Chapter 52: Syncope

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FIGURE 52-1.

INTRODUCTION AND EPIDEMIOLOGY

Syncope or fainting is a symptom complex consisting of a brief loss of consciousness associated with an inability to maintain postural tone that spontaneously resolves without medical intervention. Syncope accounts for approximately 1% to 2% of ED visits each year and up to 6% of hospital admissions.[1,2,3] In the **Framingham Heart Study**, 7814 patients were followed for 17 years, and 10.5% reported syncope.[4] Syncope in the preceding year is the best predictor of recurrence.[5] It can affect the young and the old, with the elderly having the greatest morbidity.[6] **Near-syncope**, a premonition of fainting without loss of consciousness, shares the same basic pathophysiologic process as syncope and may carry the same risks.[7,8]

PATHOPHYSIOLOGY

The final common pathway of syncope is the same regardless of the underlying cause: about 10 seconds of complete disruption of blood flow or nutrient delivery to both cerebral cortices or to the brainstem reticular activating system, or reduction of cerebral perfusion by 35% to 50%. Most commonly, an inciting event causes a drop in cardiac output, which decreases oxygen and substrate delivery to the brain. Less commonly, vasospasm reduces CNS blood flow. Cerebral perfusion and consciousness are restored by the supine position, the response of autonomic autoregulatory centers, or restoration of a perfusing cardiac rhythm.

The causes of syncope are numerous (**Table 52-1**). The major causes of syncope identified in the Framingham Heart Study were vasovagal (reflex mediated, 21%), cardiac (10%), orthostatic (9%), medication related (7%), neurologic (4%), and unknown (37%).[4] In most studies, even with exhaustive patient evaluation, the cause remains unknown in about 40% of individuals.[9,10] After ED investigation, the unknown proportion may be 50% to 60%. Diagnosis is important, because each diagnostic classification carries with it prognostic risk. In the Framingham study, cardiac syncope doubled the risk of death, neurologic syncope increased the risk of death by 50%, and syncope of unknown cause increased the risk of death by 30%, compared to the general population cohort of the study. Individuals with neurally/reflex-mediated or vasovagal syncope had no increased risk of death compared with the general population cohort.[4]

TABLE 52-1

Causes of Syncope

Cardiac*	Neural/Reflex Mediated
Structural cardiopulmonary disease Valvular heart disease Aortic stenosis Tricuspid stenosis Mitral stenosis Cardiomyopathy Pulmonary hypertension Congenital heart disease Myxoma Pericardial disease Aortic dissection Pulmonary embolism Myocardial ischemia Myocardial infarction *Dysrhythmias* Bradydysrhythmias Short or long QT syndromes Stokes-Adams attack Sinus node disease Second- or third-degree heart block Pacemaker malfunction Tachydysrhythmias Ventricular tachycardia Torsade de pointes Supraventricular tachycardia Atrial fibrillation or flutter	*Vasovagal* *Situational* Cough Micturition Defecation Swallow Neuralgia *Carotid sinus syndrome* **Other** **Orthostatic hypotension** (see text) **Psychiatric** **Neurologic** Transient ischemic attacks Subclavian steal Migraine **Medications (Table 52-2)** **Breath holding** (pediatric)*

*See chapter 131, Seizures and Status Epilepticus in Children, and chapter 165, Seizures and Status Epilepticus in Adults.

CLINICAL FEATURES

CARDIAC-RELATED SYNCOPE

Cardiac-related syncope is the most dangerous type and is a risk for sudden death. Because patients with documented cardiac syncope have a 6-month mortality rate that exceeds 10%, timely and thorough evaluation is warranted.[4,10] Well-appearing patients with undiagnosed cardiac disease are the most challenging group. The

causes of cardiac syncope are divided into two categories: structural disease and dysrhythmias (**Table 52-1**). In both settings, the heart is unable to provide adequate cardiac output to maintain cerebral perfusion.

Syncope can occur if **structural disease** limits the heart's ability to increase cardiac output to meet demand. Examples of structural cardiac disease associated with syncope include aortic stenosis, hypertrophic cardiomyopathy, pulmonary embolism, and myocardial infarction. **Consider aortic stenosis as a structural cardiac cause of syncope in the elderly.** The classic symptom constellation of aortic stenosis is chest pain, dyspnea on exertion, and syncope. **Hypertrophic cardiomyopathy** is characterized by a stiff noncompliant left ventricle, diastolic dysfunction, and outflow tract obstruction. It is the most common cause of sudden cardiac death in young adults, but the disorder may be first recognized in those >60 years old.[11] Massive acute **pulmonary embolism** may cause syncope due to obstruction of the pulmonary vascular bed and reduction in cardiac output.[12] **Acute myocardial infarction** may cause syncope if myocardial dyskinesia reduces cardiac output. Individual chapters in the Cardiovascular Disease section of this text provide more discussion on structural cardiopulmonary disorders that may cause syncope.

Although both **brady- and tachydysrhythmias** may lead to transient cerebral hypoperfusion (**Table 52-1**), there is no absolute high or low heart rate that will predictably produce syncope. Symptoms depend on both the autonomic nervous system's ability to compensate for a decrease in cardiac output and the degree of underlying cerebrovascular disease. Dysrhythmias may also result from of a primary electrolyte imbalance, as in hypomagnesemia (e.g., torsade de pointes). Dysrhythmias can occasionally occur in structurally normal hearts, such as in the familial disorders of **Brugada syndrome**, long or short **QT syndromes**, and catecholamine-associated polymorphic ventricular tachycardia. **Syncope from dysrhythmias is typically sudden and usually without prodromal symptoms.**

VASOVAGAL AND NEURALLY/REFLEX-MEDIATED SYNCOPE

Vasovagal syncope, a form of reflex-mediated or neurally mediated syncope, is associated with inappropriate vasodilatation, bradycardia, or both, as a result of inappropriate vagal or sympathetic tone.[13,14] A prodrome of lightheadedness, with or without nausea, pallor, and/or sweating, and an associated feeling of warmth may accompany vasovagal syncope. **A slow, progressive onset with associated prodrome suggests vasovagal syncope**. Vasovagal syncope may occur after exposure to an unexpected or unpleasant sight, sound, or smell; fear; severe pain; emotional distress; or instrumentation. It may also occur in association with prolonged standing or kneeling in a crowded or warm place. **Situational syncope** occurs during or immediately after coughing, micturition, defecation, or swallowing.

Carotid sinus hypersensitivity, characterized by bradycardia or hypotension, is another type of reflex-mediated syncope. The carotid body, located at the carotid bifurcation, contains pressure-sensitive receptors. The stimulation of an abnormally sensitive carotid body by external pressure may lead to two autonomic responses. Most commonly, there is an abnormal vagal response, leading to bradycardia and asystole of >3 seconds. Less commonly, there is a vasodepressor response, leading to a decrease in blood pressure of >50 mm Hg without a significant change in heart rate. Both responses may occur simultaneously. Carotid sinus hypersensitivity is more common in men, the elderly, and among those with ischemic heart disease, hypertension, and certain head and neck malignancies. Although some patients may demonstrate a hypersensitive carotid sinus response on provocative testing, unless this response culminates in syncope or recurrence of prodromal symptoms, and unless it is associated with an inciting event, such as shaving or turning

of the head, it cannot be definitely diagnosed as the cause of syncope. About 25% of patients with carotid sinus hypersensitivity have true carotid sinus syndrome with spontaneous symptoms.[5] **Consider carotid sinus hypersensitivity in older patients with recurrent syncope and negative cardiac evaluations.**

ORTHOSTATIC SYNCOPE

Orthostatic syncope is suggested when postural hypotension is associated with syncope or presyncope.[15] When a person assumes an upright posture, gravity shifts blood to the lower part of the body, and cardiac output drops. This change triggers the healthy autonomic nervous system to increase sympathetic output and decrease parasympathetic output, increasing heart rate and peripheral vascular resistance, and thus increasing cardiac output and blood pressure.[16,17] If the autonomic response is insufficient to counter the drop in cardiac output upon standing, decreased cerebral perfusion and syncope may follow. Symptom onset is usually within the first 3 minutes after assuming the upright posture, but may be more delayed in some patients. However, positive orthostatic changes have been documented in up to 40% of asymptomatic patients >70 years old and in about a quarter of those <60 years old, so orthostasis does not always result in syncope.[14,18] Causes of orthostatic syncope include intravascular volume loss and poor vascular tone caused by α-receptor disorders or medications. **Many serious causes of syncope may be associated with orthostatic changes, so consider other life-threatening causes before attributing syncope to orthostasis, especially in the elderly.**

PSYCHIATRIC DISORDERS

Psychiatric disorders are found in a modest percentage of patients with syncope[19]—up to 40% of those with vasovagal syncope and up to 62% of those with unexplained syncope.[20] In one study, the most frequent psychiatric diagnoses associated with syncope were generalized anxiety disorder and major depressive disorder.[21] Hyperventilation has been used as a provocative maneuver in diagnosing panic disorder and generalized anxiety disorders and can lead to hypocarbia, cerebral vasoconstriction, and syncope.[22] Hyperventilation may not be obvious to the observer but can be documented by end-tidal carbon dioxide monitoring. In general, a patient with syncope and a psychiatric disorder is likely to be young, with repeated episodes of syncope and multiple prodromal symptoms.[21] A psychiatric cause for syncope should be one of exclusion, assigned only after organic causes have been excluded.

NEUROLOGIC SYNCOPE

Neurologic causes of syncope are rare. To meet the definition of syncope, symptoms must be transient and with no persistent neurologic deficits. **Thus, patients with loss of consciousness with persistent neurologic deficits or altered mental status do not have true syncope.** Brainstem ischemia, vertebrobasilar atherosclerotic disease, or basilar artery migraine may result in a decrease in blood flow to the reticular activating system, leading to sudden, brief episodes of loss of consciousness. Loss of consciousness is typically preceded by other signs or symptoms, such as diplopia, vertigo, focal neurologic deficits, or nausea. **Subclavian steal syndrome** is a rare cause of brainstem ischemia. It is characterized by an abnormal narrowing of the subclavian artery proximal to the origin of the vertebral artery, so that with exercise of the ipsilateral arm, blood is shunted, or "stolen," from the vertebrobasilar system to the subclavian artery supplying the arm muscles. Anatomically, narrowing is more common on the left. Physical examination may identify decreased pulse volume and diminished blood pressure in the affected arm.

Subarachnoid hemorrhage may present with syncope but is usually accompanied by other symptoms such as focal neurologic deficits, headache, or persistent altered mental status. The mechanism for syncope is thought to be an increase in intracranial pressure with a decrease in cerebral perfusion pressure. **Subarachnoid hemorrhage can also follow a fall and head injury from syncope secondary to another cause.** See chapter 166, Spontaneous Subarachnoid and Intracerebral Hemorrhage, for further discussion.

Seizure may be confused with syncope, because brief tonic-clonic movements are often associated with syncope. However, confusion (postictal state) lasting several minutes, tongue biting, incontinence, or an epileptic aura suggests a seizure.

MEDICATION-INDUCED SYNCOPE

Medications may contribute to syncope by a variety of means (**Table 52-2**), but the most common is orthostasis.[23] β-Blockers or calcium channel blockers may lead to a blunted heart rate response after orthostatic stress. Diuretics may produce volume depletion, and some medications have proarrhythmic properties, increasing the concern for dysrhythmia as the cause of syncope.

TABLE 52-2

Drugs Commonly Implicated in Syncope

Erectile dysfunction drugs
Antihypertensives
β-Blockers
Cardiac glycosides
Diuretics
Antidysrhythmics
Antipsychotics
Antiparkinsonism drugs
Antidepressants
Phenothiazines
Nitrates
Alcohol
Cocaine

PRINCIPLES OF EVALUATION

The goal of ED evaluation is to identify those at risk for immediate and future morbidity or sudden death. For patients with a specific diagnosis, the diagnosis directs the disposition plan. For patients without a specific diagnosis, risk stratification is based on a careful history, thorough physical examination, and electrocardiogram interpretation, with additional testing as needed.

HISTORY

Obtain clinical history from the patient and any witnesses of the event. Begin with a detailed description of the events preceding the loss of consciousness, including patient position, environmental stimuli, strenuous activity, or arm exercise. Record premonitory symptoms such as headache, diplopia, vertigo, or focal weakness. Ask about chest pain and palpitations. Clarify the duration of loss of consciousness and symptoms occurring after regaining consciousness. Symptoms associated with syncope that should raise concern of an immediately life-threatening diagnosis include **chest pain** (acute myocardial infarction, aortic dissection, pulmonary embolism, aortic stenosis), **palpitations** (dysrhythmia), shortness of breath (pulmonary embolism, congestive heart failure), **headache** (subarachnoid hemorrhage), and **abdominal or back pain** (leaking abdominal aortic aneurysm, ruptured ectopic pregnancy). A sudden event without warning and events associated with exertion raise suspicion for a cardiac dysrhythmia or structural cardiopulmonary lesion.[24] Ask about antecedent illness and alcohol ingestion or substance abuse. The past medical history should include questions regarding underlying structural heart disease, including congenital heart disease, valvular heart disease, coronary artery disease, congestive heart failure, pulmonary embolism, and ventricular dysrhythmias. Document any prior history of syncope, as patients with more than five syncopal episodes in 1 year are more likely to have vasovagal syncope or a psychiatric diagnosis than dysrhythmia as the cause.[5] All medications should be recorded, including over-the-counter medications such as laxatives. Patients aggressively dieting to lose weight may have electrolyte disturbances or may be taking amphetamine-like medications. The family history is important in regard to history of prolonged QT syndrome, dysrhythmias, sudden cardiac death, or other cardiac risks.

Special attention should be paid to patients presenting after single-car motor vehicle crashes (frequently with a history of driving off the road), particularly if the patients are elderly. Clinicians may become preoccupied by the trauma evaluation and miss the possibility of a syncopal event.

Seizure is the most common event mistaken as syncope. Mild, brief, tonic-clonic activity ("convulsive syncope") may accompany syncope of any etiology. The two conditions do not share the same pathophysiologic mechanisms. History is very important in differentiating syncope from seizure.[25] Premonitory and postevent symptoms may assist in differentiation. A classic aura or postictal confusion and muscle pain indicate seizure, whereas characteristic prodromal symptoms of nausea and diaphoresis suggest reflex-mediated (vasovagal) syncope. Witness information of the event may also be useful. Witnessed head turning or unusual posture during the event is consistent with seizure. A prolonged postictal phase is more common with seizure. Urinary incontinence is not useful in the distinction.

PHYSICAL EXAMINATION

Evidence of trauma without defensive injuries to the hands or knees should raise suspicion of a sudden event without warning, such as a dysrhythmia, but patients with noncardiac syncope are also just as likely to suffer significant facial and head trauma. The physical examination should focus on both the cardiovascular and neurologic systems. Obtain blood pressure measurements in both arms. Unequal blood pressures should increase suspicion of aortic dissection or subclavian steal. Take orthostatic blood pressures after 5 minutes in the supine position. Repeat measurements after 1 and 3 minutes of standing. A symptomatic decrease of >20 mm Hg in the systolic pressure is considered abnormal, as is a drop in pressure below 90 mm Hg independent of the development of symptoms. Cardiac examination may reveal the murmur of hypertrophic cardiomyopathy or aortic stenosis. The neurologic examination may uncover findings of focal neurologic disease or evidence of autonomic instability such as peripheral neuropathy. Perform rectal examination to check stool guaiac to evaluate for GI bleeding.

DIAGNOSIS

The diagnosis of syncope is clinical, with careful evaluation of the presentation and selected use of diagnostic tests. History is most important, and most diagnostic tests have low diagnostic yield.[26] The differential diagnosis is presented in **Tables 52-1** and **52-2**.

ELECTROCARDIOGRAM

Obtain a 12-lead electrocardiogram. Even though the electrocardiogram leads to a diagnosis in only a few patients, it is a simple, noninvasive test and is important for risk stratification.[27] Assess the electrocardiogram for evidence of prior cardiopulmonary disease, acute ischemia or new electrocardiogram changes, dysrhythmia, heart block, and prolonged or short QTc interval. A prolonged QTc interval has a variable definition, but the literature suggests it is defined as >470 milliseconds, with >500 milliseconds associated with significant outcomes,[28,29,30] whereas a short QTc interval <350 milliseconds is concerning as well.[31] New or old left bundle conduction abnormalities (left bundle-branch block, posterior or anterior fascicular block, QRS widening) are 3.5 times more likely to be associated with morbidity than electrocardiograms lacking these findings. Non-sinus rhythms are 2.5 times more likely to be associated with morbidity than sinus rhythms.[32] For further discussion, see chapter 18, Cardiac Rhythm Disturbances.

LABORATORY TESTING

Laboratory testing is directed by results of the history and physical examination. For example, a patient with orthostatic symptoms and a heme-positive stool test warrants at least a CBC. A reproductive-age female should have a urine pregnancy test. A transitory, wide anion gap acidosis follows a generalized seizure but is not present in simple syncope. Serum electrolytes rarely determine the cause of syncope. B-type natriuretic peptide or pro–B-type natriuretic peptide levels appear to be predictive of those at risk for morbidity. One study suggests that a level >300 pg/mL in the setting of syncope indicates risk,[33] but whether this adds any value over a history of congestive heart failure or structural disease is unclear.[34,35] See chapter 53, Acute Heart Failure, for a discussion of the diagnostic use of B-type natriuretic peptide.

ANCILLARY TESTING

Carotid Massage

Carotid massage is used to diagnose carotid sinus hypersensitivity in the patient with a history suggestive of carotid sinus syndrome. Although not generally used in emergency medicine at this time, carotid massage can be done at the bedside in the ED with continuous electrocardiographic and blood pressure monitoring, after obtaining informed consent. Each carotid body is separately massaged for 5 to 10 seconds. The test is considered positive if symptoms are reproduced in the presence of asystole >3 seconds or a decrease in systolic blood pressure of >50 mm Hg. **Do not perform carotid massage if the patient has known carotid stenosis, if bruits are present, if there is history of recent (<3 months) stroke or myocardial infarction, or if there is a history of ventricular tachycardia or fibrillation.** Neurologic deficits resulting from cardiac massage are rare, with deficits lasting more than 24 hours in approximately 0.1% of patients.[36] Only a small number of patients with carotid hypersensitivity will have the true carotid sinus syndrome. Given the small benefit

of the maneuver and that the rare potential adverse events are catastrophic, most physicians do not routinely perform carotid massage.

Hyperventilation Maneuver

A hyperventilation maneuver (open-mouthed, slow, deep breaths at a rate of 20 to 30 breaths per minute for 2 to 3 minutes) can be very useful in the young patient with undiagnosed syncope and suspected psychiatric illness. This test can easily be performed in the ED. A recurrence of prodromal symptoms or syncope significantly correlates with psychiatric (anxiety-provoking) causes of syncope.[21]

Neurologic Testing

When the history or physical examination does not suggest trauma or a neurologic cause for syncope, the clinical yield of routine CT scanning, electroencephalogram, or lumbar puncture is very low. Consequently, in asymptomatic patients who have experienced an isolated syncopal event, and in those without head trauma from the event, a head CT scan or MRI scan is not warranted.

DECISION MAKING AND RISK ASSESSMENT

DIAGNOSIS ESTABLISHED

If a cause of syncope can be determined by the initial history, physical examination, and ECG, the disposition is simple. Patients with cardiac or neurologic syncope should be admitted. Patients with vasovagal, orthostatic, and medication-related syncope have no increased risk of cardiovascular morbidity or mortality[6] and do not need admission as long as deficits are corrected.

UNEXPLAINED SYNCOPE

Despite best efforts, a diagnosis will not be established in about 40% of the patients with syncope. Several studies have assessed risk stratification variables to identify patients at risk of both short-term and 1-year morbidity and mortality. Martin et al[37] performed derivation and validation studies on cohorts of consecutive ED patients with syncope to identify predictors of arrhythmia and death at 1 year. Important risk factors were a history of arrhythmia, an abnormal electrocardiogram, a history of congestive heart failure, and age >45 years. Quinn et al[2,38] assessed adverse outcomes at 7 and 30 days in their derivation and validation of the **San Francisco Syncope Rule**. Significant predictors of adverse events (primarily arrhythmia) included (1) a history of congestive heart failure, (2) an abnormal electrocardiogram (a rhythm other than sinus, including those on rhythm strips or monitoring, conduction delays or new changes as minimal as first-degree atrioventricular block, or any morphologic changes to the QRS complex or ST segment that could not be proven to be old by prior tracings), (3) a hematocrit of <30, (4) a complaint of shortness of breath, and (5) a systolic blood pressure of <90 mm Hg in the ED. There have been inconsistent findings when validating the San Francisco Syncope Rule, which have been primarily related to definitions of syncope and when applying variables.[39] The Osservatorio Epidemiologico sulla Sincope nel Lazio study group developed a risk score based on predictors of death at 1 year, which they found to be an abnormal electrocardiogram, a history of cardiovascular disease (including congestive heart failure), age >65 years, and syncope without prodrome.[40] Sarasin et al[41] developed a prediction score for subsequent arrhythmia in patients with unexplained syncope after a standard ED evaluation;

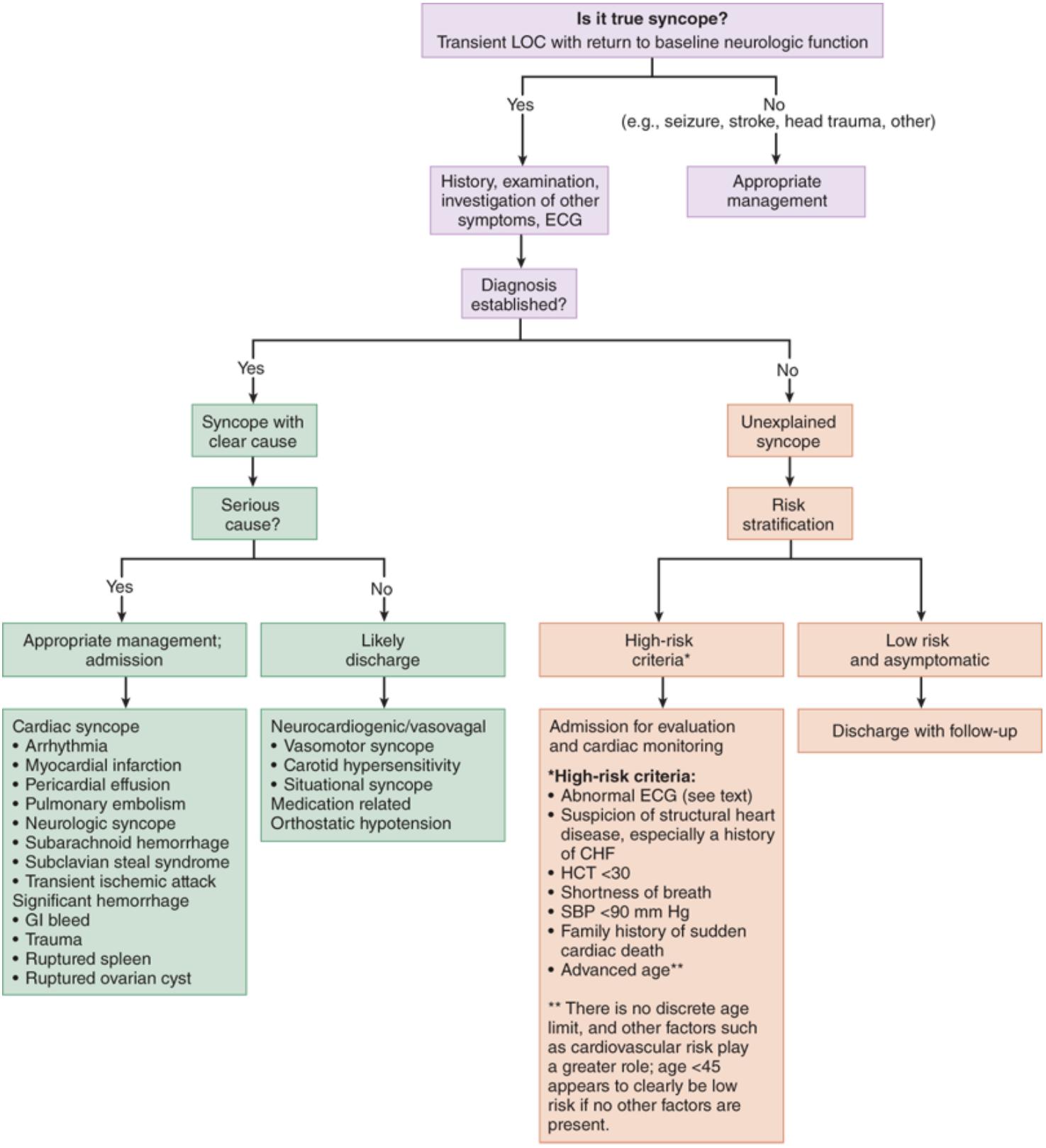
they found the significant variables to be an abnormal electrocardiogram, a history of congestive heart failure, and age >65 years. Continued analysis of the San Francisco syncope cohort, assessing 1418 consecutive patients with syncope, found the death rate to be 1.4% at 30 days, 4.3% at 6 months, and 7.6% at 1 year. The five high-risk criteria listed earlier had an 89% sensitivity and 52% specificity for death at 1 year.[42]

Using the risk factors identified in these studies can help clinicians determine patient risk and appropriate disposition. Although each study may be limited by the size of the cohort, the number of adverse events, and the definition of these events, there is a consistent theme that patients with an abnormal electrocardiogram on presentation and/or a history of heart disease, particularly structural heart disease especially characterized by a history of congestive heart failure, are clearly at increased risk.

GENERAL MANAGEMENT ALGORITHM

A general management algorithm is shown in **Figure 52-1**, which follows the recommendations of the American College of Emergency Medicine guidelines for the disposition of patients with syncope presenting to the ED and lists essential risk factors.[43]

FIGURE 52-1.
ED evaluation of syncope provides a general management strategy. CHF = congestive heart failure; ECG = electrocardiogram; HCT = hematocrit; LOC = loss of consciousness; SBP = systolic blood pressure.



Risk factors not included in Figure 52-1, but suggested by guidelines[44,45] as factors that could be used for admission or expedited follow-up decision making, include **syncope while supine, syncope during exercise, syncope without prodromal symptoms, palpations preceding syncope, and the specific age cut points of >60 or >65 years**.

TREATMENT

Treatment should be guided by the diagnosis. Patients with or at risk for life-threatening dysrhythmias can be treated with pacemakers or automatic implantable defibrillators as indicated. For patients with suspected medication causes, remove the offending agent. Rehydrate those with orthostasis and dehydration. Educate patients with vasovagal syncope; episodes are likely to recur, and patients should lie or sit down when they sense a prodrome. β-Blockers do not decrease episodes of vasovagal syncope.[46]

DISPOSITION AND FOLLOW-UP

INPATIENT EVALUATION

With the exception of patients with acute life-threatening diagnoses (e.g., stroke, aortic dissection), the core of the inpatient evaluation is focused on identification of underlying heart disease and detection of dysrhythmias (**Table 52-3**). Although admitted patients undergo continuous electrocardiographic monitoring, the utility of admission and monitoring is questioned.[26,47] Dysrhythmia as the cause of syncope is confirmed in the patient with recurrent symptoms during a monitored dysrhythmia and excluded in the patient with recurrent symptoms and sinus rhythm. An echocardiogram should be performed on patients with known or suspected heart disease to evaluate for valvular disorders, congenital anomalies, and cardiomyopathies and to determine overall cardiac function. Echocardiogram abnormalities are usually clinically apparent and will seldom be found in patients with a normal cardiac examination and electrocardiogram. Stress testing is used to identify exercise-induced dysrhythmias or ischemia or to reproduce exertional syncope once hypertrophic cardiomyopathy has been excluded by echocardiography. Electrophysiology testing is typically reserved for patients with documented dysrhythmia, preexcitation, or underlying heart disease. Electrophysiology testing involves invasive electrical stimulation and cardiac monitoring to uncover possible conduction abnormalities that predispose to tachydysrhythmias (both ventricular and supraventricular) or bradydysrhythmias.

TABLE 52-3

Post-ED Testing for Syncope/Syncope Mimics

Test	Indication	Utility
Cardiac syncope		
Electrocardiographic monitoring	Admission Outpatient ambulatory monitoring if no significant cardiac disease suspected	Cardiac syncope confirmed if recurrent symptoms occur during monitored dysrhythmia; excluded if recurrent symptoms reported during sinus rhythm
Implantable loop recorder	Recurrent syncope after admission evaluation	Long-term use with diagnostic yield of >50% in patients with recurrent syncope
Echocardiography	History, examination, or electrocardiogram suggestive of structural heart disease	Confirms and quantifies suspected structural heart disease
Electrophysiology testing	Documented dysrhythmia or serious underlying heart disease	Identifies inducible tachydysrhythmias and some bradydysrhythmias
Stress testing	Exercise-related syncope	Identifies exercise-induced dysrhythmias and postexercise syncope
Neurologic syncope		
CT/magnetic resonance angiography/carotid Doppler	Neurologic signs or symptoms	Identifies cerebrovascular abnormality or subclavian stenosis
Electroencephalography	Suspected seizure	Documents underlying seizure disorder
Reflex-mediated syncope		
Tilt-table testing	Recurrent syncope, cardiac etiology excluded	Positive test establishes diagnosis of neurocardiogenic syncope
Psychogenic		
Psychiatric testing	Young patient, no underlying heart disease	Identifies underlying psychiatric disorder predisposing to syncope

OUTPATIENT EVALUATION

Patients directed to outpatient syncope evaluation should be at low risk for serious cardiac dysrhythmias. Long-term cardiac monitoring, which includes ambulatory or event monitors, is useful to identify dysrhythmias (**Table 52-3**). The duration one should wear an ambulatory monitor is debatable, but monitors are now more portable and can be worn for long periods.[48] Long-term use of implantable loop recorders have a diagnostic yield of >50% in patients with recurrent syncope.[49,50,51] Tilt-table testing is also suggested for patients with recurrent, unexplained syncope. This test is designed to identify reflex-mediated syncope by rapidly moving the patient from a supine position on the tilt table to an upright position of 60 degrees for 45 minutes. A positive end-point is reached if syncope, hypotension, or the patient's typical symptoms are reproduced. Repeat testing with isoproterenol or sublingual nitroglycerin is performed if the initial evaluation is negative. Recurrent reflex-mediated syncope resistant to conservative therapies can be treated with a cardiac pacemaker. Psychiatric referral is recommended for young patients without underlying heart disease who have frequent syncopal events. Generalized anxiety and depressive disorders are the most commonly assigned diagnoses. Patients with a prolonged QT segment should be referred for genetic testing for the *LQTS* gene. Those who are gene negative have very little risk for fatal syncope.[52]

SPECIAL POPULATIONS

THE ELDERLY

Because of both normal physiologic changes with aging and age-related disease processes, the elderly are at increased risk for syncope and adverse outcomes.[8] Syncope in the elderly is often multifactorial, and the cause is often difficult to establish, particularly in the ED.

Various specified ages have been studied as risk factors for fatal or serious outcomes after syncope; however, there is a gradual continuum of increasing risk with increasing age. Cardiovascular risk factors appear to be better predictors than age itself. As a person ages, the blood vessels become calcified and less compliant, leading to diminished flow rates. The left ventricle also becomes less compliant, resulting in increased diastolic filling pressures and an increased dependence on the "atrial kick." There is a general decrease in adrenergic receptor responsiveness of both the heart and the peripheral blood vessels. Decreased adrenergic responsiveness contributes to the diminished chronotropic response seen after orthostatic stresses in the elderly. The incidence of vasovagal syncope actually decreases with age, in part as a consequence of the decreased responsiveness of the autonomic nervous systems. The elderly also have a less sensitive thirst mechanism and a decreased endocrine response to volume depletion, exacerbating orthostatic hypotension. Postprandial hypotension is more common in the elderly, especially in nursing home patients, and is thought to be due to a rapid rate of nutrient delivery from the stomach into the small intestine. Pathophysiologic processes that may contribute to diminished cerebral perfusion include disorders such as hypertension, atherosclerosis, and valvular disease. Atherosclerotic disease leads to ischemia, myocardial infarction, congestive heart failure, and dysrhythmias. Aortic stenosis is the most common obstructive cardiac lesion in the elderly, producing a fixed cardiac output. Diabetes may lead to autonomic dysfunction and peripheral neuropathy. Finally, medication usage is much more common in the elderly population, increasing the risk of orthostasis and decreasing autonomic responsiveness to orthostatic stress.[18]

PREGNANT WOMEN

Pregnancy is associated with numerous physiologic changes, including increased heart rate, decreased peripheral resistance, and increased stroke volume. In late pregnancy, the enlarged uterus may compress the inferior vena cava, decreasing venous return. The incidence of cardiac dysrhythmias, especially premature ventricular contractions, increases during normal pregnancy in young healthy women. However, there is no positive correlation between symptoms of presyncope or syncope and cardiac dysrhythmia in pregnant women. Important considerations for syncope in pregnancy include ruptured ectopic pregnancy and pulmonary embolism, although these disorders typically have other associated symptoms such as abdominal or dyspnea.

CHILDREN

Syncope in children is discussed in chapter 127, "Syncope, Dysrhythmias, and ECG Interpretation in Children."

PRACTICE GUIDELINES

Guidelines for the management of patients sustaining syncope have been published by the American College of Emergency Physicians,[43] the European Society of Cardiology,[44] the Canadian Cardiovascular Society,[45] and the American Heart Association/American College of Cardiology Foundation.[53]

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USEFUL WEB RESOURCES

1. American College of Emergency Physicians Clinical Policy Page—http://www.acep.org/practres.aspx?id=30060

2. American College of Emergency Physicians Clinical Policy on Syncope—http://www.acep.org/workarea/downloadasset.aspx?id=8828

3. American Heart Association/American College of Cardiology Foundation Scientific Statement on the Evaluation of Syncope—http://www.circ.ahajournals.org.une.idm.oclc.org/cgi/content/full/113/2/316

4. American Heart Association/American College of Cardiology Foundation Scientific Statement on the Evaluation of Syncope; PDF file of document—http://www.circ.ahajournals.org.une.idm.oclc.org/cgi/reprint/113/2/316

5. European Society of Cardiology Guidelines for Syncope (Guidelines on Diagnosis and Management)—http://www.escardio.org/guidelines-surveys/esc-guidelines/Pages/syncope.aspx

6. Canadian Cardiovascular Society Position Paper: Standardized Approaches to the Investigation of Syncope—http://www.onlinecjc.ca/article/S0828-282X(10)00003-6/fulltext