This chapter will give you a fundamental understanding of endocrine and metabolic disorders. You will learn to integrate your knowledge of anatomy, physiology, and pathophysiology with the Advanced Medical Life Support (AMLS) assessment pathway in order to formulate differential diagnoses for life-threatening, critical/emergent, and nonemergent conditions. You will also learn how to implement and adapt management strategies for a variety of endocrine and metabolic disorders in prehospital and hospital settings.

LEARNING OBJECTIVES
At the conclusion of this chapter, you will be able to:

- Describe the anatomy, physiology, and pathophysiology of common endocrine disorders.
- Outline primary, secondary, and ongoing assessment strategies for the patient with an endocrine disorder using the AMLS assessment pathway.
- Identify the cardinal presentations/chief complaints of a broad range of endocrine disorders.
- List and be able to recognize the signs and symptoms of acid–base imbalances, electrolyte derangements, and endocrine disorders.
- Formulate provisional diagnoses on the basis of assessment findings for a variety of endocrine disorders.
- List the causes, diagnostic techniques, and treatment strategies for diseases of glucose metabolism and thyroid, parathyroid, and adrenal disorders.
- Use clinical reasoning skills to formulate and refine a differential diagnosis on the basis of a systematic, thorough secondary survey of a patient with an endocrine disorder.
- Implement effective treatment plans consistent with your assessment findings, and determine whether to continue the treatment on the basis of your ongoing assessment.
- Describe the pathophysiologic processes responsible for electrolyte and acid–base derangements, explain their causes, and discuss common modalities used to treat them.
- Compare and contrast normal and abnormal electrocardiogram (ECG) findings in the patient with an electrolyte derangement.
You are caring for a 58-year-old woman who tells you she has severe fatigue and weakness. She has a history of type 2 diabetes, polymyalgia rheumatica, hypertension, and heart failure. Her medications include metformin, prednisone, lisinopril, furosemide, and digoxin. Her vital signs include a blood pressure of 88/52 mm Hg; pulse rate, 58 beats/min; and respirations, 20 breaths/min.

- Which differential diagnoses are you considering based on the information you have now?
- Which additional information will you need to narrow your differential diagnosis?
- What are your initial treatment priorities as you continue your patient care?

The endocrine system regulates metabolic processes of the body. The primary functions of the endocrine glands include the following:

- Regulating metabolism
- Regulating reproduction
- Controlling the balance of extracellular fluid and electrolytes (sodium, potassium, calcium, and phosphates)
- Maintaining an optimal internal environment such as regulation of blood glucose levels
- Stimulating growth and development during childhood and adolescence

Performing an assessment of a patient’s endocrine system is challenging because the locations of the majority of these glands (with the exceptions of the thyroid gland and testes) make it impossible to inspect, palpate, percuss, or auscultate. It is also difficult to assess this system because of the different effects the hormones have on various systems throughout the body. Assessment of endocrine function depends on gathering data and recognizing the underlying pattern of an endocrine disorder.

**Anatomy and Physiology**

Glands are organs that manufacture and secrete chemical substances. Glands may be endocrine or exocrine. Exocrine glands secrete chemicals to the outer surface of the body (i.e., sweat and tears) or into a body cavity (i.e., saliva and pancreatic digestive enzymes). Endocrine glands secrete chemical hormones into the bloodstream. These chemicals travel to and act upon various tissues, where they signal and affect target cells that have appropriate receptors. They then act on these cells to cause a specific cell function. The network of endocrine glands that secrete hormones throughout the body is collectively referred to as the endocrine system.
Figure 7-1 The endocrine system uses the various glands within the system to deliver chemical messages to organ systems throughout the body.

Figure 7-2 The pituitary gland secretes hormones from its two regions: the anterior pituitary lobe and the posterior pituitary lobe.
Glucose Metabolism and Control

Glucose is a vital fuel for key metabolic processes in organs, especially those controlled by the central nervous system (CNS). The CNS is particularly dependent on glucose metabolism and relatively intolerant to changes in blood glucose levels. This explains why, for example, acute episodes of hypoglycemia are manifested as mental status changes, and persistent episodes of hypoglycemia can lead to irreversible brain damage.

Cellular survival depends on maintaining a balanced serum glucose concentration. Under normal circumstances, the body is able to maintain serum glucose in the relatively tight range of 70 to 150 mg/dL (3.9 to 8.3 mmol/L) before and after meals. This control is achieved by a complex interaction of hormones, neural (autonomic) and humoral (circulating or hormonal) factors, and regulatory mediators. Glucose levels are controlled by a feedback process, with changes in glucose concentration stimulating insulin or glucagon release. The main sources of glucose are glycogenolysis (breakdown of liver glycogen), gluconeogenesis (production of new glucose), and GI absorption.

Glycogenolysis
- glucose produced as glycogen breakdown occurs in the liver

GI absorption
- direct intestinal absorption of glucose through the intestine

Gluconeogenesis: the formation of new glucose from precursors including pyruvate, glycerol, lactate, and amino acids

A complex interaction of hormones, neural (autonomic) and humoral (circulating or hormonal) factors, and regulatory mediators ensures maintenance of a normal serum glucose concentration. When the level of glucose in the blood is insufficient, glucagon is released from alpha cells in the pancreas to increase glucose production through gluconeogenesis. Glucagon release can also be triggered by exercise, trauma, and infection. These mechanisms increase glucose levels within minutes but do so only transiently. Epinephrine and norepinephrine increase glucose levels even more rapidly by enabling gluconeogenesis and hepatic glycogenolysis. Insulin, which is secreted by the pancreatic beta cells, is essential for efficient cellular glucose utilization and also drives glucose into the cells.
Level of Consciousness
A patient who is experiencing an endocrine emergency will often be in serious distress. The patient's position may give you an indication of the severity of the condition. The patient who is unresponsive is in a critical state and may be experiencing an endocrine crisis such as hypoglycemia or hyperglycemia.

Airway and Breathing
Patients with endocrine emergencies may present with a variety of breathing levels. You should immediately assess the patient's effort of breathing. Increased work of breathing, abnormal respiratory rate, and hypoxia may all be indications that oxygen administration is necessary, such as with a nasal cannula or nonrebreathing mask. Be alert for abnormal respiratory patterns such as Kussmaul's respirations, which are often present in patients experiencing a diabetic ketoacidosis event. It is one of the body's compensatory mechanisms to "blow off" excess acid that is produced in this condition by increasing both respiratory rate and volume (a respiratory compensation to a metabolic acidosis).

Circulation/Perfusion
Assess the patient's skin color, moisture, and temperature, and obtain the patient's blood pressure. A patient with pale, cool, moist skin may be in shock or have hypoglycemia, whereas a patient with hot, dry skin may have a fever or hyperglycemia. A patient in hypoglycemic crisis will have a rapid, weak pulse. Because endocrine emergencies may affect the body's compensating systems, IV administration or blood component replenishment may be necessary. As always, follow your local protocols.

▼ First Impression
The difficult part of assessing patients with endocrine emergencies is that their problems tend to affect many organ systems and the seriousness of their presentations varies greatly. Many of the patients will have had their conditions for some time and may already be receiving treatment from their primary doctor or a specialist. These patients or their family members will likely share with you that there is a history of an endocrine problem. This information, along with the common signs and symptoms associated with each endocrine emergency described in this chapter, should help you determine possible causes of the current problem and generate an initial differential diagnosis.

▼ Detailed Assessment
History Taking
Collecting a complete history is critical to identifying endocrine emergencies. In diabetic emergencies in particular, the family history can give you pertinent information. Learning that other family members have a history of diabetes is a major clue and will help you in your differential diagnosis.

OPQRST and SAMPLER
The OPQRST and SAMPLER mnemonics should be used to obtain the patient's complete history using a systematic approach. Look for any signs that may assist you in confirming the patient's reported symptoms. Additional symptoms that may occur include polyphagia, polyuria, and polydipsia in patients with undiagnosed or poorly managed diabetes. Tachycardias, premature ventricular contractions (PVCs), premature atrial contractions (PACs), and other atrial dysrhythmias may all occur with hyperthyroidism and thyrotoxicosis.

It is possible that your patient's condition has been diagnosed prior to your arrival. In that case, the patient may have a significant amount of health-related information to provide you. Document all medications the patient is currently taking and whether the patient has been compliant with the regimen. Medications often provide another clue to the patient's condition.

Ask females of childbearing age about their last menstrual period because some patients with hypothyroidism may have a history of light or absent periods. A history of gestational diabetes is also important as it increases the risk of diabetes developing in a woman following her pregnancy.

Secondary Survey
Start your examination by noting the patient's appearance and the position in which he or she is found. Seizure activity and decorticate or decerebrate posturing should also be noted. If present, both are signs of serious illness.

Your physical examination should be geared toward identifying as many atypical findings as possible. Unless the patient had an endocrine emergency that caused some sort of trauma, a focused trauma assessment is usually not necessary. As always, a full-body exam should be completed after any life threats are managed.

When you check the patient's vital signs, look for the combination of hypertension and bradycardia, which suggests increased intracranial pressure.

Your secondary survey will reveal finer abnormalities that will help you determine your treatment. For example, if the patient's skin is cold and clammy, this may signal shock or severe hypoglycemia, as from an insulin reaction and the body's response to catecholamine release. Cold, dry skin may indicate an overdose of sedative drugs or alcohol intoxication. Hot, dry skin suggests hyperglycemia, fever, or possibly heat stroke. The goals of the secondary survey are twofold. First, you want to determine your patient's level of consciousness with precision so that later assessments can readily reveal whether the patient's condition is improving or deteriorating. Second, if your patient is in a coma, you need to look for clues to determine the cause.
Diagnostics

In patients with diabetes, obtain blood specimens early on, because any administration of prehospital dextrose or other medications will significantly change the chemical makeup of subsequent blood samples. An IV with 0.9% normal saline should be administered to patients with an altered mental status. Immediately assess the patient’s blood glucose level and begin treatment if the reading is less than 60 mg/dL and the patient has a change in mental status. Give 12.5 g to 25 g of dextrose; this dose will reverse most cases of hypoglycemia.

Refine the Differential Diagnosis

The components of the primary and secondary survey will help you refine your differential diagnosis and determine the severity of the patient’s condition. Manage any life threats as they appear during the assessment process. Remember that most diseases or conditions, including endocrine disorders, are caused by more than one factor. The specific conditions described later will provide an approach for helping you determine the differential diagnosis and recognize key findings.

Ongoing Management

Remember than an important aspect of patient care is to address the patient’s emotional needs. Endocrine disorders can be stressful conditions for patients to manage. Always be empathetic and responsive to the patient’s needs. Recheck vital signs and level of consciousness frequently in unstable patients and at least every 15 minutes in stable patients. Every patient should have at least two sets of vital signs documented.

Parathyroid, Thyroid, and Adrenal Gland Disorders

Hypoparathyroidism

Hypoparathyroidism is a rare condition characterized by low serum levels of PTH or resistance to its action. Congenital, autoimmune, and acquired diseases are among its many causes. Regardless of etiology, the hallmark of the condition is hypocalcemia, which will be discussed later.

Pathophysiology

The most common cause of acquired hypoparathyroidism is iatrogenic damage or inadvertent removal of the glands during thyroidectomy. Damage (during neck dissection, for example) can be transitory or permanent.

Signs and Symptoms

Patients with acute hypoparathyroidism report muscle spasm, paresthesia, and tetany. The patient may even have seizures. These signs and symptoms are directly due to hypocalcemia.

Differential Diagnosis

In the prehospital setting, no laboratory studies are immediately available to confirm hypoparathyroidism, so you must have a high index of suspicion on the basis of history and physical examination findings. Recent anterior neck surgery is a risk factor for iatrogenic hypoparathyroidism.

You should be familiar with Trousseau’s sign (Figure 7-3) and Chvostek’s sign (Figure 7-4), both of which will help you detect muscular irritability caused by hypocalcemia. To obtain a positive Trousseau’s sign, place a blood pressure cuff around the arm, inflate it 30 mm Hg above systolic blood pressure, and hold...
it in place for 3 minutes. This will induce spasm of the muscles of the hand and forearm. The wrist and metacarpophalangeal joints flex, the distal and proximal interphalangeal joints extend, and the fingers adduct. You can elicit a positive Chvostek's sign by tapping the facial nerve against the mandibular bone just anterior to the ear, which produces a spasm of the facial muscles. This sign, however, is not as sensitive as Trousseau's sign.

Another tool available is the electrocardiogram (ECG). In patients with hypocalcemia, the QT interval is prolonged (Figure 7-5).

**Treatment**

As in any emergent condition, you must assess and stabilize the patient’s airway, breathing/ventilation, and hemodynamic status. Obtain intravenous (IV) access and provide supportive treatment. If the patient is having seizures, administer benzodiazepines. When you have a strong clinical suspicion or laboratory analysis confirms hypocalcemia, the patient may be given IV calcium. In emergent situations, administer calcium chloride or calcium gluconate, 0.5- to 1-g IV bolus. In nonemergent situations, administer 100 to 300 mg of calcium diluted in 150 mL of 5% dextrose in water (D5W) solution over 10 minutes.

**Hyperthyroidism**

Hyperactivity of the thyroid gland, or hyperthyroidism, is a common ailment that results in a hypermetabolic state called thyrotoxicosis. Contrast this with thyroid storm, which is a more rare complication of hyperthyroidism that occurs in only 1% to 2% of patients, but it is a life-threatening condition characterized by hemodynamic instability, altered mental status, gastrointestinal (GI) dysfunction, and fever.

**Pathophysiology**

Graves’ disease, also known as diffuse toxic goiter, is the most common form of hyperthyroidism. It is an autoimmune disorder in which antibodies that mimic the role of TSH produce an increase in secretion of thyroid hormones. This condition most often occurs in women of middle age, but it can occur at any age and can also affect men. Other causes of hyperthyroidism include acute intoxication with exogenous thyroid hormones, and (less commonly) as a result of drugs with high iodine loads such as amiodarone or iodinated IV contrast, which may precipitate sudden release of excess thyroid hormones in susceptible individuals. In cases of autoimmune destruction of the gland, there may be a temporary hyperthyroidism preceding the more chronic hypothyroidism.

Thyroid storm occurs when the body is stressed by a diabetic emergency, an adverse drug reaction, or some serious challenge. You should suspect thyroid storm if the patient experienced cardiac decompensation after taking amiodarone, an antiarrhythmic agent rich in iodine. Other triggers of thyroid storm are summarized in Table 7-1.

**Signs and Symptoms**

The characteristic clinical presentation of a patient with hyperthyroidism includes apprehension, agitation, edginess, heart

**Figure 7-5** Hypocalcemia prolongs the QT interval by stretching out the ST segment. Hypercalcemia decreases the QT interval by shortening the ST segment so that the T wave seems to take off directly from the end of the QRS complex.

palpitations, and weight loss of as much as 40 pounds over a few months. Heat intolerance and increased sweating caused by this hypermetabolic state are frequent symptoms.

A complete physical exam will reveal signs and symptoms of thyrotoxicosis, including the exophthalmos that is characteristic of the condition (Figure 7-6). Other signs and symptoms of hyperthyroidism include the following:

- Shortness of breath
- Disorientation
- Abdominal pain
- Diarrhea
- Chest pain
- Enlarged thyroid gland (palpable goiter)
- High-output cardiac failure
- Fever
- Drug interactions
- Altered mental status
- Jaundice
- Weakness

Apathetic thyrotoxicosis is a rare form of thyrotoxicosis seen only in older adults. In this condition, the characteristic symptoms of hyperthyroidism are absent. The patient is lethargic, has an apathetic affect, develops a goiter, and experiences weight loss.

**Differential Diagnosis**

In the prehospital setting, no laboratory studies are immediately available to confirm hyperthyroidism or thyroid storm, so you must have a high index of suspicion on the basis of history and physical examination findings. You can begin to stabilize the patient and initiate early treatment on the strength of clinical judgment alone.

In the hospital, the most rapid and useful test for hyperthyroidism is TSH serum level; if it is low and the patient has clinical signs and symptoms of hyperthyroidism, the test is essentially diagnostic. To confirm this presumptive diagnosis, the levels of the actual thyroid hormones, usually $T_4$ and $T_3$, may be obtained. Imaging studies and biopsy can help determine the specific cause of the disorder.

As part of the differential diagnosis, consider stroke, diabetic emergencies, congestive heart failure (CHF), toxic ingestion (particularly ingestion of a sympathomimetic agent), and sepsis.

**Treatment**

The key to providing optimal patient care is to differentiate among the various hypermetabolic states induced by thyroid disorders. These include subacute (chronic) hyperthyroidism, acute severe hyperthyroidism, and its most critical complication, thyroid storm. When you are caring for a patient with chronic hyperthyroidism, the patient generally requires only supportive care and early management of symptoms. If severe hyperthyroidism or thyroid storm is detected, it is imperative to stabilize the patient’s condition. As in any acute emergency, begin with the ABCs. Be alert to the fact that patients experiencing acute severe

<table>
<thead>
<tr>
<th>Medical Triggers</th>
<th>Endocrine Triggers</th>
<th>Pharmacologic Triggers</th>
</tr>
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<tbody>
<tr>
<td>Infectious disease</td>
<td>Hypoglycemia</td>
<td>Iodine therapy</td>
</tr>
<tr>
<td>Cardiac ischemia</td>
<td>Diabetic ketoacidosis</td>
<td>Amiodarone ingestion</td>
</tr>
<tr>
<td>Serious burns</td>
<td>Nonketotic hyperosmolar state</td>
<td>Administration of contrast medium</td>
</tr>
<tr>
<td>Thromboembolism</td>
<td></td>
<td>Drug interactions</td>
</tr>
<tr>
<td>Major surgery</td>
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<tr>
<td>Trauma</td>
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**Table 7-1 Triggers of Thyroid Storm**

Figure 7-6 Person with hyperthyroidism (A and B). Wide-eyed, staring gaze caused by overactivity of the sympathetic nervous system is one feature of this disorder. The accumulation of loose connective tissue behind the eyeballs also adds to the protuberant appearance of the eyes.

A © Science Photo Library; B © SPL/Custom Medical Stock Photo, Inc
hyperthyroidism or thyroid storm may exhibit altered mental status progressing to coma.

The patient with thyroid storm often has moderate to severe dehydration because of excessive diarrhea and sweating. For aggressive hydration, establish two peripheral IV lines early in the course of treatment. Although aggressive hydration is indicated, care must be taken to avoid inducing acute pulmonary edema, as these patients may experience cardiac instability.

A patient with hyperthyroidism is prone to arrhythmias such as sinus tachycardia, atrial fibrillation, atrial flutter, and premature ventricular contractions. For this reason, you should begin continuous cardiac monitoring as soon as you suspect this diagnosis.

Patients with thyroid storm may have fever associated with the pathophysiologic process itself or with an infection that precipitated the thyroid storm. Assess body temperature and treat hyperpyrexia in thyroid storm with acetaminophen. Do not use aspirin because it is associated with decreased protein binding of thyroid hormones and correspondingly increased levels of unbound or free T3 and T4, which will exacerbate symptoms.

The goals of pharmacologic treatment in the prehospital setting are to block the peripheral adrenergic hyperactivity the thyroid hormones elicit (tachycardia, fever, anxiety, and tremors) and to inhibit the conversion of T4 to T3 in peripheral tissues. Both objectives can be achieved by administering beta-blockers. The drug of choice is propranolol, given 1 mg IV every 10 minutes up to a total of 10 mg IV or until symptoms have resolved. Propranolol is contraindicated in patients with bronchial asthma, chronic obstructive pulmonary disease (COPD), atrioventricular blocks, hypersensitivity, and severe heart failure. A patient with thyroid storm and concomitant heart failure most likely has a high-output cardiac failure. This is not considered a contraindication to the use of propranolol unless he or she also has significant cardiomyopathy with systolic dysfunction. Adjunctive corticosteroid therapy—either hydrocortisone, 100 mg IV, or dexamethasone, 10 mg IV—may be given to slow the conversion of T4 to T3.

### Hypothyroidism

Hypothyroidism is an endocrine dysfunction characterized by decreased or absent secretion of thyroid hormones. Its incidence in the United States is 4.6% to 5.8%, but half of those with the condition are asymptomatic. Hypothyroidism is most common among white adult women between the ages of 40 and 50. It is highly associated with autoimmune conditions.

#### Pathophysiology

Defective thyroid hormone secretion is classified as either primary or secondary hypothyroidism. The causes of each are summarized in Table 7-2. Primary hypothyroidism involves direct thyroid injury caused by an autoimmune disorder or an adverse drug reaction. Patients who have had thyroidectomy or radioablation therapy (using radiation to decrease the amount of functional glandular tissue) for a hyperthyroid state may have resultant hypothyroidism. In secondary hypothyroidism, damage to the hypothalamus or pituitary gland results in decreased stimulation of the thyroid gland (specifically, a decline in the production and release of TSH). Many complications result from clinical hypothyroidism, including hypoxia, hypothermia, hypoglycemia, sepsis, and narcosis.

### Signs and Symptoms

Hypothyroidism has a deleterious effect on many body systems, including the integumentary, metabolic, nervous, and cardiovascular systems. The skin of a patient with this condition is cool, dry, and yellow. The patient typically has thinning of the eyebrows, coarse hair and skin, marked intolerance to cold temperatures, and neurologic changes such as altered mental status, ataxia, and delayed relaxation of deep tendon reflexes. When hypothyroidism becomes chronic and extreme, it may evolve into a life-threatening condition called **myxedema coma** (Figure 7-7), characterized by hypotension, bradycardia, hypoglycemia, and low serum sodium (hyponatremia). Precipitants of myxedema coma include the following:

- Lung infection
- Cold exposure
- Heart failure
- Stroke
- Gastrointestinal bleeding
- Trauma
- Stress
- Hypoxia
- Electrolytic disturbances
- Low serum glucose levels

#### Table 7-2 Causes of Hypothyroidism

<table>
<thead>
<tr>
<th>Primary</th>
<th>Secondary</th>
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<tr>
<td>Autoimmune hypothyroidism</td>
<td>Sarcoid infiltration</td>
</tr>
<tr>
<td>Hereditary hypothyroidism</td>
<td>Pituitary mass</td>
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<tr>
<td>Radiation therapy</td>
<td></td>
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<tr>
<td>Iodine deficiency</td>
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<tr>
<td>Use of lithium</td>
<td></td>
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<tr>
<td>Use of antithyroid medications</td>
<td></td>
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<tr>
<td>Idiopathic</td>
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The hypothyroid patient is prone to experiencing heart arrhythmias, especially bradycardia, so begin continuous cardiac monitoring as soon as possible. Be aware, however, that standard treatments for bradycardia may be ineffective until thyroid hormone has been replaced.

A patient suffering from myxedema coma may be hypothermic as a result of the pathophysiological process itself or because of an infection. Always assess body temperature, and treat hypothermia with blankets and other warming techniques. Rapidly transport the patient to a well-equipped hospital facility for definitive treatment, which may include L-triiodothyronine, 0.25 mcg IV; hydrocortisone, 100 mg IV every 8 hours; and subsequent daily oral replacement therapy if the condition proves irreversible.

### Chronic Adrenal Insufficiency

Adrenal insufficiency, the failure of the adrenal cortex to produce a sufficient amount of cortisol, is classified as primary, secondary, or tertiary, depending on whether the cortex is damaged directly or indirectly. Primary adrenal insufficiency, known as Addison’s disease, is a metabolic and endocrine ailment caused by a direct insult to, or malfunction of, the adrenal cortex. It is a chronic disease with a protracted onset. Almost any condition that directly harms the adrenal cortex can cause primary adrenal insufficiency, including autoimmune disorders, adrenal hemorrhage, and infectious diseases such as acquired immunodeficiency syndrome (AIDS), tuberculosis, and meningococcemia.

### Pathophysiology

As noted previously, the adrenal cortex produces the corticosteroid hormones aldosterone and cortisol. Aldosterone is responsible for keeping serum levels of sodium and potassium in balance. When the body experiences any stress—trauma, infection, cardiac ischemia, or a severe illness, to name a few—the adrenal glands may become unable to produce sufficient amounts of corticosteroid hormones to supply the body’s demands, triggering an acute exacerbation of Addison’s disease.

In secondary adrenal insufficiency, although the cortex itself is intact, it fails to receive a signal to produce cortisol because the pituitary gland fails to release adrenocorticotropic hormone (ACTH), which normally stimulates the adrenal cortex—thus the adrenal insufficiency is one step removed. Tertiary (third-level) adrenal insufficiency, in which the pituitary’s failure to release ACTH stems from a disorder of the hypothalamus, is even less direct.

In primary adrenal insufficiency, patients may develop hyperpigmentation of the skin due to overproduction of melanocyte-stimulating hormone (MSH). This overproduction results from the fact that MSH and ACTH are produced from the same precursor protein (pro-opiomelanocortin) in the pituitary. MSH stimulates melanocytes in the skin to produce the skin pigment melanin.

Secondary and tertiary adrenal insufficiency are not associated with hyperpigmentation of the skin because they involve low levels of MSH rather than high levels.
Signs and Symptoms
The clinical presentation of a patient with Addison’s disease is consistent with the endocrine and electrolyte disorders brought on by the disease. The patient will have chronic fatigue and weakness, loss of appetite and consequent weight loss, and hyperpigmentation of the skin and mucous membranes due to the excess MSH production (Figure 7-8). The patient will have electrolyte disturbances associated with hyponatremia, hyperkalemia, and hypotension and might also have GI disturbances such as abdominal pain, nausea, vomiting, and diarrhea.

Differential Diagnosis
Diagnostic tools for chronic adrenal insufficiency are not available in the prehospital setting. It’s important that you ask the patient for any old diagnostic laboratory reports that might be readily available. Past abnormal electrolyte findings that correlate with the patient’s current clinical presentation, such as metabolic acidosis, hyponatremia, hyperkalemia, and hypoglycemia, should raise a red flag. Definitive diagnosis of this condition is made by measuring the patient’s baseline serum cortisol level and then conducting stimulation testing, in which synthetic ACTH (called cosyntropin) is administered. If the cortisol level fails to rise shortly afterward, the patient can be diagnosed as having primary adrenal insufficiency.

Treatment
The prehospital management of an acute exacerbation of Addison’s disease, known as an “addisonian crisis,” is limited to supportive care. If the patient has tachycardia and hypotension, administer a 20-mL/kg fluid bolus of normal saline solution. Continual reevaluation of the patient’s hemodynamic state, early administration of hydrocortisone (100 to 300 mg IV, or as dictated by local EMS protocol and medical control orders) to supplement the failing adrenal function, and rapid transport to the emergency department (ED) are paramount in treating this condition. Provide correction of hypoglycemia, as well as symptomatic medical treatment of nausea and vomiting.

In the hospital, diagnostic testing will be carried out to identify electrolyte abnormalities such as hyponatremia and hyperkalemia. Elevated hematocrit levels are common. Management includes correction of electrolyte abnormalities, restoration of metabolic balance (e.g., by replacing glucocorticoids), and volume replacement for hypovolemia.

Acute Adrenal Insufficiency
Acute adrenal insufficiency is a condition in which the body’s need for glucocorticoids and mineralocorticoids exceeds the delivery of these hormones by the adrenal glands. The most common cause is abrupt discontinuation of pharmacologic steroid therapy after prolonged use. It can also occur when such a patient fails to receive an adjusted dosage during times of stress, such as during illness or after major surgery or trauma.

Pathophysiology
Like chronic adrenal insufficiency, acute insufficiency is classified as primary, secondary, or tertiary, depending on the dysfunctional endocrine gland. Primary adrenal insufficiency refers to dysfunction of the adrenal glands, secondary adrenal insufficiency refers to dysfunction of the pituitary gland, and tertiary insufficiency is linked to hypothalamic dysfunction.

Signs and Symptoms
The clinical picture of acute adrenal insufficiency will include nausea, vomiting, dehydration, abdominal pain, and weakness. Historical clues, such as tan skin on a patient who denies sun exposure, may indicate chronic adrenal insufficiency. Ask the patient about recent medication changes that may have precipitated the symptoms. When adrenal insufficiency is accompanied by hypotension, the condition is called adrenal crisis and constitutes a true life-threatening emergency.

Differential Diagnosis
The diagnosis of this condition in the prehospital setting can be challenging. The definitive confirmatory laboratory test is not available in the field, and the presentation of acute adrenal insufficiency can easily be confused with more common conditions such as GI disorders. As the EMS provider, you have to use your available tools to find indirect evidence of an adrenal disorder. Assess for hypoglycemia with a glucometer, and look for evidence of hyperkalemia on the ECG. Assess for signs and symptoms of other abnormalities such as anemia, hyponatremia, and metabolic acidosis.

Vital signs can also provide key clues. For example, hypotension that’s poorly responsive to administration of IV fluids is...
seen in adrenal crisis. Confirmation of the disorder can be performed in the ED, using the cosyntropin stimulation test.

**Treatment**

As with any life-threatening emergency, first evaluate the patient’s ability to maintain his or her airway, breathing, and circulation. For hypotension, immediate resuscitation with normal saline is warranted. Administer dextrose if hypoglycemia is present. Address a deficit of glucocorticoids with hydrocortisone, 100 to 300 mg IV, which must be ordered under medical direction in some systems. If a cosyntropin stimulation test is to be performed at a later time, dexamethasone, 4 mg IV, is preferable to hydrocortisone because hydrocortisone can create a false-positive test result. Rapidly transport the patient to the ED for definitive treatment.

**Hyperadrenalism**

Hyperadrenalism, or Cushing's syndrome, is the clinical condition caused by long-standing exposure to excessive circulating serum levels of glucocorticoids, particularly cortisol, as a result of overproduction in the adrenal cortex. It is more common in women, especially those aged 20 to 50. Cushing's syndrome can be brought on by an adrenal gland or pituitary tumor or by long-term corticosteroid use.

**Pathophysiology**

Regardless of the cause, excess cortisol causes characteristic changes in many body systems. Metabolism of carbohydrate, protein, and fat is disturbed, such that the blood glucose level rises. Protein synthesis is impaired so that body proteins are broken down, which leads to loss of muscle fibers and muscle weakness. Bones become weaker and more susceptible to fracture.

**Signs and Symptoms**

Patients with Cushing's syndrome have a distinct appearance characterized by obesity, a moon face (Figure 7-9), and other cardinal features. Signs and symptoms that tend to accompany this disorder include the following:

- Chronic weakness
- Increased body and facial hair
- Full, puffy face
- Fatty “buffalo hump” at the back of the neck
- Central body obesity
- Purple striae on the abdomen, buttocks, breasts, or arms
- Atrophied proximal muscles
- Thin, fragile skin
- Amenorrhea
- Decreased fertility or diminished sex drive
- Diabetes mellitus
- Hypertension

**Differential Diagnosis**

Definitive diagnostic testing for Cushing's syndrome is not available in the prehospital setting. Ask the patient for any old diagnostic laboratory reports that might be readily available as a part of recent discharge paperwork. Past abnormal electrolyte findings that correlate with the patient’s current clinical presentation, such as metabolic alkalosis, hypernatremia, hypokalemia, and hyperglycemia, should raise suspicion for the disease.

**Treatment**

Patients with Cushing's syndrome often have chronic or subacute symptoms. Management is guided by the clinical presentation. Affected patients may have fluid retention or, because of
the osmotic diuresis brought on by hyperglycemia, may be dehydrated. Thus fluid replacement should be dictated by volume status. Hypertension does not require specific therapy unless it has caused end-organ dysfunction or symptoms (e.g., CHF, cardiac ischemia, encephalopathy, acute renal failure). If such a condition is present, administer antihypertensive treatment. Monitor vital signs, mental status, and cardiac rhythm closely.

**Glucose Metabolic Disorders**

Metabolic emergencies represent true diagnostic and management challenges for basic life support (BLS) providers because so few diagnostic tools are available in the prehospital setting. Many metabolic conditions are associated with nonspecific symptoms, causing a potential delay in treatment. The following sections discuss some of the fundamental clinical principles to consider in order to promptly arrive at a diagnosis and begin appropriate treatment.

**Diabetes Mellitus**

Diabetes mellitus is the most common endocrine disorder and refers to a group of conditions characterized by hyperglycemia (high blood glucose levels) resulting from defects in insulin production, insulin action, or both. Glucose is a vital energy source for the body, but insulin is required for glucose to travel into the cell, where it can be used. Insulin acts like a key to unlock the cell membrane and allow the glucose to enter.

**Pathophysiology**

Clinically, diabetes manifests as a high level of blood glucose and unbalanced lipid and carbohydrate metabolism. Untreated diabetes results in hyperglycemia. A random plasma glucose level >200 mg/dL (>11.1 mmol/L) or a fasting serum glucose >140 mg/dL (>7.7 mmol/L) meets the threshold for a diagnosis of diabetes. The percentage of glycated hemoglobin (also called glycosylated hemoglobin or HbA1c) is often used as a measure of a patient’s diabetes control because the percentage correlates to the average blood glucose levels over a 3-month period. Chronically poor glucose control, and diabetic emergencies account for between 3% and 4% of EMS calls, most of which are for hyperglycemia, and 10% to 12% of which are for acute and chronic medical problems associated with hyperglycemia. Rarely, diabetic medications may be employed in intentional overdoses, underscoring the need for blood glucose measurement in all patients with altered mental status of unknown etiology. On the other end of the spectrum, uncomplicated hyperglycemia, diabetic ketoacidosis, and hyperosmolar hyperglycemic nonketotic coma (HHNC) can occur.

**Signs and Symptoms**

Current classification of diabetes is based upon the underlying pathologic process related to insulin production and insulin resistance. The three main categories are as follows:

- **Type 1 diabetes mellitus**: characterized by being unable to produce any insulin due to pancreatic β-cell destruction. This type of diabetes is typically diagnosed during childhood or adolescence and accounts for 5% to 10% of all cases of diabetes mellitus. Patients with type 1 diabetes usually require daily insulin administration to stay alive.
- **Type 2 diabetes mellitus**: characterized by progressive cellular insulin resistance and a gradual failure of pancreatic β-cell insulin production. Type 2 diabetes accounts for 90% to 95% of all diagnoses of diabetes, is most common among older adults, and is associated with physical inactivity and obesity. It is common for patients with type 2 diabetes to remain asymptomatic for years before they begin to show signs and symptoms. Type 2 diabetes is often treated initially with oral hypoglycemic medications but may eventually require insulin therapy to maintain adequate glucose control. There has been a significant rise in the number of pediatric patients who are being diagnosed with type 2 diabetes. Increasing rates of childhood obesity and decreasing levels of physical exercise appear to be contributing factors.
- **Gestational diabetes**: characterized by glucose intolerance that can occur among pregnant women. It typically has the same clinical presentation as type 2 diabetes. Patients usually have hyperglycemia but no acidosis. Gestational diabetes predisposes women to developing type 2 diabetes.

The classic clinical manifestations of diabetes mellitus are referred to as the three P’s: polyuria, polydipsia, and polyphagia. As the levels of glucose increase in the bloodstream, the kidneys’ ability to reabsorb glucose may be overwhelmed, causing a “spilling” of glucose in the urine and an osmotic diuresis. Normally glucose is not found in the urine, so the presence of any glucose in the urine is an abnormal finding. Weight loss, thirst, blurred vision, and fatigue may also be present.

**Differential Diagnosis**

Hyperglycemia secondary to other causes may include hormonal tumors, pharmacologic agents, liver disease, and muscle
disorders. Diagnostic procedures for diabetes are complex and include a thorough history, physical exam, urinalysis, and blood analysis.

**Treatment**

Using a glucometer to quantify serum glucose at the patient’s side has become a standard of care in modern EMS practice. In the past, dextrose was given empirically to all patients with altered mental status without first quantifying serum glucose. Later, researchers found that few patients benefited from such an approach. A glucometer gives rapid bedside glucose results, and its use in the prehospital setting has been found to be safe and accurate. Ideally, you should measure glucose using capillary blood samples, not venous blood obtained during placement of an IV line, because the latter can produce inaccurate readings. Many glucose strips are required to be stored in temperature-controlled, airtight compartments in the ambulance to ensure their accuracy and reliability.

**Hypoglycemia**

Hypoglycemia, a frequent complication of diabetes, is the most common endocrine emergency. **Hypoglycemia** is defined as a blood glucose level < 60 mg/dL (3.3 mmol/L). Keep in mind that individual responses to blood glucose levels vary, and the levels discussed here represent averages. Generally, as the plasma glucose level falls below 60 mg/dL (3.3 mmol/L), the following sequence of events occurs in quick succession:

- First, the body decreases insulin secretion in an effort to arrest the decline in blood glucose levels.
- Next, there is an increase in the secretion of counterregulatory hormones, primarily epinephrine and norepinephrine.
- Finally, signs and symptoms, including impaired cognition, become apparent. Once the glucose level falls below 50 mg/dL (2.8 mmol/L), significant mental status changes occur.

Untreated hypoglycemia is associated with significant morbidity and mortality. To decrease these risks, you should be able recognize the signs and symptoms and be prepared to initiate treatment quickly and effectively.

**Pathophysiology**

Hypoglycemia in persons with insulin-dependent diabetes often is the result of having taken too much insulin, too little food, or both. The tissues of the central nervous system (including the brain), unlike other tissues that can usually metabolize fat or protein in addition to sugar, depend entirely on glucose as their source of energy. If the level of glucose in the blood drops dramatically, the brain is literally starved. Triggers of hypoglycemia are given in Table 7-3.

<table>
<thead>
<tr>
<th>Table 7-3 Triggers of Hypoglycemia</th>
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<tbody>
<tr>
<td><strong>Decreased Food Intake</strong></td>
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<tr>
<td>Exogenous insulin administration</td>
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<tr>
<td>(factitious hypoglycemia)</td>
</tr>
<tr>
<td>Medications</td>
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<tr>
<td>▪ Oral hypoglycemic agents</td>
</tr>
<tr>
<td>▪ Beta-blockers</td>
</tr>
<tr>
<td>▪ Antimalarial drugs</td>
</tr>
<tr>
<td>Alcohol abuse</td>
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<tr>
<td>Aggressive treatment of hyperglycemia</td>
</tr>
<tr>
<td>▪ Diabetic ketoacidosis</td>
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<tr>
<td>▪ Nonketotic hyperosmolar state</td>
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<tr>
<td>▪ Uncontrolled blood glucose levels</td>
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<tr>
<td>▪ Administration of excessive doses of therapeutic insulin</td>
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<tr>
<td>Malnutrition</td>
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<tr>
<td>Medication adjustments</td>
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<tr>
<td>Insulin pump failure</td>
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<tr>
<td>Sepsis</td>
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</tbody>
</table>

Hypoglycemia in patients who have no history of diabetes is called fasting or postprandial hypoglycemia. Fasting hypoglycemia is usually the result of an imbalance between glucose utilization and production. Postprandial hypoglycemia is characterized by alimentary hyperinsulinism and is commonly seen in patients who have undergone gastric surgery. A number of conditions may elicit fasting hypoglycemia. Among the most common are severe liver disease, pancreatic tumors (such as insulinomas), enzyme defects, drug overdoses (for example, insulin, sulfonylureas), and severe infection. The clinical characteristics are similar to those of diabetic hypoglycemia.

**Signs and Symptoms**

Clinical manifestations of hypoglycemia usually evolve rapidly. The patient will seek treatment for a myriad of signs and symptoms related to the release of endogenous stress hormones, including diaphoresis, tachycardia, tremors, and pale, cold, clammy skin. If hypoglycemia is not treated, an altered mental status and generalized seizures may develop in the patient. It is important to check the serum glucose in every patient who is having an active seizure to rule out hypoglycemia. While the
Definition of hypoglycemia is a blood glucose level < 70 mg/dL, the absolute level at which signs and symptoms appear may be altered by the patient's medical history, age, sex, and overall health. For example, an older adult with a complex medical history may have signs of severe hypoglycemia at a glucose level above 50 mg/dL (> 2.8 mmol/L). A young adult, however, may have signs of severe hypoglycemia at a level well below 50 mg/dL (< 2.8 mmol/L).

Most clinical manifestations of hypoglycemia are generated by secretion of counterregulatory hormones (e.g., epinephrine), which are secreted in response to a low glucose concentration. Signs and symptoms may include the following:

- Sweating
- Tremors
- Nervousness
- Tachycardia
- Altered level of consciousness (LOC) or behavior
- Seizures
- Coma

You must consider that the patient may be taking a medication, such as a beta-blocker, whose effects initially mask signs of hypoglycemia. Such patients can rapidly lose consciousness or begin having a seizure in the absence of any early symptoms of hypoglycemia.

Differential Diagnosis

Differential diagnoses may include Addison's disease, anxiety disorders, cardiogenic shock, adrenal crisis, and insulin resistance. A comprehensive history and physical exam that is suspicious for hypoglycemia can be confirmed with a serum glucose test. As part of the initial evaluation, obtain a complete set of vital signs.

Treatment

Pharmaceutical management of diabetes emphasizes tight plasma glucose control, which means getting the blood glucose level as close to normal (nondiabetic) as you safely can, using subcutaneous insulin injections, oral antihyperglycemics, or a combination of both. This tight control helps to decrease the risks for long-term complications such as renal failure and heart disease. However, patients on such medications are at increased risk of hypoglycemic episodes. To prevent further complications, such as seizures or permanent brain damage, begin providing glucose immediately when symptomatically hypoglycemic. The simplest option is to give oral glucose in the form of a small snack, a sugar-containing beverage, or a sugar gel. This option should always be considered in awake and alert patients who are able to swallow. For patients who have altered mentation or cannot safely swallow due to risk of aspiration, administration of 50 mL of D$_5$W has been the standard, but such a high concentration of glucose can have serious complications if extravasation or infiltration occurs. Recently, EMS has begun to embrace the use of D$_{10}$W. Studies have found no difference in the amount of time necessary for a hypoglycemic patient to regain consciousness when the two solutions are compared. When patients are given D$_{10}$W, they may receive a considerably smaller amount of glucose while achieving the same therapeutic response and are less likely to have a high glucose level after treatment.

If quick IV access proves difficult, intramuscular (IM) administration of glucagon can be an effective alternative. Glucagon may not work, however, in patients with chronic illness who have depleted glycogen stores (e.g., those with alcoholism and chronic liver disease). Recovery time with glucagon is significantly longer than with IV dextrose, and glucagon may cause side effects such as nausea and vomiting. If it is used, the standard dose is 1 to 2 mg IM.

Management of hypoglycemia in nondiabetic patients is similar to management of the condition in patients with diabetes. However, hypoglycemia in nondiabetic patients may recur, especially in patients with drug overdose. Such patients may require more than one dose of dextrose or even a continuous infusion.

Patients may wish to refuse transportation to the hospital after successful treatment of hypoglycemia by EMS. While there is some literature supporting this practice, extreme care must be taken when the patient is taking any long-acting antihyperglycemic medications (insulin or oral medications) due to the risk of recurrent hypoglycemia. Additionally, a careful search for the cause of the hypoglycemic episode should be undertaken. Some patients may have an obvious cause such as change in medication regimen or lack of oral intake that can be addressed. Unexplained episodes of hypoglycemia may be the first manifestation of other conditions that are increasing the body's metabolic needs (i.e., infection, trauma).

Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA) is characterized by a plasma glucose concentration > 350 mg/dL (> 19.4 mmol/L), ketone production, a serum bicarbonate level < 15 mEq/L, and anion gap metabolic acidosis. The mortality rate for DKA ranges from 9% to 14%. DKA is an acute endocrine emergency in which insulin deficiency and an excessive glucagon level combine to create a hyperglycemic, acidic, volume-depleted state. The condition is often associated with electrolyte imbalances.

Pathophysiology

DKA may be elicited by certain metabolic stressors such as infection, myocardial infarction, trauma, and sometimes pregnancy. The common trigger among these conditions is often the interruption of the insulin regimen of a person with diabetes. Lack of insulin prevents glucose from entering cells, and consequently, the cells become starved of glucose for cellular metabolism and turn to other sources of energy such as fat. As a result, glucose begins to accumulate in the bloodstream.
Overflow of glucose into the renal tubules draws water, sodium, potassium, magnesium, and other ions into the urine, creating a significant osmotic diuresis. This diuresis, combined with vomiting, produces volume depletion, electrolyte imbalances, and, consequently, shock. These osmotic changes are largely responsible for the declining mental status of a patient with DKA and are particularly dangerous in children. The clinical hallmark of DKA is metabolic acidosis, which is discussed later. Physiologically, the body attempts to compensate and eliminate acids by breathing faster and deeper (Kussmaul's respiration) and trying to use more bicarbonate. Acidosis encourages the shift of potassium into the bloodstream, where it is lost in the osmotic diuresis occurring in the kidneys. This process results in a pseudohyperkalemia or initially high blood level that rapidly changes to hypokalemia with the treatment of DKA.

Signs and Symptoms
Patients with DKA are dehydrated and will appear ill. They usually report polydipsia, polyphagia, and polyuria. Patients with severe DKA will exhibit altered mental status during the initial examination. Tachycardia, rapid breathing, and orthostatic changes are likely to be present. Additionally, 
\[\text{ETCO}_2\] will be low, reflecting the metabolic acidosis present in DKA. The signs and symptoms include the following:

- Nausea and vomiting
- Abdominal pain (especially common in children)
- Tachypnea/hyperpnea
- Fruity breath odor
- Fatigue and weakness
- Increased diuresis
- Altered LOC
- Orthostatic hypotension
- Cardiac dysrhythmia
- Seizures
- Hemodynamic shock in severe cases

Differential Diagnosis
Several conditions bear a clinical resemblance to DKA and distinguishing among them may be difficult in the field without the diagnostic testing used in hospitals. Conditions that produce acidosis like sepsis—for example—may mimic DKA. Prolonged fasting in a third-trimester pregnant patient or nursing mother who is not eating properly can also resemble DKA. People who abuse alcohol may have a fruity breath odor and a rapid respiratory rate due to alcoholic ketoacidosis. Remember that rapid breathing should raise your suspicion that the body is trying to compensate for metabolic acidosis. It is critical to check the patient's blood glucose level to try to narrow the differential diagnosis.

Make sure to perform a 12-lead ECG if you suspect DKA. The information it provides could change your management strategy (e.g., if the ECG reveals a myocardial infarction). In addition, electrolyte abnormalities often accompany diabetic emergencies, and a 12-lead ECG could reveal worrisome anomalies. Although there are many conditions that can present similarly to DKA, initial treatments steps are often the same.

Treatment
Patients with severe DKA look critically ill and require immediate treatment. A patient with an altered LOC may be actively vomiting, putting him or her at risk of aspiration. In such cases, consider early intubation to protect the airway. Remember that patients with DKA breathe rapidly to compensate for their metabolic acidosis. Therefore, if you intubate such a patient, you must maintain hyperventilation to prevent deterioration of acid-base status. Initiate aggressive fluid resuscitation using 0.9% normal saline administered through two peripheral lines. Adult patients with DKA usually require 3 to 6 L of fluid during initial resuscitation. Children may have similar fluid deficits but must be managed much more cautiously to prevent severe complications resulting from rapid electrolyte shifts. Monitor patients in DKA closely because their condition can decompensate rapidly. Patients with a history of heart failure can easily go into fluid overload; therefore be cautious when administering IV fluids. Consider underlying causes of DKA, such as myocardial infarction, and provide appropriate treatment.

Insulin therapy is a mainstay of treatment for DKA along with fluid resuscitation and electrolyte correction. Generally, however, insulin is not administered in the prehospital setting. EMS services that transport patients on insulin infusions (i.e., interfacility services) should have a protocol in place to guide management of such patients during transport. You must be able to recognize potential and common side effects of continuous insulin therapy. High-dose insulin is associated with iatrogenic hypoglycemia and hypokalemia, for example. This is caused by a shift of glucose and potassium into the cells after insulin administration. Although patients with DKA initially appear to be hyperkalemic, this is only due to a temporary shift of potassium out of the cells into the bloodstream caused by the acidosis. They typically have a total-body deficiency in potassium. Abnormal potassium levels can result in life-threatening cardiac arrhythmias, so you should confirm the patient's most recent potassium level before transport.

Key treatment considerations for patients with DKA or HHNC include the following:

- If the patient is intubated, maintain hyperventilation to prevent worsening of acidosis.
- Provide fluid rehydration. You may need to rapidly administer 1 to 2 liters of normal saline. Monitor glucose levels regularly because fluid resuscitation will decrease glucose levels.
- Evaluate the ECG for signs of hyperkalemia (peaked T waves, widened QRS complex, loss of P waves, bradyarrhythmias, or sign wave morphology), and treat accordingly.
In pediatric patients, administer initial fluid resuscitation of 20 mL/kg. Additional fluids should be administered only with expert consultation or direction from on-line medical control.

For extended critical care transports, consider the following treatment:

- Change the IV solution to D_W in 0.45% normal saline when glucose levels fall below 300 mg/dL (< 16.6 mmol/L).
- Correct electrolytes when indicated, using the following guidelines:
  - Potassium: If the potassium level is low, first ensure that the patient's renal function is adequate, and then add 20 to 40 mEq/L of potassium chloride for each liter of fluid administered.
  - Magnesium: If the magnesium level is low, correct the level with 1 to 2 g of magnesium sulfate in the first 2 liters of fluid administered.
  - Acidosis: If the pH falls below 7, correct it by adding 44 to 88 mEq/L of sodium bicarbonate to the first liter of IV fluid administered.
- Complications: Be aware of the potential complications of insulin infusions, such as hypokalemia and hypoglycemia.
- Remember, constant monitoring is essential. Treat underlying causes if possible, and transport the patient to a hospital with ICU capabilities.

An important consideration in DKA treatment relates to fluid administration in pediatric patients. Rapid shifts in fluid and electrolyte balances cause potentially fatal cerebral edema in a small percentage of children with DKA. Although there is still no definitive answer as to the specific risk factors for development of cerebral edema, consensus guidelines recommend a measured approach to fluid resuscitation in pediatric DKA. While these patients are most certainly volume depleted, they are rarely in hypovolemic shock, and the initial bolus should not exceed 10 to 20 mL/kg over 1 to 2 hours unless there is hemodynamic instability.

Complications of Treatment of DKA

The treatment of diabetic ketoacidosis is difficult and complex, requiring the participation of a multidisciplinary group of medical professionals. Even then, complications may develop in patients. Five major complications increase morbidity and mortality in DKA:

- Hypokalemia: can occur as a result of inadequate potassium replacement during treatment because aggressive insulin treatment shifts potassium into the cells.
- Hypoglycemia: can be attributed to aggressive treatment and failure to closely observe glucose. It is important to begin administering a 5% dextrose in water (D_W) solution when glucose levels fall below 300 mg/dL.
- Fluid overload: can be caused by aggressive fluid resuscitation in patients with congestive heart failure.
- Alkalosis: can be caused by overly aggressive treatment with bicarbonate. Alkalosis can further complicate electrolyte imbalances, specifically by increasing potassium requirements as potassium is displaced into body cells.
- Cerebral edema: the most feared complication of DKA treatment. It occurs as a result of rapid osmolar shifts. Cerebral edema generally appears 6–10 hours after the initiation of therapy and carries a mortality rate of 90%. You should suspect this complication in a patient who becomes comatose after acidosis is reversed during treatment of DKA.

Hyperosmolar Hyperglycemic Nonketotic Coma

Hyperosmolar hyperglycemic nonketotic coma (HHNC) is a serious diabetic emergency, carrying a mortality rate of 10% to 50%. You may not be able to differentiate DKA from HHNC in the field, but you should suspect it based on the patient’s history, extremely elevated glucose, and absence of low \textit{EtCO}_2. HHNC is more common in patients with type 2 diabetes mellitus and is triggered by the same stresses that cause DKA. The condition is characterized by the following:

- Elevated plasma glucose concentration, often greater than 600 mg/dL (> 33.3 mmol/L)
- Absent ketone production
- Increased serum osmolality, usually > 315 mOsm/kg

HHNC is associated with significant dehydration and a decline in mental status. Occasionally it progresses to full coma. In contrast to DKA, acidosis and ketosis are usually absent, so \textit{EtCO}_2 will not be decreased. It is important to realize that other factors such as underlying sepsis or respiratory dysfunction may still alter the \textit{EtCO}_2.

Pathophysiology

The pathophysiology of HHNC is complex but similar to that of DKA. The condition does not usually develop suddenly but evolves over a period of several days. The time frame varies, depending on the patient’s overall health. HHNC usually occurs in older adults and in patients debilitated by comorbid conditions. As in DKA, the hallmark is decreased insulin action, which triggers a volley of counterregulatory mechanisms that increase serum glucose. Once insulin function decreases, gluconeogenesis (the body’s internal manufacture of glucose), glycolysis (the release of glucose stored as glycogen), and decreased glucose uptake in the periphery begin to dominate. Hyperglycemia then pulls fluid into the extracellular space, triggering osmotic diuresis, which in turn causes hypotension and volume deficit. Patients are initially able to maintain intravascular volume with constant fluid intake, but the diuresis eventually overtakes the
Keep in mind that other conditions such as sepsis may be causing further volume depletion. Common causes of HHNC include the following:

- Trauma
- Drugs
- Myocardial infarction
- Cushing’s syndrome
- Sepsis
- Cerebrovascular accident (stroke)
- Dialysis
- CNS insult (e.g., subdural hematoma)
- Hemorrhage
- Pregnancy

**Signs and Symptoms**

Patients with HHNC are usually acutely ill, with marked volume depletion, nausea, vomiting, abdominal pain, tachypnea, and tachycardia. It is common for these patients to have a 25% fluid deficit. In addition, they may have focal neurologic deficits and seizures or signs of stroke. Signs and symptoms of HHNC include the following:

- Fever
- Dehydration
- Vomiting and abdominal pain
- Hypotension
- Tachycardia
- Rapid breathing
- Thirst, polyuria or oliguria, polydipsia
- Focal seizures
- Altered LOC
- Focal neurologic deficits

**Differential Diagnosis**

Many conditions have signs and symptoms similar to those of DKA (see earlier discussion) and HHNC. In most cases, your initial intervention will be similar for all of these possible illnesses, but be alert for time-sensitive underlying conditions that can cause DKA and HHNC, such as myocardial infarction and sepsis.

To differentiate HHNC from DKA, remember that the former is usually accompanied by a more profound decrease in mental status. Additionally, $\text{ETCO}_2$ may help distinguish the presence or lack of a metabolic acidosis. Signs and symptoms of HHNC can be confusing because they may be similar to those of hypoglycemia. If blood glucose cannot be rapidly evaluated, hypoglycemia must be assumed until proved otherwise. The administration of dextrose could minimally worsen glucose levels in HHNC, but it can be lifesaving in a patient with hypoglycemia.

**Treatment**

The initial management of a patient with HHNC is the same as that of a patient with DKA. Take immediate steps to stabilize the airway, breathing, and circulation. The patient may have significant volume depletion; begin IV fluid resuscitation immediately. The initial fluid of choice is 0.9% normal saline. Early boluses may be necessary to stabilize the patient hemodynamically. Use caution, however, when the patient has comorbidities such as CHF. Remember that fluid administration alone will correct much of the hyperglycemia. DKA management controversies apply to HHNC as well. For example, rapid correction of serum osmolality can predispose patients—especially children—to the development of cerebral edema.

**Acid–Base Disorders**

As previously discussed, endocrine disorders involve the body’s overproduction or underproduction of certain hormones. In comparison, acid–base disorders affect the body’s ability to process certain nutrients and vitamins.

**Acid–Base Balance**

The body requires a delicate balance, or homeostasis, to function optimally. Fluid, electrolytes, and pH all play critical roles in maintaining homeostasis. Acid–base stability is crucial to sustain life and maintain health. Acid–base balance is achieved through a variety of buffer systems and compensatory mechanisms. Body fluids, the kidneys, and the lungs play a pivotal role in maintaining this balance. Acid–base balance is measured by examining pH (the concentration of hydrogen) and is associated with a narrow safety margin (serum pH is 7.35–7.45). Acid–base balances can vary in severity based on the degree of pH change. A pH below 7.35 constitutes acidosis. In contrast, a pH level above 7.45 constitutes alkalosis. These pH derangements are classified according to their primary cause as either metabolic or respiratory. Death can occur if serum pH levels fall below 6.8 or rise above 7.8. Changes can occur because of various conditions including infections, organ failure, or trauma. In many cases the acid–base fluctuations can cause more negative effects than the causative condition; therefore, the resulting acid–base imbalance is often corrected before treating the underlying condition.

Two body systems can compensate for pH imbalances—the renal and respiratory systems. If the cause of the imbalance originates within one of those systems, the other system will have to be the primary compensatory mechanism. The system will not be able to resolve its own problem. Thus, if the problem originates in the lungs, the kidneys will manage it. If the problem originates outside the lungs, the lungs will manage it.

**Buffers**

Buffers are the chemicals that combine with an acid or base to resist changes in pH. Buffering is an immediate reaction to counteract pH variations until longer-term compensation is established. The body has four major buffer mechanisms—the
bicarbonate–carbonic acid system, the phosphate system, the hemoglobin system, and the protein system.

Respiratory Regulation
The respiratory system manages pH deviations by changing the amount of expired carbon dioxide (acid excretion). Speeding up respirations will lead to excretion of more carbon dioxide, thereby decreasing acidity. Slowing down respirations will lead to excretion of less carbon dioxide, increasing acidity. Chemoreceptors that sense pH changes trigger this change in breathing pattern. The only way the lungs can remove acids is through the elimination of carbon dioxide from carbonic acid—the lungs cannot remove other acids. The respiratory system is also a mechanism that can respond quickly to pH imbalances, but its quick action is short lived. The respiratory system reaches its maximum compensatory response in 12 to 24 hours, but it can maintain the changes in breathing pattern for only a limited time before becoming fatigued.

Renal Regulation
The renal system is the slowest mechanism to react to pH changes, taking hours to days to achieve its buffering effect, but it is the longest lasting. The kidneys respond by changing the excretion or retention of hydrogen (acid) or bicarbonate (base). The renal system acts to balance pH levels by permanently removing hydrogen from the body. Additionally, the kidneys can reabsorb acids or bases and produce bicarbonate to correct pH imbalances.

Compensation
To maintain homeostasis, the body will take actions to compensate for the pH changes. The body never overcompensates; the pH is adjusted so that it remains just within the normal range. The cause of the imbalance often determines the compensatory change. For example, if pH is becoming more acidic because of lung disease that limits gas exchange (e.g., emphysema), the renal system will kick in to compensate for the problem by releasing more bicarbonate and excreting more hydrogen. If a lung disease is increasing carbon dioxide excretion (e.g., hyperventilation), which will increase pH, the kidneys will compensate by decreasing bicarbonate production and hydrogen excretion. In contrast, if the problem originates outside the lungs, the lungs can compensate for it. For example, if a condition increases the loss of an acid (e.g., vomiting), the lungs will decrease the rate and depth of respirations to retain more carbon dioxide. If a condition increases the loss of a base (e.g., diarrhea), the lungs will increase the rate and depth of respirations to excrete more carbon dioxide. If the kidneys and lungs cannot compensate to restore the pH levels to normal range, cellular activities are affected, leading to disease states. Various mathematical formulas exist to calculate expected levels of compensatory responses and help determine if a condition is acute or chronic.

Respiratory Acidosis
Respiratory acidosis is one of the most common acid–base problems encountered in the prehospital setting. Respiratory acidosis is characterized by a decline in pH as a result of CO₂ retention. Hypoventilation is the classic example of a clinical problem that leads to CO₂ retention. Respiratory acidosis may be classified as acute or chronic. The only way to distinguish between these states is to determine whether the body has begun to retain bicarbonate to compensate for the acidosis. During the acute phase, the serum bicarbonate level is normal. Once the body begins to retain bicarbonate, it has made the transition to chronic status.

Pathophysiology
Any disorders that result in hypoventilation (e.g., primary pulmonary problems, airway obstruction, illnesses that depress the respiratory drive) will cause respiratory acidosis. Precipitants of respiratory acidosis are summarized in Table 7-4.

<table>
<thead>
<tr>
<th>Acute Precipitants</th>
<th>Chronic Precipitants</th>
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<tbody>
<tr>
<td><strong>Pharmacologic CNS Depression</strong></td>
<td><strong>Lung Disease</strong></td>
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<tr>
<td>Narcotics</td>
<td>Chronic bronchitis</td>
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<td>Benzodiazepines</td>
<td>COPD</td>
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<td>Alcohol abuse</td>
<td>Pulmonary fibrosis</td>
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<td>Gamma-hydroxybutyrate (GHB) toxicity</td>
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<tr>
<td><strong>Lung Disease</strong></td>
<td><strong>Neuromuscular Diseases</strong></td>
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<td>Interstitial edema</td>
<td>Muscular dystrophy</td>
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<td>Pneumonia</td>
<td>Myasthenia gravis</td>
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<td><strong>Airway Problems</strong></td>
<td><strong>Obesity</strong></td>
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<td>Foreign body</td>
<td>Sleep apnea</td>
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<td>Aspiration</td>
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<td>Bronchospasm</td>
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<td>Apnea</td>
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<td><strong>Hypoventilation</strong></td>
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<td>Pneumothorax</td>
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<td>Flail chest</td>
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<tr>
<td>Myasthenia gravis</td>
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<tr>
<td>Guillain-Barré syndrome</td>
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<tr>
<td>Primary CNS disorders</td>
<td></td>
</tr>
<tr>
<td>Brain injury</td>
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</table>

CNS, central nervous system; COPD, chronic obstructive pulmonary disease.
Signs and Symptoms
You may encounter different clinical scenarios, depending on the severity of the primary problems. Common signs and symptoms include weakness, breathing difficulty, and altered LOC. Noting the LOC is critical when evaluating a patient with suspected respiratory acidosis because it may indicate the severity of the process and signal the need for advanced airway management. For example, in a patient with COPD who has a diminished mental status, a high level of CO₂ is most likely responsible for the altered LOC. Such a patient has a higher risk of complications such as aspiration and therefore requires more aggressive intervention.

Differential Diagnosis
Many conditions can cause hypoventilation and/or impair gas exchange, resulting in respiratory acidosis (see Table 7-4).

Treatment
Standard monitoring equipment should be used according to your provider level, including an ECG monitor, Spo₂, and etco₂. The etco₂ measurement is an approximate measure of PaCO₂, and is generally felt to be accurate to within 5–10 mm Hg. After your initial evaluation and stabilization of the patient’s airway, breathing, and circulation (ABCs), therapy should focus on correcting minute ventilation to decrease CO₂ levels and thereby correct the acidosis. Depending on the etiology, you can accomplish this either by assisting ventilation or by providing pharmacologic intervention. Ventilatory assistance can range from airway positioning to bag-mask ventilation with nasopharyngeal airway or oropharyngeal airway, continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP), or endotracheal intubation with ventilator support. Pharmacologic intervention, such as naloxone administration, can reverse respiratory depression in patients whose hypoventilation can be attributed to the toxic effects of opiate overdose. Albuterol, ipratropium, and other medications may improve hypoventilation in patients with COPD.

All hypoxic patients should be treated with supplemental oxygen. Patients with chronically elevated CO₂ levels (i.e., patients with COPD who chronically retain CO₂) may have switched from relying on the normal hypercarbic respiratory drive to relying on the hypoxic drive and must be monitored for decreased respiratory effort when supplemental oxygen is administered. See Chapter 2 for a discussion of the hypercarbic and hypoxic drives.

Respiratory Alkalosis
An increase in ventilation per minute is the cause of respiratory alkalosis, characterized by a decreased PaCO₂ level and increased pH. The only way to differentiate between acute and chronic respiratory alkalosis is to measure serum bicarbonate. A patient with acute respiratory alkalosis will have a normal serum bicarbonate level. A patient with chronic respiratory alkalosis, however, will have a decrease in serum bicarbonate level.

Pathophysiology
Respiratory alkalosis is usually seen as a secondary compensatory mechanism to a primary metabolic problem, but it can be a primary derangement as well. Some causes of primary respiratory alkalosis include aspirin overdose, anxiety reaction, and pulmonary embolism. On occasion, it may be a normal physiologic response. The classic example is alkalemia of pregnancy, in which the pH is 7.46 to 7.5. This condition is primarily respiratory in origin and is characterized by a PaCO₂ of 31 to 35 mm Hg. Precipitants of respiratory alkalosis are given in Table 7-5.

<table>
<thead>
<tr>
<th>Pulmonary</th>
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<tbody>
<tr>
<td>Pulmonary embolism</td>
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<tr>
<td>Pneumonia (bacterial or viral)</td>
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<tr>
<td>Acute pulmonary edema</td>
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<tr>
<td>Atelectasis</td>
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<tr>
<td>Assisted hyperventilation</td>
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<th>Infections</th>
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<td>Septicemia</td>
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<tr>
<th>Drug Induced</th>
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<tr>
<td>Vaspressors</td>
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<tr>
<td>Thyroxine</td>
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<td>Aspirin or caffeine toxicity</td>
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<table>
<thead>
<tr>
<th>Hypoxia</th>
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<tbody>
<tr>
<td>Ventilation-perfusion mismatch</td>
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<tr>
<td>Altitude changes</td>
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<tr>
<td>Severe anemia</td>
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<thead>
<tr>
<th>Hyperventilation</th>
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<tbody>
<tr>
<td>Hysteria/anxiety</td>
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<tr>
<td>Psychogenic disorders</td>
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<tr>
<td>Central nervous system tumor</td>
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<tr>
<td>Stroke</td>
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<tr>
<th>Metabolic and Electrolyte Disturbances</th>
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<tbody>
<tr>
<td>Hepatic insufficiency</td>
</tr>
<tr>
<td>Encephalopathy</td>
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<tr>
<td>Hyponatremia</td>
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Signs and Symptoms
The patient’s clinical presentation depends on whether the respiratory alkalosis is chronic or acute. Most signs and symptoms are nonspecific and are related to peripheral or CNS complaints such as paresthesia of the face or lips, lightheadedness, dizziness, and muscular pain or cramps.

Differential Diagnosis
A diagnosis of respiratory alkalosis may not be obvious because some of its signs and symptoms are almost identical to those of certain electrolyte emergencies such as hypocalcemia. A thorough history and physical exam will yield clues to the underlying cause of the respiratory alkalosis, which may guide your management strategies. Be careful not to overlook life-threatening toxicologic causes such as aspirin overdose.

Treatment
Administer oxygen to patients with hypoxemia without delay, and take steps to stabilize and support the airway, breathing, and circulation. For hyperventilation caused by anxiety, use coaching techniques to calm the patient. Instruct him or her to use pursed-lip breathing. To avoid precipitating hypoxia, do not use a paper bag or a nonrebreathing mask without oxygen attached.

Metabolic Acidosis
Metabolic acidosis is caused by a deficiency of bicarbonate ion (base) and an excess of hydrogen ion (acid). In the acute state, the body’s physiologic response is to hyperventilate and compensate by reducing $\text{Paco}_2$. This is sometimes referred to as “blowing off $\text{CO}_2$.” Chronic status is reached when the renal system begins to reabsorb bicarbonate in an effort to compensate for the metabolic acidosis.

Pathophysiology
Metabolic acidosis is generated by three mechanisms: decreased renal excretion of acids, increased production or ingestion of acids, and loss of buffering mechanisms in the body.

Signs and Symptoms
The clinical manifestations of metabolic acidosis are directly related to the severity of the metabolic problem. Most patients have nausea, vomiting, abdominal pain, a rapid and deep respiratory pattern (Kussmaul’s respiration), and in more severe cases, altered LOC and shock.

Differential Diagnosis
Metabolic acidosis is classified as either non–anion-gap acidosis or anion-gap acidosis. The anion gap is calculated using the following formula:

$$\text{AG} = \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$$

This information gives the provider an estimate of unmeasured anions in the plasma. An anion gap of 12 to 15 is considered normal. An elevated gap points to conditions that may cause acidosis. The mnemonic CAT MUDPILES can help you remember the precipitants of high-anion-gap metabolic acidosis. The mnemonic F-USED CARS will help bring to mind the causes of normal-anion-gap metabolic acidosis.

Providers may not have access to the laboratory information necessary to calculate the anion gap. Management decisions, therefore, are often made on the basis of sound clinical judgment, a thorough history, and physical exam findings. A specialty-care transport provider conducting a hospital transfer may have laboratory values for calculating the anion gap and can adjust the differential diagnosis accordingly. Capnometry can also provide key information. A patient with tachypnea and a low $\text{P CO}_2$ should be suspected to have metabolic acidosis or a primary respiratory alkalosis, as previously discussed.

When patients present with clinical signs of acidosis, the following five conditions must be considered:

- **Diabetic ketoacidosis.** As discussed earlier in the chapter, DKA is caused by inadequate use of insulin as a result of poor compliance or increased need. Patients with diabetes sometimes require higher insulin doses during periods of infection, after trauma, or in other circumstances that increase metabolic demand. DKA sets in when glucose utilization is impaired and fatty acids are metabolized, causing the formation of ketone bodies that generate hydrogen ions. If more acids are produced than the body’s buffering system is able to tolerate, acidosis ensues.

- **Renal failure.** The kidneys are vital in maintaining an optimal acid–base balance. Most patients with renal failure have uremia because the kidneys are unable to secrete acid by-products. The renal tubules have the primary responsibility for eliminating hydrogen ions. This function is directly related to the filtration rate of the kidneys, known as the glomerular filtration rate (GFR). Any pathology that alters this process will increase the concentration of hydrogen ions, especially in the form of hydrogen sulfate ($\text{HSO}_4^-$) and HPO$_4^{2-}$, increasing the anion gap. Patients with chronic renal failure will have some degree of anion gap acidosis; but the gap rarely exceeds 25. Patients with acute renal failure, however, more often have hyperchloremic non–anion-gap acidosis.
Lactic acidosis. Lactic acid is largely generated when a significant number of cells in the body are inadequately perfused. Hypoperfusion shifts the cellular metabolism from aerobic (with oxygen) to anaerobic (without oxygen). Anaerobic metabolism produces lactic acid as its most important end product. This reaction occurs in time-sensitive medical conditions associated with hypoperfusion (e.g., sepsis, ischemia, extreme physical exertion states, prolonged seizures, circulatory shock). Lactic acidosis occurs when lactic acid accumulates in larger amounts than the body can buffer.

- **Toxin ingestion.** Toxic metabolites that cause metabolic acidosis may be a by-product of ingestion of toxins such as acetylsalicylic acid (ASA), ethylene glycol, methanol, and isoniazid. Patients with toxin-induced metabolic acidosis show some degree of respiratory compensation. The toxin must be identified as soon as possible because an antidote may be available to prevent further adverse effects.

- **Alcohol ketoacidosis.** This is caused by abrupt cessation of intake after a prolonged period of ingesting a considerable amount of alcohol. The main problem—accumulation of keto acids—is precipitated by dehydration, hormone imbalance, and chronic malnutrition. Although the condition is similar in presentation to DKA, blood glucose levels are normal or low. Patients with alcohol ketoacidosis often have mixed acid–base disorders associated with the vomiting that accompanies alcohol withdrawal.

**Treatment**

Most patients with metabolic acidosis will require a significant amount of volume resuscitation. Rapidly establish intravascular access to replenish volume status. Support the airway, breathing, and circulation with oxygen as appropriate, and ensure adequate ventilation. In patients with history of renal failure or CHF, use caution to avoid causing pulmonary edema when administering IV fluids. If the patient needs ventilator support, be sure to maintain hyperventilation. Patients with metabolic acidosis are hyperventilating as a respiratory compensatory mechanism, and if they are sedated or paralyzed for intubation, metabolic acidosis will worsen. Initiate adjunct treatments on the basis of primary etiology. For example, patients with high-anion-gap metabolic acidosis due to DKA can be started on insulin.

The use of sodium bicarbonate may be necessary for certain conditions that elicit acute metabolic acidosis, but administration of bicarbonate can be fraught with complications, including hypocalcemia, volume overload, CNS acidosis, hypokalemia, and impaired oxygen delivery. Despite the controversy that surrounds its use, rapid administration of sodium bicarbonate may be useful in treating certain life-threatening conditions. Providers use arterial blood gas and plasma electrolyte values to guide the decision of whether to administer bicarbonate. Although it is unlikely that such information will be available in the prehospital setting, administering bicarbonate is warranted in the following circumstances:

- **Lactic acidosis.** Lactic acid is largely generated when a significant number of cells in the body are inadequately perfused. Hypoperfusion shifts the cellular
Cardiac arrest caused by acidosis associated with hyperkalemia
- Overdose with tricyclic antidepressants (ECG shows QRS complex widening > 0.10 sec)
- Hyperkalemia (presumptive diagnosis made on the basis of history and ECG findings)

### Metabolic Alkalosis
Metabolic alkalosis is produced by illnesses that raise the level of serum bicarbonate or reduce the level of hydrogen in the body, such as those that cause volume, potassium, and chloride loss.

### Pathophysiology
Metabolic alkalosis occurs by one of two mechanisms: either the body retains bicarbonate in response to hydrogen and chloride loss, or renal impairment precludes the excretion of bicarbonate. Table 7-6 provides a list of specific conditions that may precipitate metabolic alkalosis.

### Signs and Symptoms
Common signs and symptoms in patients affected by metabolic alkalosis are anorexia, nausea, vomiting, confusion, hypotension, paresthesia, and weakness. A thorough assessment may reveal the use of antacids (e.g., sodium and calcium bicarbonates), loop diuretics such as thiazide, and corticosteroids. Underlying medical illnesses such as Cushing's syndrome and renal disease are common.

**Table 7-6 Precipitants of Metabolic Alkalosis**

<table>
<thead>
<tr>
<th>Normal Saline-Responsive Metabolic Alkalosis</th>
<th>Normal Saline-Unresponsive Metabolic Alkalosis</th>
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<tr>
<td>Volume depletion</td>
<td>Mineralocorticoid excess</td>
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<tr>
<td>- Vomiting</td>
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<td>- Nasogastric suction</td>
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<tr>
<td>- Diuretic use</td>
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<tr>
<td>- Low chloride ingestion</td>
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<tr>
<td>Exogenous ingestions</td>
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<tr>
<td>- Chewing tobacco</td>
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<tr>
<td>- Licorice</td>
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<tr>
<td>Primary aldosteronism</td>
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<tr>
<td>Cushing's syndrome</td>
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<td>Bartter syndrome</td>
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The patient will present with slow, shallow respirations. ECG changes with depressed T waves that merge with P waves indicate hypocalcemia and hypokalemia. Hypotension is also present. Many patients present with muscle twitching and loss of reflexes and numbness and tingling in the extremities; a thorough neurologic exam should be performed. Arterial blood gas analysis reveals a blood pH above 7.45 and an elevated HCO₃⁻. If respiratory compensation is occurring, the PaCO₂ level may be above 45 mm Hg.

### Differential Diagnosis
To make a definitive diagnosis of metabolic alkalosis, you need to know the serum bicarbonate level and the arterial CO₂ level. A rise in the serum bicarbonate level may be a renal compensatory response to chronic respiratory acidosis. This information can be obtained only by blood gas testing.

### Treatment
Management of metabolic alkalosis is directed toward correcting the underlying cause. A comprehensive history and physical exam are vital. Administration of IV fluids is essential if the primary cause is volume depletion. Isotonic solutions are the fluids of choice. Hypokalemia may need to be corrected with potassium replacement.

### Mixed Disorders
Patients often have mixed acid–base disturbances, the diagnosis of which may be difficult even for an experienced emergency physician or intensivist. Mixed disturbances are identified on the basis of clinical history combined with blood gas analysis. Your initial clinical impression of whether the patient is sick or not sick is especially important. As always, take any immediate steps necessary to support airway, breathing, and circulation.

### Electrolyte Disturbances
Electrolyte imbalances are common findings in patients with medical emergencies. A healthy electrolyte balance is fundamental to carrying out cellular functions. Electrolyte disturbances generally cannot be diagnosed on the basis of clinical examination alone, but a thorough history and exam may point to a likely diagnosis. Severe electrolyte disturbances can be fatal. Most patients have only nonspecific chief complaints until life-threatening manifestations appear. In the following section, the most important electrolyte problems you are likely to encounter in the field are discussed.

### Hyponatremia
Sodium is the most important electrolyte in maintaining water balance in the body. As the principal cation in the extracellular...
fluid, sodium together with chloride and bicarbonate regulates osmotic forces (the flow of water in and out of cells). Water balance is maintained by hormonal regulation controlled by the brain and kidneys.

**Hyponatremia** is defined as a serum sodium concentration below 135 mEq/L. To guide management, hyponatremia is classified into the following three categories, depending on volume status:

- Hypervolemic hyponatremia occurs when an excessive amount of water is retained relative to the amount of sodium. The condition classically occurs in a patient with an edematous condition such as CHF. It may also occur in patients who have excessive water intake such as with psychogenic polydipsia or when large amounts of water are ingested intentionally over a short period of time.
- Hypovolemic hyponatremia is caused by the loss of water and sodium, with a higher degree of sodium loss relative to the amount of water loss. Common precipitants include vomiting, diarrhea, GI problems, nasogastric tubes, and third-spacing of fluids. Third-spacing (movement of intravascular and intracellular water into interstitial spaces) is a phenomenon that may occur in patients with burns, pancreatitis, and sepsis and in those who take certain medications such as diuretics.
- Euvolemic hyponatremia occurs when the serum osmolality is low despite the presence of concentrated urine.

### Signs and Symptoms

The clinical presentation of hyponatremia depends on how quickly the sodium concentration declines. Patients who experience a rapid drop in serum sodium level often begin to show symptoms around 125–130 mEq/L; however, a patient with chronic hyponatremia may tolerate a level below 120 mEq/L without symptoms.

Most signs and symptoms of hyponatremia are related to CNS manifestations, such as agitation, hallucinations, weakness, lethargy, and seizures. Abdominal pain, cramps, and headache may also occur. Patients with severe hyponatremia appear to be very ill and may have seizures or exhibit an altered mental status.

Athletic events such as marathons and triathlons can precipitate exercise-induced hyponatremia. Although the mechanisms that cause this phenomenon are not completely understood, persistent increased vasopressin levels and a decrease in glomerular function in sweat-induced dehydration may be implicated. Exercise-induced hyponatremia can cause loss of coordination, pulmonary edema, and changes in intracranial pressure that result in seizure and coma.

Patients with very high glucose levels (or excessive lipids or proteins in the blood) will exhibit pseudohyponatremia with a measured sodium level that appears quite low. This low sodium measurement must be corrected using a formula to ascertain the true sodium level.

### Differential Diagnosis

The differential diagnosis of hyponatremia as well as the identification of the underlying cause is often complex and may be a difficult task even for specialists in the hospital. In the prehospital environment the history and exam should help guide you to consideration of hyponatremia.

### Treatment

On the basis of history and physical exam, try to determine if the patient may be suffering from hyponatremia and the most likely cause. Patients who are hemodynamically unstable should have fluid resuscitation initiated with 0.9% normal saline. All fluids, however, particularly in patients who are hemodynamically stable, should be administered with extreme caution in patients with hyponatremia. Until a serum sodium level is known and a total body water deficit has been calculated, aggressive hydration runs the risk of correcting the sodium too quickly and leading to severe complications.

You will rarely have serum sodium measurements to guide management in the prehospital environment, although point-of-care testing is available in some circumstances. As a general rule, hyponatremia should be corrected extremely slowly. The recommended rate of correction is no faster than 1 to 2 mEq/L per hour. The exception to this rule is patients with severe neurologic symptoms such as altered mental status or seizures. In these patients, a rapid correction may be needed to alleviate the symptoms. This may be done with a 100-mL bolus of 3% sodium chloride (hypertonic saline). Hypertonic saline should never be administered without direct supervision of medical control. Correcting the sodium level too aggressively (either with normal saline or hypertonic saline) can cause severe neurologic complications as a result of osmotic demyelination.

### Hypokalemia

Potassium is responsible for the following vital functions in the body:

- Maintaining a normal electrical and osmotic gradient in all cells
- Facilitating neuronal transmission and cardiac impulse conduction
- Serving as a buffering mechanism in the cell membranes to help maintain acid–base homeostasis

Normal serum potassium levels range from 3.5 to 5 mEq/L but do not accurately reflect total body stores of the cation because most potassium is stored within cells. Hypokalemia is
an abnormally low serum level of potassium, usually < 3.5 mEq/L. Hypokalemia is fairly common and usually occurs secondary to decreased intake or increased excretion.

Signs and Symptoms
Hypokalemia often manifests with no signs or symptoms initially. As it progresses and the potassium level falls below 2.5 mEq/L, signs and symptoms of hypokalemia become apparent in multiple organ systems, including the neurologic, GI, and cardiovascular systems. Common symptoms include weakness, nausea, vomiting, lethargy, confusion, and paresthesia of the extremities.

A patient with severe hypokalemia (< 2 mEq/L) will appear to be very ill and may also have cardiac dysrhythmias and muscular paralysis. Frequent cardiovascular manifestations include palpitations, low blood pressure, and cardiac electrical disturbances such as heart blocks, premature ventricular contractions, and supraventricular tachycardia. Fatal types of dysrhythmia, such as ventricular fibrillation and asystole, can also occur (Figure 7-10).

Differential Diagnosis
Signs of hypokalemia apparent on a 12-lead ECG include flattened T waves, U waves, and ST-segment depression. Clinical manifestations of hypokalemia are similar to those of hyperkalemia.

Treatment
Treatment of hypokalemia may require IV fluids for dehydration. Oral potassium replacement (20 to 40 mEq per dose) is preferred over IV administration because of the potential side effects of IV potassium. Patients who are unable to take oral replacement or are critically ill will require IV potassium administered at a rate of 10 to 20 mEq per hour. Critically ill patients (those with respiratory muscle weakness) can receive higher doses, but they should be given through a central venous catheter. Overly rapid infusion of IV potassium can result in cardiac arrest. A common complaint during IV administration is burning at the site of infusion, which you can usually resolve by slowing the rate of infusion. Hyperkalemia is a complication of potassium administration, which is especially likely in patients with kidney disease. It is therefore critical to know the patient’s renal function status before you administer potassium.

Hyperkalemia
Hyperkalemia, a level of serum potassium > 5.5 mEq/L, is an electrolyte disorder that can be caused by ingestion of potassium supplements, acute or chronic renal failure, blood transfusion, sepsis, Addison’s disease, acidosis, and crush syndrome (from rhabdomyolysis).

Signs and Symptoms
Hyperkalemia manifests primarily as neurologic and cardiovascular dysfunction. The patient may have generalized weakness, muscle cramps, tetany, paralysis, or cardiac palpitations or arrhythmias.

Differential Diagnosis
In the prehospital setting, the only diagnostic study available to guide you toward a diagnosis of hyperkalemia is the ECG, which can help you determine whether the patient has an associated arrhythmia. Classically, the first change detected on the ECG of a patient with hyperkalemia is the development of peaked T waves. As serum potassium continues to increase, P waves disappear, and the QRS complex widens. If hyperkalemia is not corrected, the ECG will progress to bradycardia and then terminate in a sine wave pattern or asystole.

Treatment
Assess and treat the underlying cause of hyperkalemia, institute rapid and appropriate treatment, and transport the patient.
to a hospital facility. Treatment of hyperkalemia has the following three goals:

- **Cellular membrane stabilization and decreased cardiac irritability.** Maintain the patient on a cardiac monitor at all times. If the patient has signs of hyperkalemia on ECG, hypotension, or arrhythmias, administer 5 mL of 10% solution of calcium chloride. In many systems, this may require consultation with online medical control unless the patient is already in cardiac arrest.

- **Potassium shift into cells.** Sodium bicarbonate, 44 mEq/L IV, may be administered to drive potassium into cells and out of the serum. Nebulized albuterol, 5 to 20 mg, will lower the serum potassium level by shifting potassium into cells. The combined administration of 10 units of insulin and IV dextrose similarly produces a shift of potassium into the cells.

- **Elimination of potassium from the body.** To help eliminate potassium from the body, the use of exchange resins is common practice although supported by limited data. An oral dose of 20 g sodium polystyrene sulfonate can be used. Be careful when using exchange resins in a cardiac patient, however, since they can produce fluid overload.

### Hypocalcemia

As previously discussed in the section on hypoparathyroidism, calcium is essential for a number of body functions, including muscular contraction, neuronal transmission, hormone secretion, organ growth, and immunologic and hematologic response. Most calcium in an adult is stored as a mineral component of bone. Hypocalcemia occurs when ionized calcium levels fall below 4 mEq/L. This condition occurs as a result of increased losses or decreased intake of calcium.

#### Signs and Symptoms

Patients with symptomatic hypocalcemia may have seizures, hypotension, tetany, or cardiac dysrhythmias.

#### Differential Diagnosis

In addition to the signs and symptoms, two signs may be present—Trousseau's and Chvostek's signs—to help you narrow down the possible diagnosis.

#### Treatment

Treatment of hypocalcemia is guided principally by laboratory results, but when hypocalcemia is presumed to be the cause of the patient's symptoms, it may be reasonable to begin empirical treatment. Parenteral calcium is the primary treatment in patients with symptomatic hypocalcemia. Use one of the following two options:

- 10 mL 10% calcium chloride, which contains 360 mg elemental calcium
- 10 mL 10% calcium gluconate, which contains 93 mg elemental calcium

In an adult patient, the recommended dose is 100 to 300 mg elemental calcium. In a pediatric patient, administer 0.5 to 1 mL/kg of a 10% calcium gluconate solution over 5 minutes. To avoid significant side effects, dilution in normal saline or D5W is highly recommended. Care must be taken to ensure the peripheral catheter is working properly before administering calcium because extravasation may cause tissue necrosis. Calcium administration will increase the serum concentration of calcium for only a short period of time, so you may need to give repeated doses, especially during a long transport or interfacility transfer.

Patients whose signs and symptoms persist after adequate treatment may have concomitant electrolyte problems such as hypomagnesemia.

### Hypomagnesemia

Magnesium is the second most abundant intracellular bivalent cation in the human body. It is a cofactor in the activation of numerous enzymatic reactions. Its physiologic effects on the CNS are similar to those of calcium. Magnesium is distributed throughout the body in a unique way. Half of the total amount of magnesium (2,000 mEq/L) is stored as a mineral component of bone, and 40% to 50% is intracellular. Only 1% to 2% of magnesium in the body is in extracellular fluid; thus the serum magnesium level is a poor reflection of the body's total magnesium content.

Hypomagnesemia is one of the most common electrolyte disturbances you will see in clinical practice. It often accompanies conditions that involve malnutrition, alcoholism, dehydration, diarrhea, kidney disease, diuresis, or starvation and tends to coexist with diseases that cause hypokalemia and hypocalcemia.

#### Signs and Symptoms

Patients usually become symptomatic at magnesium levels of 1.2 mg/dL (0.06 mmol/L) or less. Common signs and symptoms include the following:

- Tremors
- Hyperreflexia
- Tetany
- Nausea or vomiting
- Altered mental status and confusion
- Seizures
- Cardiac dysrhythmias, including torsades des pointes, polymorphic ventricular tachycardia, and cardiac arrest
Treatment
Take immediate steps to maintain the airway, breathing, and circulation. It is reasonable to start magnesium replacement therapy when you suspect a diagnosis of hypomagnesemia. In patients with no history of renal problems, administer a dose of 2 g of 50% magnesium sulfate. It must be given with normal saline or dextrose, ideally administered over 30 to 60 minutes per gram. However, in a patient with severe signs and symptoms, including dysrhythmias, you may need to give a rapid infusion over the course of 5 or 10 minutes. Do not give magnesium sulfate as a bolus because this been associated with severe side effects, including bradycardia, heart block, and hypotension.

Rhabdomyolysis
Rhabdomyolysis is a breakdown of muscle tissue that causes myoglobin to be released into the bloodstream, causing kidney damage. This muscle injury usually results from prolonged periods of immobilization, certain metabolic insults, or pressure or crush force on the tissue. Patients such as those who have experienced an opioid overdose, a person pinned under an industrial machine, or an elderly person who has spent several hours on the floor after falling may all suffer from rhabdomyolysis. Regardless of the particular insult to the tissue, the end result in each of these cases is the release of intracellular contents as the individual muscle cells rupture and die. Myoglobin, one of the main proteins found in skeletal muscle cells, travels to the kidneys and causes injury and even renal failure. Electrolytes that are normally sequestered within the cell may also be released, resulting in metabolic disturbances that are only exacerbated by the concurrent renal injury. In extreme cases, patients may have massive hyperkalemia resulting in fatal cardiac arrhythmias.

Pathophysiology
Rather than being a primary problem, rhabdomyolysis occurs as a consequence of another insult. Common precipitants of rhabdomyolysis include the following:

- Metabolic problems
- Heatstroke and other severe heat-related emergencies
- Trauma
- Crush injuries
- Drugs of abuse
- Toxic ingestion/overdose
- Infections (rarely)
- Electrolyte abnormalities

Dysfunction of the Na+/K+-ATPase pump allows uncontrolled calcium influx into skeletal muscle cells. The increased intracellular calcium content leads to cellular necrosis and release of myoglobin, potassium, and intracellular enzymes, such as creatine phosphokinase. Once myoglobin enters the plasma, it is filtered and excreted through the kidneys. An excess of myoglobin can be directly toxic to the renal tubules or can obstruct them, especially if the patient is hypovolemic or acidic as a result of the primary problem. If not treated aggressively with IV fluids, rhabdomyolysis can cause severe kidney damage and renal failure.

Signs and Symptoms
Patients with rhabdomyolysis report diffuse or localized weakness and muscle pain. Once the process of rhabdomyolysis has begun, patients may have dark-colored urine. If the patient develops hyperkalemia, the aforementioned signs and symptoms may also occur.

Differential Diagnosis
Rhabdomyolysis is diagnosed in the ED by noting myoglobinuria (the presence of myoglobin, a protein released in muscle breakdown in the urine) and an elevated creatine kinase level in the blood. However, you should suspect this diagnosis on the basis of a comprehensive history (including that of the primary condition) and physical exam findings. The patient may not have rhabdomyolysis initially, but an emergent condition may induce the condition later. A thorough physical exam is the key to identifying potential causes. For example, you may discover dark or even cola-colored urine, which is a strong indicator of the presence of rhabdomyolysis.

Treatment
Aggressive fluid hydration is crucial. IV fluids should be given (taking care to avoid hypothermia) in an effort to mitigate the complications of rhabdomyolysis. In addition to routine medical care, consider the following:

- Aggressive saline infusion early, especially in patients with trauma or crush injuries. Saline infusion is vital in the treatment of rhabdomyolysis.
- Titration of saline infusions to obtain a urine output of 200 to 300 mL/h. Be aware of potential electrolyte complications (such as hyperkalemia with hypocalcemia) that may elicit malignant cardiac dysrhythmias. If they occur, you must treat them aggressively.
- Administration of mannitol for osmotic diuresis.
- Initiation of a bicarbonate infusion to begin alkalinizing the urine if you already know the patient's primary diagnosis (e.g., when carrying out an interfacility transfer).
Putting It All Together

Patients with endocrine and metabolic disorders can be some of the most challenging problems a healthcare provider faces. Similarities and differences in the chief complaint/cardinal presentation are sometimes subtle, and your ability to determine the underlying diagnosis can be obscured, delaying appropriate interventions. Using the AMLS assessment pathway will assist you in obtaining a comprehensive history and focused physical exam. The assessment-based approach supports putting your knowledge of anatomy, physiology, and pathophysiology to work to figure out both the common and uncommon causes of these diverse disease processes. The use of pattern recognition can help you compare your patient’s clinical presentation to the chief complaint and formulate a working diagnosis. Becoming proficient in analyzing and synthesizing information to safely, efficiently, and effectively care for these patients will be well worth the effort it takes. Your contributions as an EMS team member are always a vital link in helping improve patient outcomes.

- Differential diagnoses may include an electrolyte imbalance such as hypokalemia or hypernatremia, metabolic alkalosis (related to Cushing’s syndrome) or metabolic acidosis (related to treatment with metformin), hyperglycemia or hypoglycemia, digoxin toxicity, sepsis, or heart failure.
- To narrow your differential diagnosis, you’ll need to complete the history of past and present illness. Perform a physical examination that includes assessment for dehydration, assessment of heart and breath sounds, and mental status. Diagnostic testing should include blood glucose, ECG monitoring and 12-lead ECG, SaO₂, ETCO₂, and blood chemistry if available.
- The patient has signs that may indicate shock, infection, or electrolyte imbalance. Signs of shock may be masked by prednisone treatment, and the presence of digoxin will prevent the increase in heart rate to compensate for shock. Administer oxygen, establish vascular access, and administer IV fluids. Continue to monitor the ECG, and transport the patient to the closest appropriate hospital.

Scenario Solution

- The endocrine system is responsible for hormone regulation, including homeostasis, reproduction, growth, development, and metabolism, and is composed of the pituitary, thyroid, parathyroid, and adrenal glands, as well as the pancreas, ovaries, and testes.
- Hormones stimulate growth and development throughout the body, regulate the flow of water in and out of cells, help muscles contract, control blood pressure and appetite, modulate the sleep cycle, and influence many other functions.
- Endocrine glands are interdependent on one another.
- Parathyroid glands are composed of three types of cells and are responsible for producing parathyroid hormone (PTH), detecting changes in extracellular calcium concentration, and inhibiting calcitonin secretion.
- Hypoparathyroidism is characterized by low serum levels of PTH, with the hallmark of this condition being hypocalcemia.
- The thyroid gland is composed of secretory cells, follicular cells, and C cells.
- Hyperthyroidism can result in thyrotoxicosis and, potentially, thyroid storm.
- The adrenal gland secretes glucocorticoids, mineralocorticoids, and supplemental sex hormones.
- Addison’s disease, or primary adrenal insufficiency, is a metabolic and endocrine ailment caused by direct insult to or malfunction of the adrenal cortex.
- Acute adrenal insufficiency is a condition in which the body’s need for glucocorticoids and mineralocorticoids exceeds the delivery of these hormones by the adrenal glands.
- Hyperadrenalism, or Cushing’s syndrome, is caused by long-standing exposure to excessive circulating serum levels of glucocorticoids, particularly cortisol, as a result of overproduction in the adrenal cortex.
- Glucose is a vital fuel for key metabolic processes in organs, especially those in the central nervous system (CNS).
Summary (CONTINUED)

- Cellular survival depends on preserving a balanced serum glucose concentration.
- Diabetes is the most common endocrine disorder, and hypoglycemia, a frequent complication of the treatment of diabetes, is thus the most common endocrine emergency.
- Diabetes mellitus is characterized by defective insulin production or utilization, a high level of blood glucose, and unbalanced lipid and carbohydrate metabolism. Left untreated, diabetes results in hyperglycemia.
- Hypoglycemia among diabetics is the result of a disruption in the delicate balance between the interdependent factors of exogenously administered insulin, glucose metabolism, and glucose intake.
- Hypoglycemia may occur in patients taking only oral hypoglycemic agents, but should alert the healthcare provider to the potential presence of an underlying pathophysiologic state such as new-onset renal failure.
- Type 1 diabetes is characterized by pancreatic beta cell destruction, which renders the body incapable of producing the insulin necessary to carry out cell metabolism.
- Type 2 diabetes is characterized by cellular insulin resistance and a gradual failure of pancreatic insulin production.
- Gestational diabetes is a form of glucose intolerance that can occur in pregnant women.
- Healthy cellular function is directly related to a precise acid–base balance in the body, with the kidneys and lungs maintaining this balance, which is measured by pH.
- Hypoglycemia results in a decrease in insulin secretion and secretion of counterregulatory hormones such as epinephrine. Symptoms include impaired cognition. If untreated, hypoglycemia can lead to significant morbidity and mortality.
- Hypoglycemia in nondiabetic patients is characterized by alimentary hyperinsulinism, commonly seen in patients who have undergone gastric surgery or as the result of an imbalance between glucose utilization and production.
- Diabetic ketoacidosis is an acute endocrine emergency in which insulin deficiency and an excessive glucagon level combine to create a hyperglycemic, acidotic, volume-depleted state.
- Hyperosmolar hyperglycemic nonketotic coma (HHNC) is a serious diabetic emergency, carrying a mortality rate of 10% to 50%.
- Respiratory acidosis is characterized by a decline in pH as a result of CO₂ retention. An increase in ventilation per minute is the cause of respiratory alkalosis, which is characterized by an increased PaCO₂ and increased pH.
- Metabolic acidosis is caused by the accumulation of acids in excess of the body's buffering capabilities. The most common serious causes of metabolic acidosis are diabetic ketoacidosis, renal failure, lactic acidosis, toxic ingestion, and alcoholic ketoacidosis.
- Respiratory alkalosis is characterized by a decline in pH as a result of CO₂ retention. An increase in ventilation per minute is the cause of respiratory alkalosis, which is characterized by an increased PaCO₂ and increased pH.
- Metabolic acidosis is caused by the accumulation of acids in excess of the body's buffering capabilities. The most common serious causes of metabolic acidosis are diabetic ketoacidosis, renal failure, lactic acidosis, toxic ingestion, and alcoholic ketoacidosis.
- Metabolic alkalosis is produced by illnesses that raise the level of serum bicarbonate or reduce the level of hydrogen in the body, such as those that cause volume, potassium, and chloride loss.
- A healthy electrolyte balance is fundamental to carrying out cellular functions; electrolyte imbalances include hyponatremia, hypokalemia, hyperkalemia, hypocalcemia, and hypomagnesemia.
- Rhabdomyolysis is a skeletal muscle injury characterized by release of cellular contents, specifically myoglobin, potentially leading to acute renal failure and hyperkalemia.

KEY TERMS

Addison’s disease  An endocrine disease caused by a deficiency of corticosteroid hormones produced by the adrenal cortex. The disease is characterized by nausea, vomiting, abdominal pain, and tanning of the skin.

adrenal crisis  An endocrine emergency caused by a deficiency of corticosteroid hormones produced by the adrenal cortex. The disease is characterized by nausea, vomiting, abdominal pain, hypotension, hyperkalemia, and hyponatremia.
**KEY TERMS (CONTINUED)**

**diabetic ketoacidosis (DKA)** An acute endocrine emergency caused by a lack of insulin. The condition is characterized by an elevated blood glucose level, ketone production, metabolic acidosis, dehydration, nausea, vomiting, abdominal pain, and tachypnea.

**hyperosmolar hyperglycemic nonketotic coma (HHNC)** An endocrine emergency characterized by a high plasma glucose concentration, absent ketone production, and increased serum osmolality (> 315 mOsm/kg). The syndrome causes severe dehydration, nausea, vomiting, abdominal pain, and tachyphnea.

**hypoglycemia** A plasma glucose concentration of less than 70 mg/dL. This condition is often associated with signs and symptoms such as sweating, cold skin, tachycardia, and altered mental status.

**myxedema** Severe hypothyroidism associated with cold intolerance, weight gain, weakness, and declining mental status.

**thyroid storm** An endocrine emergency characterized by hyperfunction of the thyroid gland. This disorder is associated with fever, tachycardia, nervousness, altered mental status, and hemodynamic instability.

**thyrotoxicosis** A condition of elevated thyroid hormone levels, which often leads to signs and symptoms of tachycardia, tremor, weight loss, and high-output heart failure.

**BIBLIOGRAPHY**


**CHAPTER REVIEW QUESTIONS**

1. Your patient complains of discomfort in his hand as you inflate the cuff to assess the blood pressure. You note flexion of the wrist and adduction of his fingers. Which endocrine disorder do you suspect?
   a. Addison’s disease
   b. Cushing’s syndrome
   c. Hypoparathyroidism
   d. Myxedema

2. A 47-year-old woman is anxious and complaining of heart palpitations. She reports a recent diagnosis of “thyroid problems.” On exam you note exophthalmos. Her vital signs include a blood pressure of 108/72 mm Hg; pulse rate, 128 beats/min; and respirations, 20 breaths/min. Interventions should include administration of:
   a. Amiodarone
   b. Aspirin
   c. Intravenous fluids
   d. Methylprednisolone
3. Which assessment finding(s) should you anticipate in a patient who has myxedema?
   a. Chvostek's sign
   b. Dry, yellow skin
   c. Exophthalmos
   d. Hyperactive reflexes

4. Which treatment should you anticipate in a patient with a history of Addison's disease who has the following vital signs: a blood pressure of 94/58 mm Hg; pulse, 124 beats/min; respirations, 20 breaths/min?
   a. Blood products
   b. Catecholamines
   c. Potassium
   d. Hydrocortisone

5. Which finding should you anticipate on the physical examination of a patient with Cushing's syndrome?
   a. Blood glucose, 180 mg/dL (10 mmol/L)
   b. Blood pressure, 94/54 mm Hg
   c. Heart rate, 50 bpm
   d. Thin face and body profile

6. When serum glucose levels drop below 70 mg/dL (3.9 mmol/L), which of the following occurs?
   a. Epinephrine secretion increases.
   b. Glucagon secretion decreases.
   c. Growth hormone secretion increases.
   d. Insulin production increases.

7. A 22-year-old man complains of a 2-day history of abdominal pain. His skin is flushed, and he has a fruity odor on his breath. Assessment reveals a blood pressure of 106/54 mm Hg; pulse rate, 128 beats/min; respirations, 28 breaths/min; and glucose level, 568 mg/dL. Your highest-priority intervention would be to:
   a. Administer glucagon IM.
   b. Perform endotracheal intubation.
   c. Infuse normal saline rapid IV.
   d. Perform a 12-lead electrocardiogram.

8. Which patient would be an appropriate candidate for immediate intravenous administration of sodium bicarbonate?
   a. A 22-year-old who is unresponsive after a heroin overdose with respirations of 8 breaths/min
   b. A 22-year-old with anxiety, a racing heart rate, and tachypnea
   c. A 34-year-old with nausea, vomiting, and diarrhea of 4 days' duration, who has shallow respirations and mild confusion
   d. A 45-year-old who initially complained of chest pain and is now in cardiac arrest and unresponsive to treatment

9. A 72-year-old complains of a headache and being depressed, intermittent twitching in the facial muscles, and general weakness over the past 2 weeks. She has a medical history of hypoparathyroidism. The ECG reveals a prolonged QT segment. During transport she has a seizure. Which electrolyte imbalance is most likely?
   a. Hypocalcemia
   b. Hyperkalemia
   c. Hypercalcemia
   d. Hyponatremia

10. A 10-year-old boy presents with lethargy. His vital signs include a blood pressure of 106/70 mm Hg; pulse rate, 140 beats/min; respirations, 32 breaths/min; oxygen saturation, 98%; and temperature, 99.0°F (37.2°C). Which of the following is most consistent with a diagnosis of DKA?
    a. Glucose 406 mg/dL; \text{ETCO}_2, 40 mm Hg
    b. Glucose 806 mg/dL; \text{ETCO}_2, 60 mm Hg
    c. Glucose 806 mg/dL; \text{ETCO}_2, 35 mm Hg
    d. Glucose, 406 mg/dL; \text{ETCO}_2, 25 mm Hg