gait, clumsy movements, confusion, and a general decrease in performance.

Management

Because glucose levels in the blood are low compared to high levels of insulin, treatment focuses on getting 10 to 15 g of a fast-acting carbohydrate

into the system quickly. This can be found in 4 oz (one-half cup) of juice or regular soda, 1 tablespoon of honey or corn syrup, 2 tablespoons of raisins, 4 packets or 4 teaspoons of sugar, and four or five saltine crackers.[10] Chocolates, which contain a high level of fat, should not be used for treating a hypoglycemic reaction, because the fat interferes with the absorption of sugar. If the person is unconscious or unable to swallow, the patient should be rolled on his or her side, and close attention should be given to the airway so that saliva drains out of the mouth, not into the throat. Sugar or honey should be placed under the tongue, because it is absorbed through the mucous membrane. Recovery usually is rapid.

After initial recovery, the patient should wait 15 minutes and check the blood sugar level. If the level is still less than 70 mg per dL or no meter is available and the patient still has symptoms, another 10 to 15 g of carbohydrates should be administered. Blood testing and treatment should be repeated until the BG level has normalized. Even when the BG level has returned to normal, however, physical performance and judgment may still be impaired, or the patient may relapse if the quick sugar influx is rapidly depleted. After the symptoms resolve, the patient should be instructed to have a good meal as soon as possible to increase carbohydrates in the body.

Many individuals with diabetes who experience repeated bouts of hypoglycemia have glucagon injection kits that contain a syringe prefilled with a diluting solution and a vial of glucagon powder. Once the solution and powder are mixed, they are injected into the upper arm, thigh, or buttock. Although the individual may instruct friends and family members on how to mix, draw up, and inject the glucagon, athletic trainers may not automatically be included in the process. It is critical, however, that athletic trainers know whether and under what circumstances a glucagon injection should be administered as well as when to activate the emergency plan. The athletic trainer should be properly trained by the individual's physician or designee, and permission to administer the solution should be documented.

Diabetic Coma

Etiology

Without insulin, the body is unable to metabolize glucose, leading to hyperglycemia. As the body shifts from carbohydrate metabolism to fat metabolism, an excess of ketoacids in the blood can lower the blood pH to 7.0 (i.e., normal pH is 7.35 to 7.45), leading to a condition called diabetic ketoacidosis. This is manifested by ketones in the breath, **ketonemia**, and **ketonuria**.

As the name implies, diabetic coma is a serious condition and is considered to be a medical emergency. The emergency plan, including summoning emergency medical services (EMS), should be activated.

Signs and Symptoms

Symptoms appear gradually and often occur over several days. The patient becomes increasingly restless and confused and complains of a dry mouth and intense thirst. Abdominal cramping and vomiting are common. As the patient slips into a coma, signs include dry, red, warm skin; eyes that appear deep and sunken; deep, exaggerated respirations; a rapid, weak pulse; and a sweet, fruity acetone breath similar to nail polish remover.

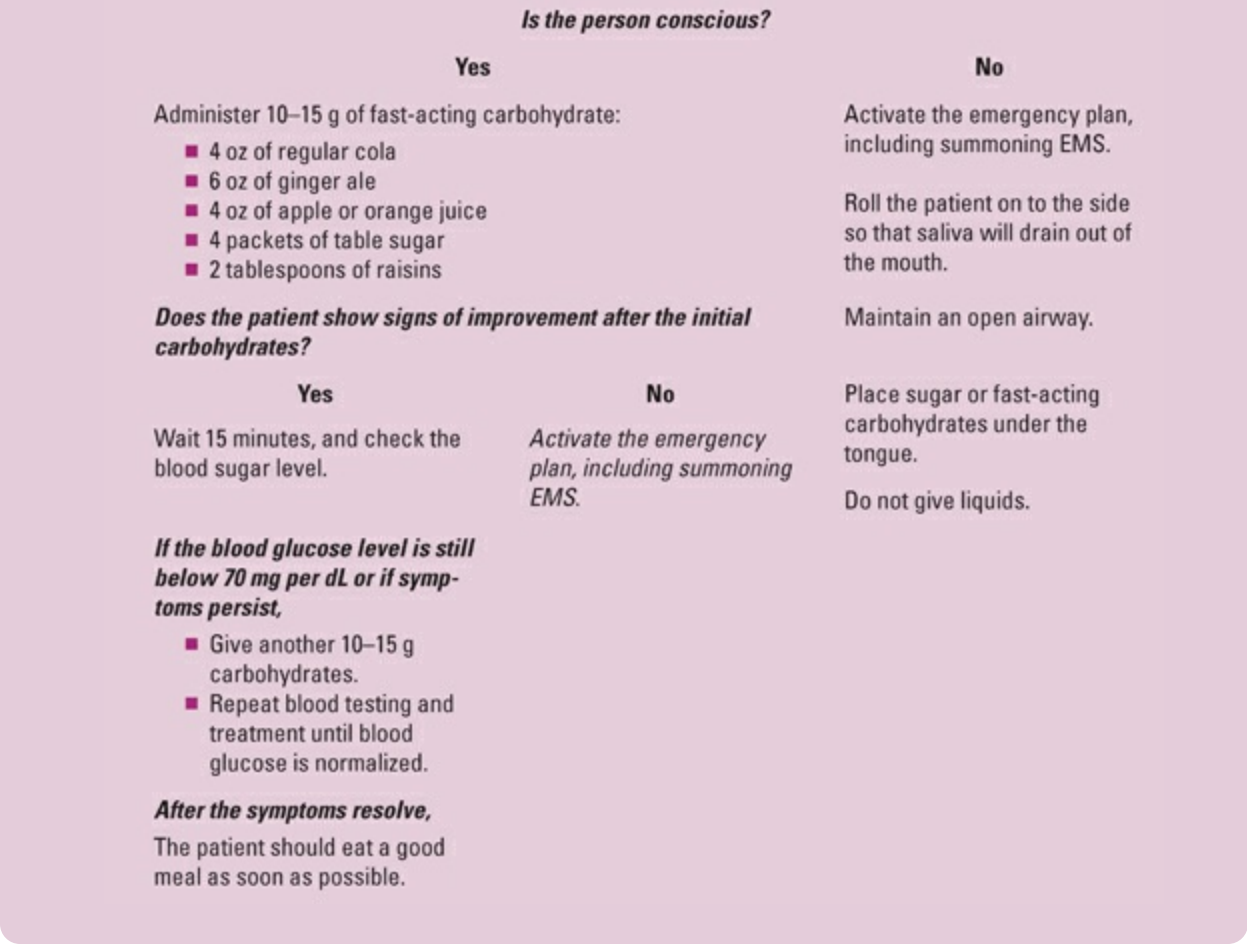
Management

It is not usually possible to diagnose with certainty whether a patient is in a diabetic coma or insulin shock. As such, a conscious patient should be given glucose or orange juice. If recovery is not rapid, a medical emergency exists, and the emergency plan should be activated. The additional glucose will not worsen the condition provided that the patient is transported immediately. If the person is unconscious or semiconscious, nothing should be given orally. Instead, an open airway should be maintained, the person treated for shock, and the emergency plan activated. Application Strategy 28.1 summarizes the management of insulin shock and diabetic coma.

APPLICATION STRATEGY 28.1

Management Algorithm for Diabetic Emergencies

Look for a medic alert tag.



Is the person conscious?

Yes

Administer 10–15 g of fast-acting carbohydrate:

- 4 oz of regular cola
- 6 oz of ginger ale
- 4 oz of apple or orange juice
- 4 packets of table sugar
- 2 tablespoons of raisins

Does the patient show signs of improvement after the initial carbohydrates?

Yes

Wait 15 minutes, and check the blood sugar level.

If the blood glucose level is still below 70 mg per dL or if symptoms persist,

- Give another 10–15 g carbohydrates.
- Repeat blood testing and treatment until blood glucose is normalized.

After the symptoms resolve,

The patient should eat a good meal as soon as possible.

No

Activate the emergency plan, including summoning EMS.

No

Activate the emergency plan, including summoning EMS.

Roll the patient on to the side so that saliva will drain out of the mouth.

Maintain an open airway.

Place sugar or fast-acting carbohydrates under the tongue.

Do not give liquids.

Nutrition and Exercise Recommendations

Control of diabetes depends on a balance of glucose levels, insulin production, nutrition, and exercise. Before initiating an exercise program, a physician should be consulted about diet and normal BG levels documented, because strenuous exercise is contraindicated for some individuals with diabetes. With the advent of BG self-monitoring, exercise is encouraged if certain precautions are followed. BG levels should be taken 30 minutes before and 1 hour after exercise to determine the effects of exercise on BG; this allows better regulation of food intake and insulin dosage.

Nutritional Recommendations for Type 1 Diabetes

Normally, the body secretes a constant, baseline amount of insulin at all times,

and it secretes more insulin as the BG level rises following meals. Individuals with type 1 DM must learn to adjust their insulin doses and administration schedule to accommodate meals, physical activity, and health status. Recommendations for maintaining optimal nutritional status focus on controlling BG levels, achieving a desirable blood lipid profile, controlling blood pressure, and preventing and managing complications from diabetes. The diet should provide a consistent daily intake of carbohydrates at each meal and each snack to minimize fluctuations in the BG level. A meal should be ingested 1 to 3 hours before physical activity, and the individual should have approximately 10 to 15 g of additional carbohydrates 30 minutes before moderate activity or approximately 20 to 30 g of carbohydrates before vigorous activity. During intense exercise, 15 to 30 g of carbohydrates should be ingested every 30 minutes. A snack of carbohydrates should follow the exercise period; carbohydrates are readily available from fruits, fruit juices, yogurt, crackers, and other starches.

When exercise lasts for several hours, insulin requirements decrease; as such, total insulin dosage should be decreased 20% to 50%. Injection administration should be timed so that peak activity does not take place when high insulin levels are present (i.e., 2 to 4 hours after injection). If this is not possible, the individual should eat a high-carbohydrate snack, such as juice and crackers or milk and cookies, approximately 30 minutes before the resumption of activity.[10] In addition, food should always be available for supplemental feeding (e.g., in the locker room, on the bus, and in the athletic training kit).

Nutritional Recommendations for Type 2 Diabetes

As with type 1 DM, an individual with type 2 DM must maintain a near-normal BG level by delivering the same amount of carbohydrates each day, spaced evenly throughout the day. Eating too much carbohydrate at one time can raise BG too high, stressing the already compromised insulin-producing cells. Eating too little carbohydrate can lead to hypoglycemia. In addition, those who have elevated blood lipids may need to watch not only their carbohydrates but also their fat intake. When an individual lowers fat intake, the percentage of

calories from carbohydrates increases. A high-carbohydrate diet raises triglycerides and lowers high-density lipoprotein. For those individuals accustomed to a high-fat diet, complying with a low-fat diet may be difficult. When combined with regular exercise, even moderate weight loss (i.e., 10 to 20 lb) can improve BG control and blood lipid profiles, help to reverse insulin resistance, and reduce blood pressure.[10]

Exercise Recommendations

Exercise is a critical component in managing diabetes. Aerobic exercise can decrease the requirements of insulin and increase the body's sensitivity to it. Exercise also can help an individual to attain and maintain ideal body weight; decrease the risk for hypertensive diseases, including cardiovascular and peripheral vascular disease; and slow the progression of **diabetic nephropathy**. It is recommended that people with prediabetes or diabetes or the general adult public should aim for a minimum of 30 minutes most days. Children and teens should aim for at least 60 minutes most days. The goal is to increase the heart rate and cause the individual to break a light sweat.[10] It is recommended that physically active individuals with diabetes follow the guidelines listed in **Box 28.1**.

BOX 28.1 Guidelines for Safe Exercise by Physically Active Individuals with Diabetes

- Have a routine medical examination and be cleared for activity.
- Develop a balanced program of diet and exercise under a physician's supervision.
- Wear identification (e.g., bracelet or necklace) indicating that the individual has diabetes.
- Eat at regular times throughout the day.
- Avoid exercising at the peak of insulin action and in the evening, when hypoglycemia is more apt to occur.
- Adjust carbohydrate intake and insulin dosage before physical activity.

- Check blood glucose levels before, during (if possible), and after physical activity.
- Prevent dehydration by consuming adequate fluids before, during, and after physical activity.
- Have access to fast-acting carbohydrates during exercise to prevent hypoglycemia.
- Avoid alcoholic beverages or drink them in moderation.
- Avoid cigarette smoking.

Despite the benefits of exercise, individuals with type 2 DM who have lost protective neural sensation should not participate in treadmill walking, prolonged walking, jogging, or step exercises. Recommended exercises include low-resistance walking, swimming, bicycling, rowing, chair exercises, arm exercises, and other non–weight-bearing exercises. Activities that require resistance strength training are permissible as long as no indications of retinopathy or nephropathy are present. Scuba diving, rock climbing, and parachuting are strongly discouraged.

Hypoglycemia, or low blood sugar, should be suspected. It is necessary to get sugar into the system quickly. This can be accomplished by giving the patient table sugar, honey, sugared candy, orange juice, or a regular soda. Recovery should be rapid. The patient should be instructed to have a good meal as soon as possible. Finally, the patient should be closely monitored for any relapse.

SUMMARY

1. Hyperthyroidism is caused by overproduction of thyroxine, leading to a cluster of symptoms called thyrotoxicosis. The symptoms of thyrotoxicosis include nervousness, increased heart rate, loss of sleep, fatigue during ordinary activities, excessive perspiration, and heat

intolerance.

2. Hypothyroidism may be caused by an insufficient quantity of thyroid tissue or by the loss of functional thyroid tissue. In adults, the initial onset of symptoms such as fatigue, constipation, intolerance to cold, muscle cramps and menorrhagia, and sluggishness are barely noticeable. As the metabolism continues to slow, however, symptoms include mental clouding, diminished appetite, and weight gain.
3. Gallstones are the leading cause of acute pancreatitis. Alcohol abuse also is a leading cause of the disease.
4. The main symptom of acute pancreatitis is the sudden onset of persistent, mild to severe abdominal pain centered over the epigastric region that may radiate to the back and, occasionally, the chest.
5. Alcohol abuse is thought to be the leading cause of chronic pancreatitis.
6. Insulin is needed after carbohydrate ingestion to transfer glucose from the blood into the skeletal and cardiac muscles. It also promotes glucose storage in the muscles and liver in the form of glycogen. If little or no insulin is secreted by the pancreas, glucose bypasses the body cells and rises to abnormally high levels in the blood. The excess glucose is excreted in the urine, drawing with it large amounts of water and electrolytes and leading to weakness, fatigue, malaise, and increased thirst.
7. When glucose cannot enter the cells, the cells shift from carbohydrate metabolism to fat metabolism for energy, resulting in dehydration and ketoacidosis, which can depress cerebral function. Acetone, formed as a by-product of fat metabolism, is volatile and blown off during expiration, giving the breath a sweet or fruity odor.
8. There are four types of DM:
    - Type 1 (insulin-dependent) DM has an onset before age 30 years in people who are not obese.

- Type 2 (non–insulin-dependent) DM is the most common form. It is highly associated with a family history of diabetes, older age, obesity, and lack of exercise.
- Gestational DM occurs when a pregnant woman cannot produce enough insulin to handle the higher BG level because of hormones secreted during the latter half of pregnancy. The condition usually resolves after delivery, but it places the woman at risk for developing type 2 DM later in life.
- Other specific types of diabetes include individuals with genetic defects of β-cell function or with defects of insulin action and persons with pancreatic disease, hormonal disease, and drug or chemical exposure.

9. Chronic diabetes can lead to atherosclerosis and coronary heart disease, kidney failure, blindness, and impaired neural function, whereby the individual may become unaware of injuries or infections of the hands and feet.
10. Severe hypoglycemia can lead to insulin shock. The signs and symptoms include a rapid onset with dizziness; headache; intense hunger; aggressive behavior; pale, cold, and clammy skin; profuse perspiration; salivation; drooling; and tingling in the face, tongue, and lips.
11. An individual progresses into a diabetic coma (i.e., hyperglycemia) over a long period of time. Common symptoms include dry mouth, intense thirst, abdominal pain, confusion, and fever. Severe signs include deep respirations; rapid, weak pulse; dry, red, warm skin; and a sweet, fruity acetone breath.
12. Because it may be difficult to determine which condition is present, a fast-acting carbohydrate should be given to the individual. If the individual is in insulin shock, recovery usually is rapid. If recovery does not occur, the emergency plan should be activated.
13. An individual with diabetes should have a consistent daily intake of

carbohydrates at each meal and snack to minimize BG fluctuations. A preactivity meal should be eaten 1 to 3 hours before activity, and 10 to 15 g of carbohydrates should be ingested 30 minutes before moderate activity or 20 to 30 g before vigorous activity. In addition, 15 to 30 g of carbohydrates should be ingested every 30 minutes during activity.

14. Aerobic, low-resistance exercise is recommended for the individual with diabetes. The program should be established under the guidance of a supervising physician.

APPLICATION QUESTIONS

1. As an athletic trainer at an National Collegiate Athletic Association (NCAA) Division I University that fields 26 athletic teams, you can expect that several student athletes will have type 1 diabetes. In an effort to ensure effective care of these individuals, a management plan should be initiated as part of the preparticipation examination (PPE). Within the medical history component of a PPE, what questions might you include as part of a supplemental questionnaire to identify individuals with type 1 diabetes?
2. Travel demands associated with away contests may be more demanding for the collegiate athlete than the high school athlete due to a higher frequency of longer distances of travel, overnight, or weekend trips. As the athletic trainer for a NCAA Division II women's basketball team, you are aware that one of the team members has type 1 diabetes. Specific to events that involve traveling both prior to and during travel, what strategies might you employ to ensure effective care for that student athlete?
3. Twenty minutes after the start of soccer practice, a 16-year-old male with type 1 diabetes suddenly feels lethargic and faint. It is not readily apparent whether the individual is experiencing a diabetic coma or insulin shock. How would you manage this condition?

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