Perspective

Syncope is the sudden transient loss of consciousness with a loss of postural tone. It is a common presenting complaint in the emergency department (ED). Despite improved understanding of risk and outcomes, consensus on the diagnostic approach and disposition remains elusive. This is in part because of the varied causes of syncope and lack of definitive diagnostic studies, and in part because of confusion and lack of standard terminology to describe the disorder. Diagnostic accuracy relies largely on the synthesis of patient risk factors and reported symptoms, with limited reliance on the physical examination and ancillary testing.

Epidemiology

The prevalence of syncope in the general population is approximately 19%. Patients come to the ED at a rate of 2.8 visits per 1000 population, which accounts for 0.8% of ED visits. Approximately 32% of these patients are admitted, and syncope accounts for 1 to 6% of all hospitalized patients. Persons aged 65 years and older account for 80% of such admissions. In the pediatric population, 15% experience at least one episode of syncope. Risk factors for syncope include cerebrovascular disease, cardiac medications, and hypertension. Most causes of syncope are benign and have favorable outcomes. Patients with preexisting cardiovascular disease and syncope from any cause are at the greatest short- and long-term risk of mortality. Age, congestive heart failure, and coronary artery disease are key predictors of mortality in patients with syncope, but syncope alone does not appear to alter risk. In contrast, there is no increased risk of cardiovascular morbidity or mortality associated with neurocardiogenic (vasovagal), orthostatic, and medication-related syncope. Recurrence of syncope may be as high as 50% and is not correlated with age or sex.

Benign causes of syncope predominate in adolescents and young adults. Approximately 30% of athletes who have died during exercise, however, had a prior episode of syncope as a sentinel event. Prospective outcome studies in children are lacking, but most reports suggest that mortality rates are extremely low. Significant trauma may result from syncope and can contribute to increased risk of morbidity and mortality, particularly in the elderly. The overall U.S. medical cost of syncope is estimated at $2.4 billion annually.

Pathophysiology

The final common pathway resulting in syncope is dysfunction of either both cerebral hemispheres or the brainstem (reticular activating system), usually from acute hypoperfusion. Reduced blood flow may be regional (cerebral vasoconstriction) or systemic (hypotension). Loss of consciousness results in loss of postural tone, with the resulting syncopal episode. Less severe derangements may result in sensations of presyncope or light-headedness. In this fashion, presyncope and syncope may be considered on a continuum with shared etiologies and mechanisms. By definition, syncope is transient; therefore the cause of central nervous system (CNS) dysfunction should likewise be transient. Persistent causes of significant CNS dysfunction result in coma or depressed consciousness (see Chapter 16).

Hypoperfusion resulting in approximately 35% or more reduction in cerebral blood flow usually produces unconsciousness, and any mechanism that adversely affects the components of perfusion (cardiac output, systemic vascular resistance, blood volume, regional vascular resistance) can cause or contribute to syncope. Other mechanisms of CNS dysfunction resulting in syncope include hypoglycemia, toxins, metabolic abnormalities, failure of autoregulation, and primary neurologic derangements.

Diagnostic Approach

Differential Considerations

The potential causes of syncope are numerous and can be categorized according to their primary mechanism (Box 15-1). The first differential diagnostic consideration is to distinguish syncope from other causes of an apparent sudden loss of consciousness, especially seizure and uncommon disorders such as cataplexy. When syncope is established as the working diagnosis, the life-threatening causes, primarily cardiovascular in origin, are considered first. The principal serious causes of syncope are dysrhythmias and myocardial ischemia. Cerebrovascular disease, principally subarachnoid hemorrhage, is less frequently encountered, but equally serious. Toxic-metabolic abnormalities may induce syncope through alterations in blood pressure or cardiac rhythm. Structural cardiac lesions, such as critical aortic stenosis, and sudden interruption of right ventricular outflow by pulmonary embolism can also cause sudden loss of consciousness.
Dissection of the thoracic aorta rarely manifests primarily as syncope but is potentially catastrophic.

### Pivotal Findings

The majority of cases of syncope arise from benign causes, so the evaluation is largely focused on excluding serious pathology. The patient’s history, particularly the setting of the syncope (e.g., postmicturition, venipuncture), patient position (e.g., sitting, standing, supine), prior episodes, and the presence or absence of prodromal symptoms, is central to separating benign from serious causes of the syncopal episode. Young, healthy patients with clearly benign syncope disclosed by a thorough history require little more than a physical examination for anemia or other benign precipitating factors. The yield of an electrocardiogram (ECG) is generally low; however, it is broadly recommended and has the additional advantages of being noninvasive and relatively inexpensive. The clinical examination (history and physical examination) alone can suggest the diagnosis in 45% of cases. For a large portion of the remainder, however, a clear diagnosis for the syncope may not be established in the ED.

### Symptoms

Symptoms can often suggest the diagnosis, although the relative weight of the history diminishes in older patients and in those not able to remember clearly the events leading up to the loss of consciousness. The patient is asked to describe the character of the syncopal event. Witnesses may be able to supplement and corroborate the patient’s incomplete recall, and that history should be solicited. Key characteristics include the rate of onset (gradual or abrupt), position on symptom onset (e.g., standing, sitting, or supine), and duration and rate of recovery. Abrupt onset, occurrence while sitting or supine, and duration of more than a few seconds are usually ascribed to serious, often cardiac, causes of syncope, but firm data are lacking. Similarly, incomplete or near-syncpe may be less serious, but at least one study suggests that onset associated with a prodrome or presyncope may herald serious etiology. Significant emotional events, micturition, eating, bowel movements, emesis, and movement or manipulation of the neck causing exposure to heat stress suggests orthostasis. The myriad mechanisms that may mediate a neurocardiogenic response, including significant emotional events, micturition, eating, bowel movements, emesis, and movement or manipulation of the neck causing stimulation of the carotid sinus, should be addressed. Occurrence in supine position or the presence of acute palpitations is relatively specific for syncope of cardiac origin. Seizures may be preceded by an aura and followed by a typical postictal state. Events during the syncopal episode do not usually clarify the cause. Tonic-clonic movements, related to inadequate cerebral perfusion, can occur in any form of syncope, including benign neurocardiogenic syncope, and should be differentiated from the

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### Causes of Syncope

**Focal Hypoperfusion of CNS Structures**
- Cerebrovascular disease
- Hyperventilation
- Subclavian steal
- Subarachnoid hemorrhage
- Basilar artery migraine
- Cerebral syncope

**Systemic Hypoperfusion Resulting in CNS Dysfunction**
- Outflow obstruction
  - Mitral, aortic, or pulmonic stenosis
  - Hypertrophic cardiomyopathy
- Atrial myxoma
- Pulmonary embolism
- Pulmonary hypertension
- Cardiac tamponade
- Congenital heart disease
- Reduced cardiac output
- Tachycardia
  - Supraventricular tachycardia
  - Ventricular tachycardia
  - Ventricular fibrillation
- Wolff-Parkinson-White syndrome
- Torsades de pointes
- Bradycardia
  - Sinus node disease
  - Second-degree and third-degree AV block
- Prolonged QT syndrome
- Brugada’s syndrome
- Pacemaker malfunction
- Implanted cardioverter-defibrillator malfunction
- Other cardiovascular disease
  - Aortic dissection
  - Myocardial infarction
  - Cardiomyopathy

**CNS Dysfunction with Normal Cerebral Perfusion**
- Hypoglycemia
- Hypoxemia—asphyxiation
- Seizure
- Narcolepsy
- Psychogenic
  - Anxiety disorder
  - Conversion disorder
  - Somatization disorder
  - Panic disorder
  - Breath-holding spells
- Toxic
  - Drugs
  - Carbon monoxide
  - Other agents
- Undetermined causes

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AVA, atrioventricular; CNS, central nervous system.
The postsyncope events should be queried. Symptoms consistent with a postictal state are characteristic of seizures. Initial vital signs and cardiac monitoring by out-of-hospital medical providers may provide clues to primary cardiac dysrhythmias. Associated symptoms can offer potentially important clues. Chest pain or dyspnea can suggest myocardial ischemia, aortic dissection, or pulmonary embolism. Diaphoresis and light-headedness are nonspecific, but if prominent and accompanied by a graying of vision may suggest orthostasis or vasovagal causing. Tongue biting and incontinence of urine or stool suggest seizures.

The past medical history is critical in stratifying risk. Congestive heart failure is a key determinant of increased short- and long-term mortality in the setting of syncope. Prior coronary artery or cerebrovascular disease, diabetes, hypertension, and other significant chronic disease also appear to increase the risk of mortality after syncope.

Certain medications are well established to be associated with syncope (Box 15-2). QT interval–prolonging agents, beta-blockers, insulin, and oral hypoglycemics, in particular, deserve attention because of the likelihood of repeated syncope without careful medication monitoring.

### Signs

The physical examination focuses primarily on the elements affecting the cardiovascular and neurologic systems. Specific findings are detailed in Table 15-1. Signs of orthostasis should be sought in all cases in which this mechanism is suggested. Carotid massage is both safe and occasionally revealing; it is indicated in cases in which the history is suggestive of carotid sinus hypersensitivity. Rectal examination for gross blood or melena is recommended if anemia or gastrointestinal hemorrhage is suspected.

### Ancillary Studies

The chief diagnostic adjunct in evaluating syncope is the 12-lead ECG (Table 15-2). It is warranted in all cases of syncope except in young, otherwise healthy patients with a clear history and setting for benign neurocardiogenic (vasovagal) syncope. Dysrhythmias and shortened PR or prolonged QT intervals may be identified on the 12-lead ECG. A right bundle branch block in association with ST elevation in leads V1 through V3 suggests Brugada’s syndrome. Unanticipated cardiac hypertrophy may be revealed. Continuous limb–lead ECG monitoring in the ED may also identify transient dysrhythmias. An ECG showing right ventricular strain pattern may suggest pulmonary embolism, whereas diffuse ST elevation or electrical alternans helps diagnose pericarditis associated with pericardial tamponade.

#### Table 15-1 Directed Physical Examination in Syncope

<table>
<thead>
<tr>
<th>SYSTEM</th>
<th>PIVOTAL FINDING</th>
<th>SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital signs</td>
<td>Pulse rate and rhythm</td>
<td>Tachycardia, bradycardia, other dysrhythmias</td>
</tr>
<tr>
<td></td>
<td>Respiratory rate and depth</td>
<td>Tachypnea suggests hypoxia, hyperventilation, or pulmonary embolus</td>
</tr>
<tr>
<td></td>
<td>Blood pressure</td>
<td>Shock may cause decreased cerebral perfusion; hypovolemia or medication use may lead to orthostasis</td>
</tr>
<tr>
<td></td>
<td>Temperature</td>
<td>Fever from sepsis may cause volume depletion and orthostasis</td>
</tr>
<tr>
<td>Skin</td>
<td>Color, diaphoresis</td>
<td>Signs of decreased organ perfusion</td>
</tr>
<tr>
<td>HEENT</td>
<td>Tenderness and deformity, Papilledema</td>
<td>Signs of trauma</td>
</tr>
<tr>
<td></td>
<td>Breath</td>
<td>Increased intracranial pressure, head injury</td>
</tr>
<tr>
<td></td>
<td>Ketones from ketoacidosis</td>
<td></td>
</tr>
<tr>
<td>Neck</td>
<td>Bruits</td>
<td>Identify presence of cerebrovascular disease</td>
</tr>
<tr>
<td></td>
<td>Jugular venous distention</td>
<td>Right-sided heart failure from myocardial ischemia, tamponade, pulmonary embolism</td>
</tr>
<tr>
<td>Lungs</td>
<td>Breath sounds, crackles, wheezes</td>
<td>Infection, left-sided heart failure from myocardial ischemia, rarely pulmonary embolism</td>
</tr>
<tr>
<td>Heart</td>
<td>Systolic murmur</td>
<td>Aortic stenosis, hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>Rub</td>
<td>Pericarditis, tamponade</td>
</tr>
<tr>
<td>Abdomen</td>
<td>Pulsatile mass</td>
<td>Abdominal aortic aneurysm</td>
</tr>
<tr>
<td>Rectum</td>
<td>Stool for gross blood or melena</td>
<td>Anemia, gastrointestinal bleed</td>
</tr>
<tr>
<td>Pelvis</td>
<td>Uterine bleeding, adnexal tenderness</td>
<td>Anemia, ectopic pregnancy, hypovolemia</td>
</tr>
<tr>
<td>Extremities</td>
<td>Pulse equality in upper extremities</td>
<td>Subclavian steal, thoracic aortic dissection</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Mental status, focal neurologic findings</td>
<td>Seizure, stroke, or other primary neurologic disease</td>
</tr>
</tbody>
</table>

*HEENT, head, eyes, ears, nose, and throat.*
neurologic events rarely are the cause of syncope. Cranial com-

Kenney. Other than seizure or intracranial hemorrhage, primary

Chest radiography and serum B-type natriuretic peptide (BNP)
or exclude some uncommon causes of syncope (see Table 15-2).

Glucose, serum or blood Hypoglycemia

D-dimer, serum Pulmonary embolism

Cardiac enzymes, serum Myocardial infarction

β-hCG Pregnancy

Toxicologic screen Drug-related syncope

Arterial blood gas Acid-base disturbance

Chest x-ray examination Thoracic aortic dissection

Cranial CT or MRI New-onset or focal seizure, trauma, intracranial hemorrhage

Echocardiogram Cardiac outflow obstruction, tamponade, thoracic dissection

Ventilation-perfusion scan Pulmonary embolism

CT pulmonary angiogram Pulmonary embolism, thoracic aortic dissection

Abdominal ultrasound or CT Abdominal aortic aneurysm

Pelvic ultrasound Ectopic pregnancy

Tests Usually Performed as Part of an Inpatient or Outpatient Evaluation

Holter or loop ECG Dysrhythmia

Echocardiogram Cardiomyopathy, valvular disease

Exercise and 99mTc sestamibi ECG Myocardial ischemia

Electrophysiologic study Dysrhythmia

Carotid ultrasound Stroke, TIA

Head-up tilt-table test Orthostatic hypotension

Electroencephalogram Seizures

Table 15-2 Ancillary Studies in Syncope

STUDY INDICATION

12-Lead ECG/limb-lead ECG monitoring Cardiac dysrhythmia, ischemia, cardiomyopathy

Orthostatic vital signs Orthostatic hypotension or bradycardia

Hemogram Anemia

Electrolytes, serum Metabolic abnormality, especially hypernatremia, hyperkalemia, or hypokalemia

Glucose, serum or blood Hypoglycemia

D-dimer, serum Pulmonary embolism

Cardiac enzymes, serum Myocardial infarction

β-hCG Pregnancy

Toxicologic screen Drug-related syncope

Arterial blood gas Acid-base disturbance

Chest x-ray examination Thoracic aortic dissection

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Carotid ultrasound Stroke, TIA

Head-up tilt-table test Orthostatic hypotension

Electroencephalogram Seizures

CT, computed tomography; ECG, electrocardiogram; hCG, human chorionic gonadotropin; MRI, magnetic resonance imaging; 99mTc, technetium 99m; TIA, transient ischemic attack.

Table 15-3 Critical Diagnoses to Consider in Syncope

Table 15-3 Critical Diagnoses to Consider in Syncope

<table>
<thead>
<tr>
<th>STUDY</th>
<th>INDICATION</th>
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<tr>
<td>12-Lead ECG/limb-lead ECG monitoring</td>
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</tr>
<tr>
<td>Orthostatic vital signs</td>
<td>Orthostatic hypotension or bradycardia</td>
</tr>
<tr>
<td>Hemogram</td>
<td>Anemia</td>
</tr>
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<td>Electrolytes, serum</td>
<td>Metabolic abnormality, especially hypernatremia, hyperkalemia, or hypokalemia</td>
</tr>
<tr>
<td>Glucose, serum or blood</td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>D-dimer, serum</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>Cardiac enzymes, serum</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>β-hCG</td>
<td>Pregnancy</td>
</tr>
<tr>
<td>Toxicologic screen</td>
<td>Drug-related syncope</td>
</tr>
<tr>
<td>Arterial blood gas</td>
<td>Acid-base disturbance</td>
</tr>
<tr>
<td>Chest x-ray examination</td>
<td>Thoracic aortic dissection</td>
</tr>
<tr>
<td>Cranial CT or MRI</td>
<td>New-onset or focal seizure, trauma, intracranial hemorrhage</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>Cardiac outflow obstruction, tamponade, thoracic dissection</td>
</tr>
<tr>
<td>Ventilation-perfusion scan</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>CT pulmonary angiogram</td>
<td>Pulmonary embolism, thoracic aortic dissection</td>
</tr>
<tr>
<td>Abdominal ultrasound or CT</td>
<td>Abdominal aortic aneurysm</td>
</tr>
<tr>
<td>Pelvic ultrasound</td>
<td>Ectopic pregnancy</td>
</tr>
</tbody>
</table>

Tests Usually Performed as Part of an Inpatient or Outpatient Evaluation

Holter or loop ECG | Dysrhythmia |

Echocardiogram | Cardiomyopathy, valvular disease |

Exercise and 99mTc sestamibi ECG | Myocardial ischemia |

Electrophysiologic study | Dysrhythmia |

Carotid ultrasound | Stroke, TIA |

Head-up tilt-table test | Orthostatic hypotension |

Electroencephalogram | Seizures |

CT, computed tomography; ECG, electrocardiogram; hCG, human chorionic gonadotropin; MRI, magnetic resonance imaging; 99mTc, technetium 99m; TIA, transient ischemic attack.

Routine hematologic and urine studies have limited usefulness in the evaluation of syncope and are generally not indicated.21 When suggested by the history and physical examination findings, however, selective use of the hemogram, serum electrolytes and glucose, urine drug screen, and pregnancy test may identify or exclude some uncommon causes of syncope (see Table 15-2). Chest radiography and serum B-type natriuretic peptide (BNP) testing are warranted if heart failure is suspected or known by history. Other than seizure or intracranial hemorrhage, primary neurologic events rarely are the cause of syncope. Cranial computed tomography is indicated only when intracerebral hemorrhage is suspected on the basis of sudden syncope with accompanying headache, particularly of sudden onset, or in the presence of abnormalities on neurologic examination.22

In otherwise healthy patients in whom a benign dysrhythmia, such as episodic supraventricular tachycardia or atrial fibrillation, is suspected, Holter or event ECG monitoring may be helpful. In patients with significant underlying cardiac disease or when a significant dysrhythmia is a possible cause of the syncope, echocardiography, continuous monitoring, or cardiovascular stress testing may be helpful in the inpatient or ED observation unit setting. Depending on the results of initial evaluation, electrophysiologic studies or magnetic resonance imaging may be indicated. Electroencephalography is useful only when a seizure episode is suspected. Tilt table testing, although infrequently used in the United States, may have diagnostic value in elderly patients and children in whom chronic orthostatic hypotension is possible.15 Orthostatic vital signs, although unreliable in evaluation of volume status, may be helpful when positional changes are accompanied by typical presyncopal symptoms and a significant rise in heart rate or fall in blood pressure.16,21 A schematic of selected diagnostic testing strategies for syncope is depicted in Figure 15-1.

DIAGNOSTIC ALGORITHM

The critical diagnoses to consider are listed in Table 15-3.

The emergent causes of syncope are protean and are included in Box 15-1. Many other causes such as neurocardiogenic and reflex-mediated syncope have benign mechanisms.

After stabilization and assessment, the clinical features coupled with onset and recovery can suggest the cause (Table 15-4). A logical approach to the history, physical examination, and diagnostic testing is depicted in Figure 15-2. The emphasis is on risk stratification because short-term mortality risk in syncope is related to structural cardiac disease, heart failure, and dysrhythmias.23

EMPIRICAL MANAGEMENT

Syncope is by definition a transient event, so most patients are asymptomatic on presentation. Patients with significantly abnormal vital signs, recurrent syncope, or associated symptoms of a concerning nature such as chest pain, hypotension, abdominal or back pain, or shortness of breath should undergo rapid evaluation.

Patients with critical diagnoses are stabilized in the ED and admitted to the intensive care unit (ICU) or other appropriate inpatient unit. Those with emergent diagnoses are typically admitted to telemetry units. Patients with nonemergent diagnoses can be treated as outpatients.

Several scoring systems have been proposed as aids in the admission decision-making process, most notably the San Francisco Syncope Rule.24 In essence, this guideline suggests that in the absence of abnormal ECG findings, shortness of breath, hypotension (systolic blood pressure <90 mm Hg), anemia (hematocrit <30%), or a history of heart failure, the patient is at sufficiently low risk for outpatient disposition to be considered. The safety and efficacy of the San Francisco Syncope Rule as well as other
### Table 15-4 Clinical Features of Common and Serious Causes of Syncope

<table>
<thead>
<tr>
<th>CAUSE</th>
<th>ONSET AND RECOVERY</th>
<th>FEATURES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysrhythmia</td>
<td>Classically abrupt onset and rapid recovery</td>
<td>Classic presentation uncommon; past cardiac history, risk factors for CAD more common in elderly; implanted pacemaker or cardioverter-defibrillator</td>
</tr>
<tr>
<td>Cardiac outflow obstruction</td>
<td>Exertion causes abrupt symptoms; rapid recovery with rest</td>
<td>Murmurs not always audible; mechanical valves warrant close monitoring</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>Exertion or at rest; recovery often incomplete with chest pain persisting</td>
<td>Past cardiac history, risk factors for CAD; chest pain and shortness of breath common but frequently absent in diabetics and the elderly</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>Abrupt onset; recovery often incomplete with dyspnea persisting</td>
<td>Chest pain, dyspnea, hypercoagulable state, DVT, pregnancy</td>
</tr>
<tr>
<td>Thoracic aortic dissection</td>
<td>Spontaneous; recovery often incomplete with chest or upper back pain persisting</td>
<td>Tearing chest pain; associated with hypertension, Marfan syndrome, cystic medial necrosis</td>
</tr>
<tr>
<td>Abdominal aortic aneurysm</td>
<td>Spontaneous onset; recovery often incomplete with abdominal pain persisting</td>
<td>Abdominal or low back pain; associated with peripheral vascular disease</td>
</tr>
<tr>
<td>Pericardial tamponade</td>
<td>Penetrating chest trauma or thoracic cancers</td>
<td>Beck’s triad of hypotension, JVD, muffled heart sounds</td>
</tr>
<tr>
<td>Anomalous left coronary artery</td>
<td>Onset with exercise, Valsalva maneuver</td>
<td>Left coronary artery arises from pulmonary artery; usually detected in childhood</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>Rapid onset; sentinel event may resolve</td>
<td>Focal neurologic findings; “thunderclap” worst headache; nuchal rigidity</td>
</tr>
<tr>
<td>Vertebrobasilar insufficiency</td>
<td>Posture change or neck movement</td>
<td>Vertigo, nausea, dysphagia, dysarthria, blurry vision common associated symptoms</td>
</tr>
<tr>
<td>Hypovolemia</td>
<td>Bleeding, emesis, heat stress, dehydration; gradual onset</td>
<td>Orthostatic hypotension</td>
</tr>
<tr>
<td>Anemia</td>
<td>Bleeding, often occult or gradual from menses or gastrointestinal sources; iron deficiency or decreased red blood cell production</td>
<td>Orthostatic hypotension commonly associated</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Gradual onset; incomplete spontaneous recovery common</td>
<td>Diabetes, ingestion or injection of hypoglycemics or insulin; diaphoresis, anxiety, jitteriness</td>
</tr>
<tr>
<td>Hypoxemia</td>
<td>Usually gradual onset; spontaneous recovery if asphyxiating circumstance is reversed</td>
<td>Carbon monoxide, natural gas, sewer gas, bleach-ammonia mix</td>
</tr>
<tr>
<td>Subdural hematoma</td>
<td>Onset with or after trauma (which may be trivial in high-risk patients)</td>
<td>Elderly, alcoholics, patients on anticoagulants at greater risk</td>
</tr>
<tr>
<td>Air embolus</td>
<td>Diving</td>
<td>Hyperbaric oxygen a key treatment</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>Associated with myocardial infarction or pulmonary embolus</td>
<td>Risk factors for myocardial infarction or pulmonary embolism</td>
</tr>
<tr>
<td>Drug syncope</td>
<td>Medication associated with syncope</td>
<td>Consider illicit and alternative drug use; elderly at risk for polypharmacy and drug interactions</td>
</tr>
<tr>
<td>Ruptured ectopic pregnancy</td>
<td>Patient often unaware of pregnancy</td>
<td>Abdominal pain, abnormal tenderness; positive β-hCG test</td>
</tr>
<tr>
<td>Seizure</td>
<td>Abrupt or with aura; postictal state common</td>
<td>Past history common</td>
</tr>
<tr>
<td>Carotid sinus sensitivity</td>
<td>Carotid sinus sensitivity; rapid onset and recovery</td>
<td>Shaving, necktie, sudden neck movement; carotid massage may provoke symptoms</td>
</tr>
<tr>
<td>Reflex syncope</td>
<td>Gastrointestinal, genitourinary, or thoracic stimulation</td>
<td>Urination, defecation, cough, eating, swallowing, weightlifting</td>
</tr>
<tr>
<td>Neurocardiogenic (vasovagal)</td>
<td>Emotion, pain are common triggers; upright posture; gradual onset; rapid recovery once supine</td>
<td>Prodrome of light-headedness, graying or blurring of vision, nausea, sweats common</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>Emotion, pain; gradual onset; patient often unaware of rapid respirations</td>
<td>Perioral tingling, carpopedal spasms, extremity numbness</td>
</tr>
<tr>
<td>Narcolepsy</td>
<td>Often spontaneous</td>
<td>Known history</td>
</tr>
<tr>
<td>Basilar artery migraine</td>
<td>Specific triggers often known to patient</td>
<td>Visual prodrome often absent; more common in young women; vertigo and nausea common</td>
</tr>
<tr>
<td>Trigeminal or glossopharyngeal neuralgia</td>
<td>Sudden onset; specific triggers often known to patient</td>
<td>Lancinating pain in characteristic location</td>
</tr>
<tr>
<td>Subclavian steal</td>
<td>Moving affected arm</td>
<td>Thoracic outlet syndrome</td>
</tr>
<tr>
<td>Psychogenic</td>
<td>Variable</td>
<td>Anxiety or psychiatric history; diagnosis by examining symptom pattern and excluding organic cause</td>
</tr>
<tr>
<td>Breath-holding</td>
<td>Deliberate breath-holding</td>
<td>Usually toddlers or young children</td>
</tr>
<tr>
<td>Drop attack</td>
<td>Unpredictable</td>
<td>Not true syncope—no loss of consciousness; usually elderly; loss of tone, ataxia, vertigo</td>
</tr>
</tbody>
</table>

*CAD,* coronary artery disease; *DVT,* deep vein thrombosis; *hCG,* human chorionic gonadotropin; *JVD,* jugular venous distention; *TIA,* transient ischemic attack.
Figure 15-1. Management algorithm for patients with syncope.
The ED evaluation of syncope is often inconclusive. After a history, physical examination, and 12-lead ECG, up to 50% of patients do not have a firm diagnosis. Patients younger than 45 years and without worrisome symptoms, signs, or ECG findings are generally at very low risk for adverse outcome and may often be treated as outpatients. Discharged patients should be warned of the hazards of recurrent syncope occurring during activities such as driving or working at heights.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.
References