Diagnosis and Evaluation of Syncope in the Emergency Department

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Syncope accounts for approximately 1.3% of all presentations to the emergency department (ED). Determining the exact cause remains a diagnostic challenge, even with an in-patient admission and comprehensive work-up. Studies have shown that the cause of syncope is diagnosed with variable degrees of certainty in only about 50% of patients after an initial ED evaluation,1,2 and about 30% of patients remain undiagnosed on discharge from a hospital admission.3–5 However, with a careful history and physical examination and select diagnostic studies, the differential diagnosis can be narrowed, and physicians can effectively risk stratify patients to determine whether an in-patient admission is necessary. One group of investigators concluded that a reasonable diagnosis can be made in about 80% of ED patients with a more focused history and physical examination and directed investigations.3 For those without a clear diagnosis, physicians can effectively risk stratify patients to determine whether in-patient admission is necessary. This article reviews the diagnosis and ED work-up of syncope, the different classifications of syncope, and prognosis. The use of specific decision rules in risk stratification and syncope in the pediatric population are discussed in another article.

CLASSIFICATION AND DIFFERENTIAL DIAGNOSIS

Common Classifications

There are 5 major classifications of syncope. Their frequencies from the Framingham Heart Study of nearly 8000 patients are6:

- Reflex-mediated (21%)
- Orthostatic (9%)
• Neurologic (4%)
• Unknown (including psychiatric causes) (37%).

While it may not be possible to determine the cause of syncope in the ED, it is useful to divide the differential diagnoses into benign and dangerous to guide diagnostic work-up and disposition, as shown in Table 1.

**Benign Causes**

Reflex-mediated and orthostatic causes are usually benign causes of syncope. After initial evaluation, these patients rarely require admission. The exception is the elderly patient who may require admission to rule out more malignant mechanisms. However, repeat benign episodes that are very similar to past contexts by history usually do not require extensive work-up and admission.

**Reflex-mediated syncope**

Reflex-mediated syncope is considered the same as the term “neurally mediated syncope.” The mechanism is an inappropriate neural control over the circulation resulting in vasodilation, with or without bradycardia. Under the classification of reflex-mediated syncope, there are several types, including vasovagal, situational, and carotid sinus hypersensitivity.

Vasovagal syncope is induced by emotion or pain. It is sometimes termed “neurocardiogenic,” and it is sometimes used as a synonym for reflex-mediated syncope. Situational syncope is also in this category and is induced by such physiologic mechanisms as cough, micturition, or defection. Patients diagnosed with vasovagal syncope have excellent prognosis. Patients believed to have vasovagal syncope in the Framingham study had a lower long-term mortality compared with those patients followed who never had syncope. However, it is problematic that vagal symptoms tend to be subjective and vague, and physicians do not always agree on their diagnosis or presence. Nonetheless, those given the diagnosis are clearly at low risk for mortality or significant morbidity.

Carotid sinus hypersensitivity is a reflex-mediated syncope and should be considered in patients who are older and have syncope in the context of stimulation of their carotid arteries. Some examples include wearing a tight necktie, shaving the neck, or a history of neck malignancy. Carotid sinus hypersensitivity is more common in the elderly, men, and those with structural heart disease.

<table>
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<tr>
<th>Table 1</th>
<th>Benign versus malignant causes of syncope</th>
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<tr>
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<td>Dangerous</td>
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<tr>
<td>Reflex-mediated (neurally mediated)</td>
<td>Cardiac</td>
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<tr>
<td>Vasovagal</td>
<td>Arrhythmias</td>
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<td>Situational</td>
<td>Ischemia</td>
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<td>Carotid sinus hypersensitivity</td>
<td>Structural cardiopulmonary abnormalities</td>
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<td>Orthostatic (autonomic failure)</td>
<td>Neurologic</td>
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<tr>
<td>Medications</td>
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<tr>
<td>Postprandial hypotension</td>
<td>Subarachnoid hemorrhage</td>
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<tr>
<td>Intravascular volume loss</td>
<td>Subclavian steal</td>
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<td>Migraines</td>
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Even though syncope from carotid sinus hypersensitivity is usually benign, cardiac pacing can be useful in at-risk patients.\textsuperscript{10,11}

**Orthostatic syncope**

Orthostatic syncope is the same as autonomic failure. The mechanism in orthostatic syncope is an insufficient autonomic response to counter a drop in cardiac output. The cardinal difference between orthostatic and reflex-mediated syncope is that in orthostatic syncope, the autonomic nervous system attempts to control blood pressure with increased heart rate but fails, whereas in reflex-mediated syncope the autonomic nervous system acts inappropriately resulting in reflex bradycardia and vasodilation.

Most causes of orthostatic syncope are benign. Orthostasis can be caused by medications such as antihypertensives, diuretics, and antidepressants. It can be triggered by postprandial hypotension, which is thought to be secondary to various causes such as impaired baroreflex compensation for splanchnic blood pooling during digestion, inadequate postprandial increases in cardiac output, insulin-induced vasodilation, and release of vasodilatory gastrointestinal peptides.\textsuperscript{12}

Orthostatic syncope may also be caused by intravascular volume depletion such as in dehydration or blood loss. This type of syncope is often considered benign, as the treatment may involve simple repletion of intravascular volume. However, orthostatic syncope can also result from life-threatening hemorrhage, such as from trauma, gastrointestinal bleeding, retroperitoneal bleeding, a ruptured spleen, a ruptured aortic aneurysm, a ruptured ectopic pregnancy, or a ruptured ovarian cyst.

**Psychiatric causes of syncope**

Nearly 40\% of patients presenting with syncope may not have a determinable organic cause for their presentation. Psychiatric causes are often by default classified under this category. In one study, the most frequent psychiatric diagnoses were generalized anxiety disorder and major depressive disorder.\textsuperscript{13} However, patients with hypoxia and poor cerebral confusion may also appear similarly confused or anxious.

**Dangerous Causes**

The physician must recognize the potential for cardiac or neurologic syncope, as both are considered dangerous causes and should be reasonably excluded before discharge.

**Cardiac syncope**

Cardiac syncope is the most dangerous cause of syncope and is the reason for most syncope admissions. Without intervention, the 6-month mortality associated with cardiac causes of syncope is greater than 10\%.\textsuperscript{3,14} Fortunately, most of these can be identified by risk stratifying patients in the ED with a thorough history, physical examination, and an electrocardiogram (ECG).

The basic mechanism underlying cardiac syncope is the inadequacy of cardiac output to maintain cerebral perfusion. Often, an underlying structural abnormality or preexisting heart condition is the primary cause or contributing cause of cardiac syncope. These abnormalities include dysrhythmias, ischemia, and structural cardiovascular lesions. Dysrhythmias are clearly the most common and the most dangerous cause of cardiac syncope, although most lethal arrhythmias such as ventricular tachycardias arise from structural heart lesions. Ischemia is an infrequent cause of syncope (<3\% of all presentations)\textsuperscript{15} and rarely occurs alone without concomitant chest pain, and ECG findings. Primary structural lesions with reduced cardiac output such as valvular disease and cardiomyopathies are yet another
important cause. Pulmonary embolism can be included here as a rare but serious cause of syncope when cardiac output is significantly obstructed.

**Neurologic syncope**

True syncope is defined as a transient loss of consciousness (LOC) with return to baseline neurologic function. Although older patients are often admitted to rule out cerebrovascular accidents or transient ischemic attacks (TIA) as the cause of their syncope, isolated LOC without accompanying neurologic signs or symptoms is actually a rare cause of syncope, usually reported as 1% to 3%. For syncope to occur, transient interruption of blood flow to the brain stem or cerebral cortex must take place. Subarachnoid hemorrhage is another perilous cause of syncope. These patients almost always complain of headache or have other focal neurologic findings on examination. In addition, because many syncope patients sustain a head injury related to falling, there is a tendency to associate subarachnoid hemorrhage with syncope, when in fact syncope may be posttraumatic and not primary.

Subclavian steal may also cause syncope. In this syndrome, stenosis of the subclavian artery results in the poststenotic portion of the artery receiving additional blood supply through the ipsilateral vertebral artery, causing blood in that artery to flow inferiorly, and drawing blood away from the brainstem. These patients usually present with different blood pressures in each arm. They can also experience a LOC after using their arm on the affected side, usually the left.

Migraine headache is a benign cause of neurologic syncope and often presents with an aura. It is a diagnosis of exclusion in the correct clinical context and requires ruling out other, more malignant causes. Furthermore, there is likely an orthostatic, reflex-mediated, or situational component to the root cause of syncope in patients with migraine.

**DIAGNOSIS**

Nearly half of syncope patients can be diagnosed on the grounds of history and physical examination alone. Recent studies suggest that yield can be increased with better and more focused and detailed history and physical examination in the ED.

**History**

The most important first step in evaluating a patient with LOC or near LOC is to establish whether this is true syncope. Syncope is a symptom complex that is composed of a brief LOC associated with an inability to maintain postural tone that spontaneously and completely resolves to baseline neurologic function without resuscitation. It is, by definition, transient. Syncope is distinct from vertigo, seizures, stroke, coma, and other states of altered consciousness. Near syncope has the same implications as syncope and deserves the same work-up.

Syncope can be an extremely broad symptom. When patients present with the chief complaint of passing out or fainting, that might be very different from a physician’s idea of syncope. Thus, it is imperative to elicit a comprehensive history. The circumstances surrounding a syncopal event are most important and even minute details may be significant. Any witnesses to the incident should be interviewed. The most important aspects of a patient’s history are listed in Box 1.

**Syncope versus other altered mental states**

The first branch point for the physician approaching a patient who presents with LOC is to establish whether this is truly syncope or not. One of the biggest challenges facing clinicians is discerning between syncope and seizure. In several studies, 20% to 30%
of patients initially diagnosed with seizures were subsequently diagnosed with syncope. These patients can present with sudden cardiac death when cardiac syncope is not initially considered. Furthermore, some seizure conditions are associated with prolonged QT and sudden death. In general, cardiac abnormalities should be considered in patients with seizure with the minimum of a history, physical examination, and ECG. Table 2 lists important clues when trying to differentiate the 2 entities.

Aside from seizures, other neurologic conditions such as strokes, metabolic disorders such as hypoglycemia, and toxic causes such as alcohol can lead to LOC and mimic syncope. Further diagnostic strategies are discussed later in this article and other articles in this issue. However, these conditions are not transient and usually present with persistent altered mental status and should not, by proper definition, be termed syncope.

Age
Younger patients are more likely to have a benign cause of syncope but can still have rare life-threatening causes. One should consider arrhythmias in young patients, such as the athlete who presents after a syncopal event while exercising or the young patient with a question of a first-time seizure. At the opposite end of the age spectrum, elderly patients tend to have more risk factors and comorbidities categorizing them as higher risk, but using a discrete age cut-off for high and low risk is nonspecific and

<table>
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<th>Box 1 Detailed history</th>
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<tr>
<td>Age</td>
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<td>Prodrome</td>
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<td>Previous episodes</td>
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Table 2 Syncope versus seizure

<table>
<thead>
<tr>
<th>Syncope</th>
<th>Seizure</th>
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<tr>
<td>No aura</td>
<td>Aura</td>
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<tr>
<td>Post-LOC jerks</td>
<td>Pre-LOC jerks</td>
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<tr>
<td>Asynchronous jerks</td>
<td>Synchronous jerks</td>
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<tr>
<td>Tongue bite at tip</td>
<td>Tongue bite lateral</td>
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<tr>
<td>Flaccid</td>
<td>Stiff</td>
</tr>
<tr>
<td>Quick recovery</td>
<td>Postictal</td>
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<tr>
<td>No anion gap acidosis</td>
<td>Transient anion gap acidosis</td>
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difficult. Several studies have suggested using 60 or 70 years of age as the upper limit of low risk.\textsuperscript{26,27} Another study assessed 1 month outcomes in syncope patients older than 50 years of age, and showed no significant short-term outcomes in patients with unremarkable ED work-ups.\textsuperscript{16} Given the variation in studies, it is safe to say that age alone is a marker for increased morbidity and mortality regardless of the patient’s presentation, and it may be more helpful to use age as a gradual continuum in risk stratification associated with other factors, rather than choosing a specific cut-off.

**Position**
The position the patient was in when the event occurred can often provide an important clue to the cause. Syncope while lying supine may be more likely caused by an arrhythmogenic trigger. Positional changes, such as from sitting to standing, may be indicative of orthostasis. The position the patient assumed on recovery that improved the symptoms may also be helpful. In pregnant women, inferior vena cava compression is not an uncommon cause of syncope. However, position is only a small part of the overall history; that is, a patient may still have a malignant cardiogenic cause for the syncope, even if the event occurred while changing positions.

**Prodrome**
Any prodrome must be elicited from the patient’s history. This includes any auras, which may distinguish syncope from seizure. Other clues in the history that may aid in differentiating syncope from seizure are shown in Table 2. A sudden LOC without preceding symptoms, is more suggestive of arrhythmia.\textsuperscript{28,29} One study evaluating patients with implantable loop recorders found that arrhythmia was the cause in 64\% of patients experiencing a sudden LOC without prodrome.\textsuperscript{30} In another study, two-thirds of patients with benign vasovagal syncope had prodromal symptoms elicited from their history, most often a feeling of warmth, diaphoresis, or nausea.\textsuperscript{31}

**Triggers**
Asking the patient about circumstances surrounding the syncopal event may be helpful, especially in determining whether it was neurally mediated. Important hints include an emotional stimulus, pain, or fear. Situational triggers such as cough, micturition, or defecation are common in reflex-mediated syncope. Carotid sinus hypersensitivity can often be elicited by asking the patient about neckties, tight collars, or turning their head while driving a vehicle in reverse.

Patients experiencing syncope while exerting themselves are a concern for an arrhythmia or significant outflow tract obstruction from hypertrophic cardiomyopathy.\textsuperscript{32,33} This is a consideration in the classic example of the young athlete who has a syncopal event while playing basketball, such as the well-documented cases of Hank Gathers and Reggie Lewis.

**Associated symptoms**
Often patients are so startled and distressed by the LOC that they may neglect to give a complete history; the physician needs to perform a complete review of systems, with emphasis on palpitations, chest pain, shortness of breath, headache, paresthesias, slurred speech, aphasia, and focal weakness. The review of neurologic symptoms helps separate syncope from other forms of altered consciousness such as stroke. Although TIA that present with isolated LOC are technically considered syncope, as the patient returns to neurologic baseline without resuscitation, it is important to note that TIA presenting without any other neurologic deficits is exceedingly rare (<1\%).\textsuperscript{34}
In addition, chest pain accompanying syncope may indicate an acute coronary syndrome. Shortness of breath may suggest pulmonary embolus. Headache, in the appropriate context, may prompt one to investigate for subarachnoid hemorrhage. Tongue biting and incontinence may indicate seizure rather than syncope, as shown in Table 2. By extracting these details from the history, the physician will be able to narrow the differential diagnosis and pursue a directed diagnostic work-up. The work-up of lone syncope is very different from the work-up of syncope with associated symptoms.

Determining the duration of altered consciousness will again help the physician decide if the event was truly syncope or another altered mental state. True syncope exists only when patients’ symptoms are transient and self-limited. Any patient who presents in a postictal state or with persistent neurologic symptoms did not, by definition, have a syncopal event. Patients who present with symptoms of near syncope should be treated as if they had syncope. Near syncope and true syncope are on a spectrum, with similar underlying causes resulting in decreased blood flow to the brain.

**Associated injury during a syncopal event**
Contrary to common belief, injuries suffered from syncope do not help in determining whether the inciting mechanism is dangerous or benign. It is often thought that those without prodrome are more likely to be cardiac and would therefore be more likely to have associated injuries. However, those with prodrome who ignore their prodrome can also incur significant injury. In one study of patients in whom the diagnosis of vasovagal syncope was established, 10% suffered injury severe enough to require hospitalization and nearly 30% reported at least one traumatic injury.35 Another study looking at associated injuries among consecutive ED patients found no significant differences between those with serious and benign outcomes.36

Although injury may not necessarily signify a malignant cause, patients may still require hospitalization for their specific injury and to prevent further injury from future syncopal events.

**Previous episodes**
Patients’ previous episodes of syncope, and the context in which they occurred, are especially helpful in determining cause and the need for admission. If patients have had complete work-ups in the past demonstrating a benign cause of syncope, they can often be discharged without additional extensive work-ups.

The number of previous episodes and duration between episodes may also help differentiate benign and malignant causes. Several episodes over a short period of time in a patient without a past history may suggest a more significant underlying disorder. In contrast, a single episode or multiple episodes over many years may be more suggestive of a benign cause.

**Family history**
A family history of sudden cardiac death is an important historical feature to elicit and document. A positive family history, especially in younger patients should lead to further cardiac work-up.37 Family history can be particularly important in determining inherited cardiac disorders that may often be fatal. Classic examples of this include hypertrophic cardiomyopathy and prolonged or short QT syndromes.38–40

**Physical Examination**
Although the history is of tremendous value, especially because patients are at their neurologic baseline by the time of ED presentation, a thorough physical examination
can be useful and sometimes diagnostic. A physical examination, like the history, can help the physician distinguish between true syncope and other altered states of consciousness.

**Abnormal vital signs**

Transient hypotension and an abnormal heart rate are the most common and immediate causes of many syncopal events. Abnormal vital signs during the event will often normalize on presentation to the ED; however, persistently abnormal vital signs should be of concern. For example, a low oxygen saturation rate or tachypnea may be signs of congestive heart failure (CHF) or pulmonary embolism.

In order for orthostatic vital signs to be helpful, patients who are orthostatic by number ought to have associated symptoms and a supporting history. These signs should be used to supplement and confirm decision making, as opposed to existing as a sole diagnostic tool. Multiples studies have confirmed that orthostatic vital signs are neither sensitive nor specific. Up to 40% of asymptomatic outpatients older than 70 years of age have positive orthostatic vital signs. In another study, 16% of 2500 people of all ages in a population-based study had orthostatic changes in vital signs. Thirty-one percent of syncope patients of all ages have orthostatic vital signs, regardless of the cause of the syncope. In a recent retrospective review, orthostatic vital signs were found to be cost-effective from an inpatient perspective after other more expensive tests were negative, but again, orthostatic vital signs cannot stand alone to confirm a diagnosis in medium- and high-risk patients to justify discharge without further testing in the ED.

**Head-to-toe physical examination**

Any bleeding or lacerations in the mouth suggestive of tongue biting should be investigated. Tongue biting on the lateral side may indicate seizure activity rather than a syncopal event. Although tongue biting is neither sensitive nor specific for seizure activity, it rarely occurs in syncope, and if it does, it is most often at the tip.

Carotid artery stenosis can be found with careful auscultation of the neck, although severe stenosis may not produce a bruit. Carotid murmurs radiating from the precordium are usually caused by aortic stenosis. Increased jugular venous pressure may suggest CHF.

In the appropriate context, as suggested by age and history, carotid sinus massage can be attempted in certain patients, as shown in Fig. 1. Carotid sinus massage should not be performed in patients with or suspected to have had a myocardial infarction, stroke, or TIA in the past 3 months. Relative contraindications include older age, carotid bruit heard on examination, or history of ventricular tachycardia or

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**3 Steps to Carotid Sinus Massage**

1. Patient should lie supine for 5 minutes.
2. Massage over point of maximal carotid impulse, medial to sternomastoid muscle at level of upper border of thyroid cartilage.
3. Massage for 5 seconds on both sides (not simultaneously) with 1-minute interval between sides.

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**Fig. 1.** Carotid sinus massage.
fibrillation. The maneuver should be performed by an experienced physician, with careful cardiac and respiratory monitoring.

Any findings on the cardiac examination suggestive of structural heart disease or CHF deserve special attention. Patients with hypertrophic cardiomyopathy can have a laterally displaced and abnormally forceful, apical precordial impulse. They may also have an S₃ and S₄.

Murmurs are especially important to identify structural or valvular heart disease. About 30% to 40% of patients with hypertrophic cardiomyopathy have midsystolic ejection murmurs, thought to be secondary to the systolic anterior motion of the mitral valve on the thickened interventricular septum, producing high-velocity flow. These murmurs are best appreciated over the left second or third intercostal space along the sternal border. Aortic stenosis is yet another urgent diagnosis that can be easily diagnosed in the ED with a careful cardiac examination. Classically, aortic stenosis produces a harsh crescendo-decrescendo murmur heard loudest at the right upper sternal border, which increases with squatting and radiates to both carotid arteries. In addition, these patients tend to present with narrow pulse pressures.

Patients with CHF can have an S₃ or S₄ that is best heard in the lateral decubitus position. These findings, especially if associated with crackles or increased jugular venous pressure suggest CHF. Although wheezes are not a sign of CHF, new wheezes could be concerning for pulmonary embolism. All findings on cardiac examination suggesting structural heart disease or CHF require further investigation. An echocardiogram is usually the best test to determine the significance of any of these findings.

The abdominal examination can also be useful in diagnosing any orthostatic causes of syncope resulting from hemorrhage. These include ruptured abdominal aortic aneurysm, ruptured spleen, gastrointestinal bleeding, and retroperitoneal bleeding. Any abnormalities in the examination should prompt the physician to do a rectal examination.

Patients with syncope, by definition, return to baseline neurologic function. A thorough neurologic examination should be performed to identify any focal abnormalities that may suggest stroke, TIA, or other neurologic diagnoses.

The physician must screen for any traumatic injury. Although this may not differentiate between benign and more malignant causes of syncope, related traumatic injuries may require admission. Common injuries include facial fractures, hip fractures, wrist fractures, and subdural hematomas from syncope-related falls. Special consideration should be given to even minor head injuries in elderly patients on anticoagulation and antiplatelet agents.

### Diagnostic Tests

**ECG**

Level A recommendations are generally accepted principles for patient management that reflect a high degree of clinical certainty (ie, based on strength of evidence Class I or overwhelming evidence from strength of evidence Class II studies that directly address all of the issues.) The American College of Emergency Physicians recommends obtaining a 12-lead ECG in all patients presenting to the ED with syncope. Although the yield may be low (estimated to be < 5%), it is a noninvasive, inexpensive, and quick test that can detect life-threatening conditions. It is also the most consistent test used in risk stratification rules and guidelines.

There are many definitions about what constitutes an abnormal ECG in syncope, and all abnormalities are not of equal risk. For example, up to 45% of bundle branch blocks may be associated with cardiac causes of syncope, whereas nonspecific changes are less likely to be problematic. The primary reason why such variability
exists in the effectiveness of clinical decision rules is because there is such a wide spectrum of abnormal ECG definitions, resulting in the subjectivity of ECG interpretation.\textsuperscript{53–55}

**Laboratory data**

Laboratory tests should only be ordered based on the history, physical examination, and ECG. Multiple studies have concluded that routine, unguided laboratory tests are not valuable.\textsuperscript{31,36,37,56} Measuring serum electrolytes can be useful in the correct clinical context. For example, a serum chemistry panel can help further validate dehydration as a likely cause of syncope. Furthermore, a hematocrit less than 30% has been shown to be a predictor of adverse events.\textsuperscript{7}

Qualitative urine human chorionic gonadotropin (HCG) should be performed on all women of childbearing age. This inexpensive and relatively quick test may help detect normal or ectopic pregnancy, either of which can present with true syncope.\textsuperscript{57,58} If the urine HCG is positive, the physician should have a low threshold for ruling out an ectopic pregnancy with a pelvic ultrasound.

Although many lone syncope patients are admitted to rule out cardiac ischemia, studies have shown this to be very low yield. Cardiac enzymes are positive in less than 3% of patients with syncope. Moreover, nearly all of these patients with a significant cardiac cause related to their syncope present with chest pain or ECG changes.\textsuperscript{15} One study demonstrated that of more than 300 syncope patients (65 years and older), 50% had cardiac enzymes drawn; of those patients, only 3 were positive. Furthermore, of those 3 patients, 2 presented with chest pain and ECG changes, and the third patient had dementia preventing a thorough history but did have a new left bundle branch block.\textsuperscript{59} In another study, ED management was only affected in 1% of 2000 patients admitted with syncope who had their cardiac enzymes cycled.\textsuperscript{32} Therefore, cardiac enzymes should be ordered only if deemed necessary after a careful history, physical examination, and an ECG. Thus, it is not recommended to admit lone syncope patients without chest pain to complete two sets of cardiac enzymes when they otherwise would have been discharged.

**Further diagnostic studies**

Echocardiography can be a useful test in the evaluation of patients with syncope because people with structural heart disease and/or impaired ejection fraction are at serious risk. However it is not recommended as a routine test and is unlikely to identify patients at risk that could not be identified by history examination and ECG.\textsuperscript{4,60} If a patient is at risk or needs further investigation for suspected structural or valvular heart disease or CHF, an echo can be a very useful next test.

Any neuroimaging or investigation should not be used routinely in patients with syncope unless indicated and guided after a history and physical examination. Computed tomography and magnetic resonance imaging scans of the head are generally low-yield tests in the context of a normal physical examination.\textsuperscript{61} Should the history suggest that head imaging may be necessary secondary to trauma or anticoagulation, the Canadian or the New Orleans head CT rules can provide guidance.\textsuperscript{62} Likewise, if a TIA is suspected based on history and physical examination, then neuroimaging may be appropriate.

Similarly, neurovascular ultrasound such as transcranial Doppler and carotid ultrasound have also been found to be nondiagnostic with a normal physical examination. In 1 study of 140 syncope patients in a stroke-age population, 95% of those with positive neurovascular ultrasound results had marked findings on physical examination.
Only 1% of those with findings on imaging had lesions that could have been contributing to syncope, but none of the lesions were concluded to be the primary cause.\textsuperscript{63}

As indicated in Fig. 2, there are many signs and symptoms that can help the physician differentiate syncope from seizure. However, at times, the two can be challenging.

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<th>Cardiac Abnormality</th>
<th>Classic ECG Findings</th>
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<tr>
<td>Hypertrophic Cardiomyopathy</td>
<td>LVH, large voltages, prominent Q waves.</td>
<td><img src="image1" alt="ECG" /></td>
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<tr>
<td>Brugada</td>
<td>R888 with ST elevation in right precordial leads</td>
<td><img src="image2" alt="ECG" /></td>
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<tr>
<td>Long QT</td>
<td>QTc&gt;450ms in males, QTc&gt;470ms in females</td>
<td><img src="image3" alt="ECG" /></td>
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<tr>
<td>Short QT</td>
<td>QTc&lt;330ms</td>
<td><img src="image4" alt="ECG" /></td>
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<tr>
<td>Mobitz Type II 2° AV Block</td>
<td>constant PR interval, periodic dropped QRS</td>
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<tr>
<td>Wolff-Parkinson-White</td>
<td>Delta wave, short PR, prolonged QRS</td>
<td><img src="image6" alt="ECG" /></td>
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<td>Arrhythmogenic Right Ventricle Dysplasia (ARVD)</td>
<td>Epsilon wave, inverted T waves in precordial leads</td>
<td><img src="image7" alt="ECG" /></td>
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Fig. 2. Clinically significant electrocardiograms.
to distinguish. In these rare cases, an electroencephalogram may be helpful, but in general they are low yield.

Admission for telemetry
While in the ED, all syncope patients should be placed on continuous cardiac monitoring. Patients, especially the elderly, are often admitted for cardiac monitoring, searching for arrhythmias, bradycardia, or pauses on telemetry. Most patients should have some high-risk finding on history, examination, or ECG to warrant admission. For low-risk patients, physicians can arrange outpatient Holter monitoring, when practical. This includes younger patients with normal ECGs and palpitations who are suspected of having nonmalignant rhythms such as supraventricular tachycardia or paroxysmal atrial fibrillation. For those with suspected vagal syncope, telemetry is very low yield (<1%).

MANAGEMENT
Management of syncope patients is guided by diagnosis. Those with malignant rhythms need admission for urgent placement of pacemakers or implantable defibrillators. These patients need to be stabilized in the ED with medications, defibrillation, and temporary pacemaking, as indicated. Similarly those with pulmonary embolism or strokes need anticoagulation and thrombolysis as indicated. In general, those with a clear diagnosis have clear management pathways. Those without a clear diagnosis have less defined management pathways. Risk stratification of these patients into high and low risk to guide further investigation and admission seems to be the best way to manage these patients efficiently.

SUMMARY
With a careful history, physical examination, and directed investigation, physicians can determine the likely cause of syncope in more than 50% and perhaps up to 80% of patients. Understanding the cause of syncope allows clinicians to determine the disposition of high- and low-risk patients. Patients with a potential malignant cause, such as a cardiac or neurologic condition, should be treated and admitted. Those with benign causes can be safely discharged. Those remaining patients with an unclear cause should be accurately risk stratified, and their disposition determined by their risk factors as discussed in the next article.

REFERENCES


