EMERGENCY MEDICINE REPORTS

Practical, Evidence-Based Reviews in Emergency Care

OCTOBER 1, 2018 VOL. 39, NO. 19

AUTHOR

Frank J. Edwards, MD, FACEP,
Program Director, Emergency Medicine Residency, Arnot Ogden Medical Center, Elmira, NY

PEER REVIEWER

Steven M. Winograd, MD, FACEP,
Assistant Clinical Professor of Emergency Medicine, Mt. Sinai Medical School, Jamaica Queens, NY

FINANCIAL DISCLOSURE

Dr. Farel (CME question reviewer) owns stock in Johnson & Johnson. Dr. Schneider (editor), Dr. Stapczynski (editor), Ms. Light (nurse planner), Dr. Edwards (author), Dr. Winograd (peer reviewer), Ms. Mark (executive editor), Ms. Coplin (executive editor), and Ms. Hatcher (editorial group manager) report no financial relationships with companies related to the field of study covered by this CME activity.

RELIAS MEDIA

Syncope

Syncope often presents difficult diagnostic and disposition challenges to emergency physicians.[1] Most syncope and presyncope patients are not suffering from an ominous condition, but for those at risk, the stakes are high. Researchers estimate that between 3-5% of emergency department (ED) visits[2] — and as many as 6% of all hospital admissions — are because of syncope or presyncope.[1,3] Approximately 80% of those admissions involve patients older than 65 years of age.[1] The majority of admitted patients with syncope undergo costly inpatient workups with no specific etiology for their "fainting fit," as Osler described it a century ago.[3,4] The estimated cost per admission is $5,400. The admission threshold for syncope patients varies widely from hospital to hospital; in the United States, the range is 13% to 83%.[5,6] The admission rates for syncope vary widely from country to country, with Canada at 12% and France at 63%.[6] Despite the development of several standardized syncope admission "rules" during the first decade of the 21st century, the proportion of patients admitted for syncope remained constant from 2001 through 2010.[3]

Relying on the most current literature, this article discusses the causes of syncope and syncope mimics, provides the best practice evaluation strategies, and will refamiliarize emergency physicians with current state-of-the-art practices regarding syncope risk stratification guidelines. The article emphasizes the clinical red flags that can be harbingers of true danger in patients who have fainted.

Causes of Syncope

Syncope is defined as a transient loss of consciousness resulting from global hypoperfusion of the cerebral cortex or reticular activating system.[1,4,7-11] The loss of consciousness generally lasts less than a minute, and is followed by a rapid return to baseline neurologic status.[8] The patient may or may not have prodromal symptoms.[12,13] The general differential diagnosis includes seizures, hypoglycemia, metabolic derangements, intoxication, psychogenic issues, and others, which will be discussed in greater detail.[1,11,14] Given syncope's rapid on-and-off nature, differentiating it from other events usually is straightforward. The emergency physician's main challenge lies in searching for a cause. But despite our best efforts, only 38-47% of patients will have a definite etiology for their syncope episode diagnosed in the ED.[15]

Medical opinion has varied over the years as to whether presyncope should be considered etiologically synonymous with syncope. The major problem is that its clinical picture is less precise — a feeling of being about to faint vs. a complete loss of consciousness — and this leads to an overlap with nonspecific dizziness, malaise, and lightheadedness.[16] A recent prospective cohort study focused exclusively on patients with presyncope in two academic EDs and measured 30-day outcomes.[17] In this cohort, presyncope made up 0.5% of ED

EXECUTIVE SUMMARY

- Syncope can be divided into reflex (neurally mediated) syncope, hypotensive syncope, and cardiovascular syncope. Presyncope is less serious, but in general should be evaluated like syncope.
- Reflex syncope has three major causes: vasovagal, carotid sinus, and situational.
- Cardiac syncope can be from abnormal rhythms or structural heart disease. The electrocardiogram (ECG) may show some abnormalities, although the critical rhythm may have normalized.
- All patients with syncope should have an ECG to assess for abnormal rhythm and Brugada.

visits. Patients with presyncope tended to be younger than syncope patients, had a lower frequency of comorbidities, and had about half the rate of serious cardiac-related outcomes as patients with syncope (5% vs. 10%).[17] The study suggests that emergency physicians had difficulty predicting which patients would go on to have negative outcomes.[16,17] Until there are better assessment tools, presyncope should be considered as a partial form of syncope, in which consciousness is not completely lost, that shares a similar array of causes. It resembles a syncope prodrome minus the loss of consciousness.[8]

Syncope can be divided into three etiologic categories: "reflex" syncope, orthostatic (or postural) hypotensive syncope, and cardiogenic syncope.[7] (*See Table 1.*)

Reflex Syncope

Reflex syncope, also called neurally mediated syncope, causes approximately 60% of all syncopal events seen in the ED.[13] It can be divided into three subcategories: vasovagal syncope, carotid sinus syncope, and situational syncope.[18,19]

Vasovagal Events. Vasovagal syncope is the most frequent single type of faint seen in the ED.[20] Mechanisms differ to some extent, but it usually begins with a fall in sympathetic output (vasodilation), which reduces venous return to the heart. This, in turn, generates a transient cardiac "hypercontractile" state, which then activates stretch receptors and provokes a paradoxical bradycardic response.[18] The combination of sympathetic inhibition and parasympathetic excitation results in a precipitous drop in cerebral perfusion pressure. Studying a population of British World War I veterans in 1918, Cotton and Lewis first suggested this mechanism.[21] Hypothesizing that the vagus nerve was involved, Lewis later demonstrated that atropine reversed the bradycardia but did not restore blood pressure, suggesting that diminished sympathetic tone also was involved. Based on this, he coined the term "vasovagal."[22] The precise reason for this autonomic dysmodulation still is unknown but likely involves the brainstem nucleus tractus solitarii.[18] There is evidence that cerebral vasoconstriction also plays a role in some cases.[23] Various authors have suggested that vasovagal syncope may have conferred a survival advantage in early hominids by causing them to "play possum" during conflict and predation scenarios, allowing the trait to linger in the gene pool.

Vasovagal syncope can be triggered by emotional distress, fear, pain, prolonged standing, physical exertion in a warm ambient temperature, and even the sight of blood.[11,18] For some individuals, simply being in a medical environment can trigger a spell. There is a familial predisposition to vasovagal syncope. The most common age of onset is during adolescence, likely because of a more vigorous autonomic response capability in this age range.[11,24] The hallmarks of vasovagal syncope are: the presence of an inciting event; a prodrome of nausea, lightheadedness, grayed vision, anxiety, a feeling of being hot or cold, diaphoresis, and pallor; and a loss of consciousness that lasts on average about 30 seconds. Observers often will notice some myoclonic muscle movements. Although the prodrome symptoms may persist for several minutes after the event, there will be a full recovery of consciousness without neurologic deficits.[18]

To be confident the event was vasovagal syncope, there should be no abnormal vital signs (including hypoxia and persistent orthostasis), no signs of anemia, no electrocardiogram (ECG) abnormalities, and no history of coronary artery disease or structural cardiac abnormality. It also is reassuring when patients report having similar events in comparable settings. If these criteria are met, patients can be discharged home.[8]

Vasovagal events can occur in the elderly, but are seen at lower rate, likely because of the age-related blunting of sympathetic and parasympathetic activity.[20] The risk of a more ominous cause increases with age. Vasovagal syncope in patients older than 60 years of age is more likely to have a shorter or even absent prodrome, making the faint more difficult to distinguish from a cardiac event.[24] The possibility of an unwitnessed fall being caused by syncope remains a concern for older adults and requires the clinician to take a careful history.[24,25]

Most patients with a likely vasovagal event need no further testing. Patients with recurring, clustering, or worsening vasovagal-like syncopal episodes need referral to elucidate the cause. Further evaluation may include head-up tilt table testing and loop recorder monitoring.[8,26]

Beyond encouraging patients to avoid triggering stimuli, the long-term treatment of recurrent vasovagal syncope involves good hydration, salt supplements, biofeedback, and the use of medications such as fludrocortisone and vasoconstrictors (such as midodrine hydrochloride).[20] Beta-blockers do not have proven efficacy, but selective serotonin reuptake inhibitors (SSRIs) have shown promise.[20] Cardiac pacing is not recommended as a first-line treatment for vasovagal syncope,[20] although the ISSUE-3 trial (2012) showed some benefit of pacing in patients with recurrent syncope who were found to have asystolic episodes on a loop recorder.[27]

Table 1. Etiology of Syncope

Type	Incidence
Neurally mediated "reflex" syncope • Vasovagal • Carotid sinus hypersensitivity • Situational syncope	60%
Postural orthostatic syncope • Iatrogenic (drug-related) • Volume depletion • Primary autonomic dysfunction (e.g., parkinsonism) • Secondary autonomic dysfunction (e.g., diabetes)	15%
Cardiac syncope • Arrhythmic • Structural	15% (10%) (5%)
Unknown	10%

Because vasovagal syncope is a vasodilation problem as much as one of bradycardia in most patients, pacing will have marginal effectiveness.[28]

Carotid Sinus Syncope. Accounting for only 1% of fainting episodes,[29] carotid sinus syncope (or carotid sinus syndrome) is caused by inadvertent pressure applied to an overly sensitive carotid sinus baroreceptor. This leads to a vagally mediated bradycardia along with reflex depression of subdiaphragmatic venous tone via sympathetic efferents. It may be primarily a bradycardic response, a vasodepressor response, or a mixed picture.

This physiologic response becomes exaggerated with aging and atherosclerosis. Carotid sinus syncope primarily affects older males. One of its classic descriptions is "minister's disease" because of tight clerical collars. Another is "shaving syncope," which occurs when the skin of the lateral neck is lathered and stretched. Another example is the elderly male with atherosclerosis who faints while looking upward. The presence of neck tumors, radiation, and surgical scars also have been implicated.

The diagnosis of carotid sinus hypersensitivity can be made by carotid sinus massage. The 2017 guidelines from the American College of Cardiology/American Heart Association/Heart Rhythm Society (ACC/AHA/HRS) and the 2018 guidelines from the European Society of Cardiology define carotid sinus hypersensitivity as a finding during carotid sinus massage of a pause greater than three seconds and a systolic pressure drop of > 50 mmHg.[8,26] This asymptomatic finding itself is not uncommon in older males (age > 40 years).[8,30] Therefore, the guidelines state that symptoms must accompany these changes to make the diagnosis.

Carotid massage can be performed as follows: Massage the right and left carotid sinus areas sequentially for five seconds each with the patient both supine and upright, and with continuous monitoring. Contraindications include carotid bruits and transient ischemic attack (TIA), history of a stroke, or myocardial infarction (MI) within the past three months (unless carotid Doppler is negative for significant stenosis).[26] Very few emergency physicians perform carotid sinus massage for diagnostic purposes. The rate of false positives is high, and there is a small but real risk of causing a stroke. Studies show a role for pacemakers in certain patients with recurrent carotid sinus hypersensitivity syncope.[26]

Situational Syncope. Situational syncope is defined as a neurally mediated reflex fainting event associated with a specific activity, such as urination (micturition or post-micturition syncope) or defecation. (*See Table 2.*) Other examples include cough syncope[31] and swallow syncope, which is associated more commonly with cold or carbonated beverages. Gelastic syncope (also known as "sitcom syncope") is induced by vigorous laughter.[32] Hair-grooming syncope is a well described but rare type of fainting typically involving hair brushing (or combing, braiding, trimming, or blow drying) usually in children between the ages of 5 and 16 years. There appears to be a 3:1 predominance of females to males, and in males the event is more likely to be associated with hair cutting.[33]

Many of the underlying mechanisms for situational reflex syncope syndromes are known, and include afferent triggers in the pulmonary, gastrointestinal, and genitourinary systems that provoke reflex vasodilation and bradycardia.[34] The main problem in making the diagnosis of situational syncope is failing to consider other potential causes. It is important to perform a careful history and exam, and consider the presence of other risk factors, the patient's age, and whether there is a history of similar events.

The "fainting lark" can be categorized as a form of situational syncope, although the mechanism is not pathologic per se. This is a self-induced event, sometimes engaged in by adolescents and young adults for various reasons (including entertainment and malingering), wherein the individual squats, hyperventilates, then stands up abruptly while doing a Valsalva maneuver.[35] Syncope will follow in a majority of human beings through a combination of hypocarbia-induced peripheral vasodilation and cerebral vasoconstriction, along with the effects of suddenly standing while impeding venous return and increasing vagal output. It has been used in a research setting to study blood pressure levels and other factors during syncopal episodes.[36]

Orthostatic (Postural) Syncope

After reflex syncope, the second leading cause of fainting is orthostatic or postural syncope, accounting for about 15% of fainting cases seen in the ED.[13] The normal human physiologic response to gravitational stress caused by rising from a lying or sitting position to an upright posture involves a complex autonomic compensatory response that

Table 2. Causes of Situational Syncope

- Micturition
- Defecation
- Swallowing
- Coughing
- Laughing
- Hair grooming
- Fainting lark

keeps cardiac output and cerebral perfusion within narrow parameters. An inadequate compensatory response for any number of reasons allows cerebral blood flow to fall. A perfusion decrease of approximately 50% will initiate presyncope and may lead to an outright syncopal event.[13,34,37,38] In general, the orthostatic drop in pressure occurs within several minutes of the patient assuming a standing position, but significantly longer delays are possible. More of an issue in older patients, orthostatic syncope can be divided into four categories: iatrogenic (i.e., medication-related), volume depletion, primary autonomic dysfunction, and secondary autonomic dysfunction.[13,34] (*See Table 1.*)

Medication-induced failure of postural compensation is the most common cause of orthostatic syncope. The list of drugs involved in inducing syncope by this mechanism is long and includes those drugs that interfere with autonomic cardiovascular reflexes, such as the beta-blockers, calcium channel blockers, alpha-blocking agents, and phenothiazines.[13] (*See Table 3.*) By inducing fluid and electrolyte loss, diuretic agents cross over into the second category — volume depletion. Volume depletion orthostatic syncope also is seen in patients experiencing dehydration or hemorrhage.

The third category of orthostatic hypotension falls under the rubric of primary autonomic nervous system failure. A constellation of different diseases can lead directly to deterioration of autonomic pathways. Examples include parkinsonism, Lewy body dementia, multiple-system atrophy (the Shy-Drager syndrome), and pure autonomic failure (PAF). Patients with these conditions exhibit an inability to maintain adequate cerebral perfusion when upright and even may have supine hypotension.[13]

Table 3. Medications Involved in Inducing Syncope

- Angiotensin-converting enzyme inhibitors
- Alpha receptor blockers
- Calcium channel blockers
- Beta-blockers
- Phenothiazines
- Tricyclics
- Alcohol
- Opioids
- Diuretics
- Hydralazine
- Nitrates
- Sildenafil
- Monoamine oxidase inhibitors

The final class of orthostatic intolerance is secondary autonomic failure, which refers to the autonomic neuropathies accompanying conditions such as diabetes, amyloidosis, familial dysautonomia, immune-related neuropathy, autoimmune autonomic ganglioneuropathy, Sjögren's syndrome, paraneoplastic syndromes, and HIV neuropathy. Diabetes is the most common pathology.[34]

Two additional entities fall under the heading of postural orthostasis. These are postprandial hypotension and postural orthostatic tachycardia syndrome (POTS). Studies show that nearly all nursing home patients experience some degree of postural hypotension after meals, especially following breakfast. This is usually asymptomatic, but is related to some fainting episodes and falls with injury.[39] The risk of postprandial hypotension is higher in patients with multiple comorbidities. POTS, on the other hand, is seen mainly in young patients and has been associated with syncopal episodes.[40] Mainly affecting post-pubertal females younger than 19 years of age, POTS consists of an increase in heart rate of 30-40 or more beats per minute on standing, accompanied by lightheadedness and weakness.[26] A diagnosis of exclusion, POTS may develop insidiously following an infectious disease such as mononucleosis. Although it can become debilitating, the condition often resolves over a period of months with supportive treatment and conditioning.[40]

Unless accompanied by syncopal symptoms, orthostatic blood pressure drops discovered during the ED evaluation of patients who present with presyncope or syncope must be interpreted with caution. Many elderly individuals have asymptomatic baseline orthostatic changes.[41] Additionally, patients on autonomic response-blunting medications have significant comorbidities, such as coronary artery disease, hypertension, and diabetes, which put them at greater risk of cardiac-related syncope. Therefore, orthostatic syncope is a diagnosis of exclusion when other etiologies have been evaluated to the extent warranted by age, history, and other clinical factors.

Cardiac Syncope

Approximately 15% of syncope cases are due to cardiac causes, with two-thirds arising from arrhythmias and one-third related to cardiac structural disorders, both of which can lead to periods of inadequate cardiac output and diminished cerebral perfusion. (*See Table 1.*) Unlike patients with neurally mediated reflex syncope, who were shown in the Framingham Study to have no greater risk of death compared to a general population cohort, individuals with cardiac syncope were twice as likely to die of any cause during the Framingham Study's 17-year duration.[42] Other studies show that the six-month mortality rate for patients with proven cardiac syncope is 10% or greater.[41] Concern that the event may be cardiac drives the high admission rates for older adults.

The list of arrhythmias associated with syncope includes any condition leading to slow, rapid, or disorganized rhythms.[41] (*See Table 4.*) The first step in evaluating all syncope patients is to obtain an ECG. Approximately 5% of older adults with syncope will have cardiac syncope diagnosed on the initial ECG, with such entities as supraventricular tachycardia, profound bradycardia, third-degree heart block, sick sinus syndrome, long sinus pauses (> 3 seconds), Mobitz type II atrioventricular (AV) block, and ventricular tachycardia.[43] Most often, the offending arrhythmia is no longer apparent and the ECG can be completely unremarkable. In a prospective, multicenter, observational study,

Table 4. Rhythm Disturbances Associated With Syncope

- Bradycardia
- Sick sinus syndrome
- Second- or third-degree heart block
- Long and short QT syndrome
- Brugada syndrome
- Ventricular tachycardia
- Supraventricular tachycardia
- Pre-excitation pathway syndromes
- Atrial fibrillation and flutter
- Torsade de pointes
- Pacemaker malfunction

researchers identified more than 3,000 older adults (> 60 years) presenting with syncope (excluding those who had obvious cardiac syncope) and discovered that approximately 3% were diagnosed with a serious cardiac arrhythmia within the next 30 days. The following initial ECG abnormalities predicted a two- to three-fold greater risk: nonsinus rhythm, multiple premature ventricular contractions, short PR interval, first-degree AV block, complete left bundle branch block, and Q wave/T wave/ST segment changes consistent with acute or chronic ischemia. ECG abnormalities such as these are useful risk-stratification aids but are neither sensitive nor specific enough to determine the risk of a serious arrhythmic event.[43] (*See Table 5.*)

Other ECG abnormalities suggest a higher risk, including the genetically transmitted channelopathies such as the long QT syndrome (QTc > 450 ms for males, > 470 ms for females), short QT syndrome (QTc < 370 ms),[44] and the delta wave and short PR interval of Wolff-Parkinson-White pre-excitation syndrome, a form of which is inherited. Syncope patients always should be questioned about the presence of fainting spells or sudden death in family members. Brugada syndrome is a rare hereditable mutation of a gene involved in the expression of myocardial sodium channels and is a well-recognized cause of sudden arrhythmic death.[45] The classic Brugada ECG demonstrates a pattern resembling right bundle branch block in leads V1-V3, with cove-shaped ST elevation with a J-point elevation of 2 mm or greater and T-wave inversion. These classic changes may be expressed fully only when the patient receives a provocation dose of an agent such as the antiarrhythmic drug ajmaline.

Table 5. Red Flags for Cardiac Syncope

- Onset during exertion
- History of cardiac disease
- Electrocardiogram changes
- Lack of prodrome
- Palpitations at onset
- Chest pain
- Occurrence while supine
- Age > 60 years
- Family history of sudden death

Structural heart disease causes syncope by interfering with cardiac output when demand rises. Exertional syncope in the elderly that is accompanied by chest pain and dyspnea is a classic presentation of critical aortic stenosis.[41] The most common etiology of sudden death in the younger age group is hypertrophic cardiomyopathy, which also predisposes patients to tachyarrhythmia events.[46] Fainting during exercise in otherwise healthy adolescents and young adults warrants concern for this inherited condition. Other structural etiologies for syncope include myocardial ischemia, pericardial effusion, various cardiomyopathies, and atrial myxoma.[34,47] Infectious myocarditis can lead to syncopal events caused by structural myocardial dysfunction or by predisposing patients to tachyarrhythmias.[48]

Pulmonary embolism (PE) can cause a form of structural syncope by interfering with pulmonary venous return and via other mechanisms. In a recent multicenter Italian study, researchers evaluated patients admitted with a first syncopal episode and found an unexpectedly high prevalence (17.3%) of PE. Researchers noted that PEs were discovered at a higher rate in patients who did not have alternative explanations for the syncopal event, but also in patients who did have alternative explanations.[49] The ED evaluation of syncope patients needs to include an assessment of PE risk factors along with attention to relevant vital sign abnormalities, such as tachypnea, sinus tachycardia, and hypoxia, and examination for signs of deep vein thrombosis.

Cardiac syncope is more likely in males older than 60 years of age who experienced no prodrome, and when the event occurs during exertion.[11] Another red flag is the presence of palpitations before the event. Consider cardiac syncope when the faint happened while the patient was supine.

Syncope in Children

Pediatric patients have fewer comorbidities and a more dynamic autonomic response than older adults. Therefore, it is not surprising that approximately three-quarters of children and adolescents who presented to hospitals with syncope in one multicenter series were diagnosed with benign neurally mediated reflex syncope, and less than 3% were found to have cardiac-related syncope.[40,50] Up to half of all adolescent females and a quarter of adolescent males experience a vasovagal faint. In otherwise healthy children from 6 months to 4 years of age, breath-holding spells can trigger a syncopal event. The typical breath-holding spell (which may be seen in up to 5% of children) involves a triggering event such as a fall or being disciplined, after which the child cries, then holds his or her breath until facial cyanosis is visible and the child briefly loses consciousness, terrifying his or her caregivers.[51]

At minimum, pediatric patients with syncope should receive an ECG. The history should include the presence or absence of triggering events, prodromal symptoms, or palpitations. Although vasovagal episodes after exertion are common, fainting during exercise is always concerning, as is a positive family history of early sudden death or suspicious syncope. The presence of injuries suggests the fainting event occurred without warning. Abnormal cardiac findings, including murmurs or signs or congestive failure, suggest a more serious cause. Such findings warrant cardiology evaluation in a timeframe appropriate to the degree of concern.

Psychogenic pseudosyncope (PPS) events were observed at a rate of 2.3% in one large study of syncope in children and adolescents.[50] Patients with PPS may have a concomitant anxiety disorder or

Table 6. Syncope Mimics

- Seizures
- Metabolic disturbances
- Intoxication
- Psychiatric pseudo-seizures
- Large anterior circulation cerebrovascular accident
- Subarachnoid hemorrhage
- Basilar transient ischemic attacks
- Subclavian steal syndrome
- Thoracic aortic dissection
- Cataplexy

underlying depression. PPS is classified as a conversion disorder and is seen most often in young females. Possible clues include a history of frequent episodes with falls that do not lead to injury, and episodes of unresponsiveness that last longer than the typical syncope duration of a minute or less.[52]

Syncope in Athletes

Most syncopal events in athletes are neurally mediated reflex events.[53] However, even a benign syncopal event that occurs during the performance of technically dangerous athletic activity, such as motor sports or diving, raises the risk of serious injury. In one large series of athletes undergoing screening evaluations, 12% reported an episode of post-exertional syncope during the past five years. A smaller percentage experienced fainting during exercise, and in this group, there was a higher frequency of cardiac causes. Important items in the history may include a prodrome, palpitations, medication use, family history of sudden death at an early age, and an event occurring during actual exertion. Athletes who experience syncope during exertion require a focused cardiology evaluation searching for the presence of such entities as hypertrophic cardiomyopathy, ion channel disorders, and right ventricular outflow tract arrhythmias.[53]

Syncope Mimics

Syncope mimics are conditions that share features with syncope and must be considered when evaluating patients who have fainted.[8,11] (*See Table 6.*) Foremost among the syncope mimics are seizures. It is very common for a syncope patient to have an initial ED chief complaint of "seizure" because a witness saw the individual's eyes rolling back and limbs jerking. The transient cerebral hypoxia of syncope will cause many patients to have myoclonic jerking that resembles seizure activity. However, this involuntary motor activity generally will be briefer and more irregular than the tonic-clonic activity of a grand mal convulsion. Urinary and fecal incontinence can occur with both seizures and syncope, but is seen more commonly with an epileptic event. Fecal incontinence with fainting is exceedingly rare.

Another key differentiating feature is the lack of prolonged postictal obtundation or agitation after a faint. Syncope patients may feel dazed and unwell and appear confused after the event, but normal consciousness will return within several minutes. Children may feel fatigued and drowsy for half an hour or more after fainting. Tongue biting can occur with syncope, but is more likely to be at the tip of the tongue than on the lateral aspect of the tongue, as seen after seizures. This is because of a sudden mouth closure if the patient faints and hits his or her chin, as opposed to tonic-clonic clenching of the jaw during a seizure.

Carotid circulation TIAs and strokes do not cause bihemispheric perfusion deficits and, in theory, should not result in a true syncopal event. But major cerebrovascular accidents (CVAs), such as a large vessel occlusion, an intraparenchymal bleed, or subarachnoid hemorrhage, often are heralded by a transient loss of consciousness. Unlike patients with syncope, these patients will not return to baseline mental status but typically will have neurologic deficits, altered mental status, or severe headache. Posterior circulation TIAs and strokes may cause a transient loss of consciousness via a circulatory insult to the brainstem reticular activating system.[23] Basilar vascular events typically are accompanied by neurologic signs and symptoms of varying severity, such as ataxia, diplopia, dysphagia, hemiparesis, paresthesias, vertigo, and headache. However, basilar TIAs may result in syncope without lingering posterior fossa neurologic signs.

The subclavian steal syndrome can cause syncope. This uncommon condition involves an occlusion of the subclavian artery proximal to the vertebral artery. When the ipsilateral arm is exercised, retrograde flow down the vertebral artery supplies the excess demand and can result in brainstem ischemia and occasionally syncope. It usually is asymptomatic but can cause symptoms, especially in people who work with their arms raised above their heads. Episodes of vertigo occur in about half of these individuals as well. Patients with subclavian steal will have a blood pressure differential from one arm to the other.

Another vascular disorder that can result in syncope is aortic dissection. As the dissection wave proceeds between the wall layers, it can impinge upon the carotid and vertebral arteries, thus impairing cerebral or basilar circulation, respectively. Patients generally have chest pain radiating into the back.

Metabolic and toxic events, such as hypoglycemia, hypoxia, carbon monoxide poisoning, sepsis, or respiratory alkalosis secondary to hyperventilation, along with various intoxications, can result in episodes of transient loss of consciousness. The clinician usually will have ample clues to help differentiate these conditions from that of a classic syncopal episode, including laboratory and vital sign abnormalities, lingering mental status changes, or obtundation. Some individuals experiencing hypoglycemia, especially diabetes patients being treated with insulin, will have fainting events followed by a return to near baseline mental status.[34] For this reason, it is reasonable to perform bedside glucose testing on patients with otherwise unexplained syncope, especially those patients with diabetes.

Cataplexy also may mimic a syncopal episode. Rarely seen except in patients with narcolepsy, a cataplexy event usually is triggered by intense emotion, such as anger, fear, or laughter. It involves a sudden transient loss of muscle tone that may be mild or severe enough to make the patient collapse. The key feature distinguishing it from syncope is that patients experiencing a cataplexy episode will not lose consciousness.[34] As many as 70% of patients with narcolepsy can experience cataplexy spells.

Psychogenic pseudosyncope can mimic both cataplexy and syncope. Its characteristics are described in the section on syncope in children.

In older literature, the term "drop attack" often is listed under the heading of syncope mimics. A "drop attack" generally refers to a sudden fall without loss of consciousness. Used to describe events caused by many conditions, including orthopedic, cardiac, and neurologic events, the term is ambiguous, and the clinician is better served by more specific descriptors of such episodes.

ED Evaluation of Patients With Syncope

History

A good history, obtained from the patient and any available witnesses, remains vital to the evaluation of syncope, and will suggest the most likely diagnosis in 50% or more of cases.[9] The first step involves confirming that the event really was syncope and was not a seizure. The loss of consciousness of syncope usually is less than a minute and full consciousness is regained quickly. Involuntary myoclonic motor activity is common with syncope but usually quite brief. Urinary incontinence may occur with syncope, but occurs less often than during a seizure.

The next step is to gather as much information as possible about the setting, the presence of a prodrome, and other situational and historical data. Was there a possible triggering event, such as standing for a long time in a warm environment or the sight of blood? Was the event postural in nature? Was the patient looking up or wearing a tight collar? Was there some reason the patient might have been poorly hydrated, such as working outside on a hot, humid day without much fluid replenishment? Was there a prodrome of nausea, diaphoresis, and lightheadedness, and how long did the prodrome last?

Syncope without warning raises the red flag for a cardiac cause. (*See Table 5.*) Palpitations, chest pain, and the occurrence of fainting during exertion or while supine also suggest a cardiac cause, either ischemic, dysrhythmic, or structural. Syncope with dyspnea raises concern for a pulmonary embolism, chest pain for MI or dissection, headache for subarachnoid hemorrhage, back pain for aortic dissection, abdominal pain for ruptured abdominal aortic aneurysm, and pelvic pain for a ruptured ectopic pregnancy. If the patient was exercising an arm when the event occurred, consider subclavian steal syndrome.

Focus past history on the existence of previous similar events; the presence of underlying structural, valvular, or atherosclerotic cardiac disease; or any comorbidities such as congestive heart failure, diabetes, hypertension, thromboembolic disorder, or parkinsonism. The patient's medication list should be reviewed for drugs such as diuretics, beta-blockers, calcium channel blockers, and other arrythmogenic or vasoactive agents. Has there been a recent change in medications? Had the patient been using alcohol or any illicit substances prior to the event? For family history, are there any instances of sudden death at a young age to suggest an occult congenital condition, such as the long QT syndrome or Brugada syndrome?

Physical Exam Features

Vital sign abnormalities may yield clues immediately to the nature of the syncopal episode, such as slow or rapid heart rates pointing toward an arrhythmic event, hypoxia and tachypnea suggesting a pulmonary embolism, or the presence of a fever. Orthostatic blood pressure measurement is warranted in most syncope patients. An altered sensorium raises concern for toxic or metabolic insults, cerebrovascular accidents, or postictal states and may be seen in aortic dissection as well. Cardiovascular examination focuses on identifying cardiac murmurs, signs of congestive failure, or unequal extremity pulses. A rigid, tender abdomen may herald a ruptured ectopic or abdominal aortic aneurysm. Search for neurologic deficits, including posterior fossa signs such as vertigo, abnormal nystagmus, diplopia, and ataxia. If anemia is suspected, a rectal exam to check for gastrointestinal bleeding may be warranted.

The physical exam also should evaluate for signs of trauma. In elderly patients who have fallen, assume that syncope might have precipitated the fall, unless the history clearly indicates that the fall was mechanical in nature. The same holds true for individuals involved in otherwise unexplained motor vehicle accidents. If an individual receives injuries during a syncopal event that suggest they did nothing to try and protect themselves during a fall, it goes against the likelihood of the event being a psychogenic pseudoseizure.

Testing Strategies

An ECG is the only test that should be performed on all syncope patients.[41] Although it usually will be normal, the ECG is a quick, inexpensive, and noninvasive risk-stratification tool. Analyze the ECG for obvious rhythm disturbances (bradycardia, atrial fibrillation, ventricular fibrillation) and signs of ischemia or myocardial damage, along with conduction abnormalities including AV blocks. Search for either short QT interval (QTc < 350 milliseconds [ms]) or long QT interval (QTc > 450 ms in males, > 470 ms in females). New or old left bundle branch block patterns and anterior or posterior fascicular block are associated with greater morbidity in the setting of syncope.[41] Also evaluate all ECGs for the right bundle branch block, ST elevation pattern of Brugada.

The need for laboratory testing or imaging is driven by the clinical situation.[26,41] All reproductive-capable females who experience syncope should have a pregnancy test. Concern for anemia requires a complete blood count. Patients with diabetes should have their glucose tested. A chemistry panel is warranted if there is a possibility of electrolyte abnormality. Chest pain or ECG changes suggest the need for troponins. Patients at low probability for pulmonary embolism can be risk-stratified with a D-dimer assay. Go straight to CT angiography for patients at moderate to high risk for pulmonary embolism or for patients in whom the diagnosis of thoracic aortic dissection is being considered. Carotid or vertebral artery dissections are a rare cause of syncope, but in patients with syncope and neck pain or injury, CT angiography of the neck is indicated. Cardiac echocardiogram is mandated at some point for patients whose syncope may have arisen from structural heart conditions, such

Table 7. Validated Syncope Risk Guidelines

Name	Variables	Results	Sensitivity
San Francisco Syncope Rule (2004)[56]	• Abnormal electrocardiogram • Congestive heart failure history • Hematocrit • Dyspnea • Systolic blood pressure < 90 mmHg	Consider admission if one or more variables is present	87% (conflicting internal and external validations)
Boston Syncope Criteria (2007)[15]	• Volume depletion • Abnormal vital signs or patient is symptomatic or has an abnormal exam • Acute coronary syndrome • Conduction disease • Worrisome cardiac history • Family history of sudden death • Central nervous system event • Valve disease by history or on exam	Consider admission if one or more variables is present	97% (small sample internal validation)
Risk Stratification Of Syncope in the Emergency Department (ROSE) (2010)[60]	• Hemoglobin < 9 g • Brady < 50 beats per minute • B-type natriuretic peptide (BNP) > 300 ng/L • Chest pain • Q waves (except lead III) • O_2 sat < 94% • Fecal occult blood (if gastrointestinal bleed is suspected)	Consider admission if one or more variables is present	87% (small sample internal validation)

as valvular disease, atrial myxoma, or hypertrophic cardiomyopathy.

Tilt table testing can elucidate the cause of recurrent neurally mediated or postural syncope, although this seldom if ever will be performed in the ED.[26] Evaluation for arrhythmogenic cardiac syncope warrants a period of cardiac monitoring. If the patient meets high-risk criteria, as discussed below, this should be done in the inpatient setting. Other things being equal, lower-risk patients sometimes may be discharged safely from the ED with portable short-term cardiac monitoring devices (Holter) and firm follow-up instructions. For patients with recurrent unexplained syncope, implantable loop recorders have shown promise.[54,55]

Carotid sinus massage, the technique that has been described previously, can be used to diagnose carotid sinus hypersensitivity, but is seldom used in the ED. This has less to do with the rare but potentially catastrophic danger of causing a CVA, and relates more to the high rate of false positives.[41]

Unless the history and examination suggest head trauma or a CVA, routine CTs of the head are not indicated in patients with no further symptoms after an isolated syncopal event.[41]

Decision to Admit: Syncope Risk-stratification Rules

To address the issue of ED overadmission of syncope patients who cannot be diagnosed with a benign cause for the event,[41] several clinical decision-making rules have been developed over the past 15 years to risk stratify patients. (*See Table 7.*) One of the earliest and most extensively validated tools is the San Francisco Syncope Rule (SFSR), published in 2004.[56] Against an outcome measure of serious events at seven days (including death, MI, PE, CVA, arrhythmias, subarachnoid hemorrhage, significant hemorrhage, and related return ED visits or readmission), the SFSR assessed the predictive value of the following variables in patients with syncope or presyncope: abnormal ECG, history of congestive heart failure; hematocrit < 30%, dyspnea, and systolic blood pressure < 90 mmHg. A subsequent meta-analysis of studies using the SFSR suggested that in patients whose syncope remains undiagnosed after ED evaluation, the presence of one of these variables yields a sensitivity of 0.87 in predicting an adverse outcome.[57] The original SFSR investigators calculated that application of the rule could reduce admission rates safely by 10%.[9]

The most recently published set of syncope guidelines is the Canadian Syncope Risk Score, which was published in 2016 and has not yet been fully validated.[58] In this prospective observational study of more than 4,000 patients — the largest study yet involving risk stratification in syncope — researchers assigned positive or negative values to various clinical features in a cohort of patients 16 years of age and older. Positive predictors of adverse outcomes include: history of heart disease; any systolic pressure < 90 mmHg or > 180 mmHg; elevated troponin

levels; abnormal QRS axis; QRS > 130 ms; QTc > 480 ms; and an ED diagnosis of cardiac syncope. Negative values were assigned to two additional factors: a patient's predisposition to vasovagal symptoms and an ED diagnosis of vasovagal syncope.

One of the interesting features of this study is that it incorporates clinical judgment into the scoring paradigm. This integrates the findings from a meta-analysis of several syncope rules, which suggested that clinical judgment is as sensitive as any rules yet developed in predicting adverse outcomes in syncope.[59] The total points in the Canadian Syncope Risk Score stratify patients into categories ranging from very low risk to very high risk. This is now in the process of being validated.

Summary

The evaluation of patients with syncope remains a frequent challenge facing emergency physicians. Careful history taking, examination, and the judicious use of testing, if needed, will allow physicians to diagnose many patients with benign, neurally mediated, reflex syncope events. But at least half the patients seen in the ED with syncope will receive no definite diagnosis.[41] A small percentage of them will have fainted because of immediate life-threatening conditions, such as acute coronary syndromes, malignant arrhythmias, pulmonary embolism, aortic dissection, subarachnoid hemorrhage, and other catastrophic conditions. As long as physicians remain alert for the red flags of abnormal vital signs and associated symptoms, such as dyspnea, headache, and chest pain, these conditions are relatively easy to suspect. But a certain percentage of patients will have cardiac syncope because of factors that will not be obvious during the ED evaluation, and these patients have the potential for adverse events if discharged with an incomplete evaluation.

The use of syncope risk stratification guidelines can help make rational admission decisions, although as of yet they do not replace the gestalt of experienced clinicians, who will be aware of the following high-risk features and incorporate them into their decision making: syncope without a prodrome; syncope accompanied by chest pain, shortness of breath, or palpitations; syncope during activity; syncope while supine; a history of CHF, coronary artery disease, or other cardiac conditions, including valvular disease; abnormal ECG findings, as discussed previously; the presence of anemia; family history of sudden death at a relatively young age; hypotension or hypertension; and age older than 60 years.

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CME/CE Questions

Questions 1-5 refer to the following case: A mother brings her 16-year-old daughter to the ED after the teenager had a "seizure" in gym class. There is no significant history of major illness, no history of similar events, and no available witnesses to this episode.

1. Which of the following features is most suggestive of a vasovagal event?
   a. Evidence of tongue biting on exam
   b. Patient's description of a prodrome consisting of nausea, sweating, and lightheadedness
   c. The event occurred while the patient was actively playing soccer.

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d. Urinary incontinence

2. Which of the following is most likely to lead to a diagnosis?
   a. Orthostatic blood pressures
   b. Clear history from the patient and head computed tomography
   c. An eyewitness
   d. Electroencephalogram
3. The patient's physical exam is unremarkable, her vital signs including orthostatics are normal, and she denies any acute illness in recent days. An electrocardiogram is pending. Which of the following tests should be done in the ED?
   a. Chest computed tomography
   b. Pregnancy test
   c. Electroencephalogram
   d. Head computed tomography
4. What is the most important question to ask the patient's mother?
   a. Does the patient have a history of anxiety or depression?
   b. What is the likelihood that the patient may be using illicit drugs?
   c. Is there a history of sudden death at a relatively young age in blood relatives of the patient?
   d. Is the patient on any prescription medications?
5. Which of the following features would mandate further specialty evaluation for her episode of syncope?
   a. Syncope while running
   b. Positive pregnancy test
   c. Syncope after running
   d. Syncope during laughter
6. When scanning an ECG for classic Brugada changes (prolonged QRS appearance, coved ST elevation, negative T wave), in which lead(s) will these changes appear most prominently?
   a. aVR
   b. II, III, aVF
   c. V1, V2, V3
   d. V4, V5, V6
7. The San Francisco Syncope Rule includes which of the following assessment factors?
   a. Hematocrit < 30%
   b. Systolic blood pressure < 60 mmHg
   c. Abnormal right- and left-sided ECGs
   d. Widened mediastinum on chest X-ray
8. A young orchestra conductor is brought to the ED after fainting toward the end of the first movement of Mozart's 40th symphony. His examination and ECG are completely normal. He reports that he had begun experiencing right (dominant) arm cramping and dizziness recently after taking a new job that involved conducting long concerts. An effective way to exclude a possible subclavian steal syndrome would be to:
   a. perform carotid Doppler flow studies.
   b. check the blood pressure in each arm.
   c. order a spiral CTA of the chest.
   d. order an MRI with and without gadolinium contrast.
9. When assessing whether a patient had a syncopal episode or a seizure, the presence of which feature makes epilepsy the more likely diagnosis?
   a. Involuntary motor activity
   b. Urinary incontinence
   c. Abrasions on the forehead
   d. Fecal incontinence

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Upon completion of this educational activity, participants should be able to:

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- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

EMERGENCY MEDICINE REPORTS™
(ISSN 0746-2506) is published semimonthly by Relias Learning, 111 Corning Road, Suite 250, Cary, NC 27518-9238. Periodicals postage paid at Cary, NC, and additional mailing offices. POSTMASTER: Send address changes to *Emergency Medicine Reports*, Relias Learning, 111 Corning Road, Suite 250, Cary, NC 27518-9238.

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EMERGENCY MEDICINE **REPORTS**

Syncope

Etiology of Syncope

Type	Incidence
Neurally mediated "reflex" syncope • Vasovagal • Carotid sinus hypersensitivity • Situational syncope	60%
Postural orthostatic syncope • Iatrogenic (drug-related) • Volume depletion • Primary autonomic dysfunction (e.g., parkinsonism) • Secondary autonomic dysfunction (e.g., diabetes)	15%
Cardiac syncope • Arrhythmic • Structural	15% (10%) (5%)
Unknown	10%

Medications Involved in Inducing Syncope

- Angiotensin-converting enzyme inhibitors
- Alpha receptor blockers
- Calcium channel blockers
- Beta-blockers
- Phenothiazines
- Tricyclics
- Alcohol
- Opioids
- Diuretics
- Hydralazine
- Nitrates
- Sildenafil
- Monoamine oxidase inhibitors

Causes of Situational Syncope

- Micturition
- Defecation
- Swallowing
- Coughing
- Laughing
- Hair grooming
- Fainting lark

Rhythm Disturbances Associated With Syncope

- Bradycardia
- Sick sinus syndrome
- Second- or third-degree heart block
- Long and short QT syndrome
- Brugada syndrome
- Ventricular tachycardia
- Supraventricular tachycardia
- Pre-excitation pathway syndromes
- Atrial fibrillation and flutter
- Torsade de pointes
- Pacemaker malfunction

Red Flags for Cardiac Syncope

- Onset during exertion
- History of cardiac disease
- Electrocardiogram changes
- Lack of prodrome
- Palpitations at onset
- Chest pain
- Occurrence while supine
- Age > 60 years
- Family history of sudden death

Syncope Mimics

- Seizures
- Metabolic disturbances
- Intoxication
- Psychiatric pseudo-seizures
- Large anterior circulation cerebrovascular accident
- Subarachnoid hemorrhage
- Basilar transient ischemic attacks
- Subclavian steal syndrome
- Thoracic aortic dissection
- Cataplexy

Validated Syncope Risk Guidelines			
Name	**Variables**	**Results**	**Sensitivity**
San Francisco Syncope Rule (2004)[56]	• Abnormal electrocardiogram • Congestive heart failure history • Hematocrit • Dyspnea • Systolic blood pressure < 90 mmHg	Consider admission if one or more variables is present	87% (conflicting internal and external validations)
Boston Syncope Criteria (2007)[15]	• Volume depletion • Abnormal vital signs or patient is symptomatic or has an abnormal exam • Acute coronary syndrome • Conduction disease • Worrisome cardiac history • Family history of sudden death • Central nervous system event • Valve disease by history or on exam	Consider admission if one or more variables is present	97% (small sample internal validation)
Risk Stratification Of Syncope in the Emergency Department (ROSE) (2010)[60]	• Hemoglobin < 9 g • Brady < 50 beats per minute • B-type natriuretic peptide (BNP) > 300 ng/L • Chest pain • Q waves (except lead III) • O_2 sat < 94% • Fecal occult blood (if gastrointestinal bleed is suspected)	Consider admission if one or more variables is present	87% (small sample internal validation)

Supplement to *Emergency Medicine Reports*, October 1, 2018: "Syncope." Author: Frank J. Edwards, MD, FACEP, Program Director, Emergency Medicine Residency, Arnot Ogden Medical Center, Elmira, NY.

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