Approach to the adult patient with syncope in the emergency department

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INTRODUCTION

Syncope is a transient loss of consciousness associated with loss of postural tone, followed quickly by a spontaneous return to baseline neurologic function requiring no resuscitative efforts. Syncope may be caused by benign or life-threatening conditions and it is a relatively common reason for presenting to the emergency department (ED). Often, the underlying cause of a syncopal episode cannot be clearly identified in the ED, and the primary responsibility of the ED clinician becomes determining which patients are at high risk for adverse outcomes.

This topic review will discuss how to evaluate and manage patients presenting to the ED with syncope. Detailed discussions of specific types of syncope and the evaluation of syncope in children and adults are found separately.

- (See "Syncope in adults: Epidemiology, pathogenesis, and etiologies".)
- (See "Syncope in adults: Clinical manifestations and initial diagnostic evaluation".)
- (See "Reflex syncope in adults and adolescents: Clinical presentation and diagnostic evaluation".)
- (See "Emergency evaluation of syncope in children and adolescents".)
- (See "Causes of syncope in children and adolescents".)

TERMINOLOGY AND EPIDEMIOLOGY

Syncope is a transient loss of consciousness associated with loss of postural tone, followed by a spontaneous return to baseline neurologic function requiring no resuscitative efforts. The underlying mechanism is global hypoperfusion of both the cerebral cortices or focal hypoperfusion of the reticular activating system. Syncope is not to be confused with a loss of consciousness associated with altered mental status or stroke, or with vague dizziness and chronic lightheadedness. Presyncope (ie, near syncope or near loss of consciousness) and true syncope should be considered a spectrum of the same symptom. Although presyncope is less dramatic, ED clinicians should approach the evaluation in a similar fashion [1-4]. When researchers have made a clear effort to distinguish true presyncope from dizziness, ataxia, or vague lightheadedness, the outcomes of syncope and presyncope have been found to be similar in terms of significant underlying causes and outcomes [1]. The terminology and epidemiology of syncope is explored in greater detail separately. (See "Syncope in adults: Epidemiology, pathogenesis, and etiologies".)

Syncope is a common presenting symptom in the ED, accounting for 1 to 2 percent of all ED visits and hospital admissions in the United States [5-9]. Lifetime prevalence rates range from 10.5 to 19 percent [10,11]. Although most causes are benign and self-limited, others are associated with significant morbidity and mortality, particularly among older adults [12]. The Framingham study identified patients with a cardiac cause for syncope as having twice the risk of death as those without syncope and patients with a neurologic cause as having a 50 percent increased risk [10]. Those with an unknown cause of syncope still has a 30 percent increased risk of death, whereas patients with vasovagal (ie, vasovagal) syncope are at no increased risk.

The differential diagnosis is broad, and management focuses on the underlying cause when this is discernible. However, during the ED evaluation, even though the cause of syncope often remains unclear, it is important to try to determine the cause and focus management on risk stratification to differentiate between patients safe for discharge and those who require immediate investigation and in-hospital management. (See 'Differential diagnosis' below and 'Risk stratification' below.)

DIFFERENTIAL DIAGNOSIS

After reviewing the history, physical examination findings, and the electrocardiogram (ECG), physicians in the ED are able to determine a clear underlying diagnosis only about 50 percent of the time [13,14]. Patients often remain undiagnosed despite exhaustive diagnostic testing [7,15-

17]. Tables listing the most dangerous and most common etiologies of syncope are provided (table 1 and table 2 and table 3).

Life-threatening conditions — The primary responsibility of the emergency clinician is to assess whether a life-threatening cause of syncope is present and to provide appropriate management and disposition. The most important causes to consider are cardiac syncope, blood loss, pulmonary embolism, and subarachnoid hemorrhage. Other conditions such as seizure, stroke, and head injury do not meet the technical definition of syncope but should be considered during the initial assessment.

- **Cardiac syncope** – Cardiac causes are the most common life-threatening conditions associated with syncope and thus the most important to diagnose or predict. They include arrhythmia, ischemia, structural/valvular abnormalities (eg, aortic stenosis, particularly in older adults; idiopathic hypertrophic heart disease), cardiac tamponade, and pacemaker malfunction. Prospective studies of short-term and one-year outcomes following syncope have found patients with cardiac syncope to be at significant risk for sudden death [6,18]. Patients with a history of cardiac disease, particularly heart failure, were at greatest risk [19]. (See "Syncope in adults: Epidemiology, pathogenesis, and etiologies", section on 'Causes of syncope' and "Syncope in adults: Management and prognosis", section on 'Treatment'.)

- **Hemorrhage** – Large blood loss, particularly acute severe hemorrhage, can manifest as syncope. Important potential causes include trauma, gastrointestinal bleeding, ruptured aortic aneurysm, ruptured ovarian cyst, ruptured ectopic pregnancy, and ruptured spleen. (See "Approach to acute lower gastrointestinal bleeding in adults" and "Evaluation and management of ruptured ovarian cyst" and "Ectopic pregnancy: Clinical manifestations and diagnosis" and "Management of splenic injury in the adult trauma patient" and "Management of symptomatic (non-ruptured) and ruptured abdominal aortic aneurysm", section on 'Introduction' and "Approach to shock in the adult trauma patient".)

- **Pulmonary embolism** – Hemodynamically significant pulmonary embolism is a relatively uncommon but well-documented and important cause of syncope. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Clinical presentation'.)

- **Subarachnoid hemorrhage** – Patients presenting with syncope associated with headache require evaluation for a possible subarachnoid hemorrhage. (See "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis".)

Common conditions

- **Vasovagal syncope** – Vasovagal syncope, a type of reflex syncope, is the most common cause of syncope, accounting for 25 to 65 percent of cases [14]. Patients diagnosed with vasovagal syncope have an excellent prognosis with no increase in long-term mortality or morbidity [10].

  Autonomic activation causes vasovagal syncope. Three types of responses are seen: a cardioinhibitory response, a vasodepressor response, and a mixed response with features of both. Significant bradycardia and/or hypotension accompany the acute loss of consciousness. Potential triggers are numerous, and determination of the underlying cause is often made in the outpatient setting. (See "Reflex syncope in adults and adolescents: Clinical presentation and diagnostic evaluation".)

  Most patients with vasovagal syncope experience a slow, progressive prodrome that may include some combination of dizziness or lightheadedness, a sense of warmth, pallor, nausea and vomiting, abdominal pain, changes in vision, or diaphoresis associated with some precipitating event. Examples include micturition or defecation syncope, situational syncope (eg, while having blood drawn), or cough-mediated syncope.

- **Carotid sinus hypersensitivity** – Carotid sinus hypersensitivity is a reflex-mediated variant of vasovagal syncope, resulting from pressure at the carotid sinus. External pressure to the neck can induce this reflex response, causing bradycardia and hypotension. Common causes include shaving, a tight collar, and turning of the head. Clinicians should consider carotid hypersensitivity in older patients with recurrent syncope and a prior negative cardiac workup. (See "Carotid sinus hypersensitivity and carotid sinus syndrome".)

- **Orthostasis** – Orthostatic syncope comprises between 5 and 24 percent of syncope cases and is defined by syncope associated with a drop in systolic blood pressure of 20 mmHg or more with a positional change (lying to sitting or sitting to standing). Orthostatic syncope is often associated with a reflex tachycardia of more than 20 beats per minute. Orthostasis is most often caused by a loss of intravascular volume, which can be exacerbated by instability of the autonomic nervous system. Nevertheless, clinicians should remain cautious because orthostasis can occur with cardiac syncope, acute gastrointestinal bleeding, or autonomic insufficiency, particularly in older adults. Syncope from orthostatic hypotension should be a diagnosis of exclusion in the ED, reserved for low-risk patients who have symptoms consistent with the diagnosis. (See "Mechanisms, causes, and evaluation of orthostatic hypotension".)

Orthostatic vital signs are neither sensitive nor specific in assessing volume status or diagnosing orthostatic syncope [20-22]. Symptomatic orthostasis is most important. Many patients become symptomatic if their systolic blood pressure drops below 90 mmHg with or without a corresponding drop in blood pressure or elevated heart rate sufficient to meet the criteria for orthostatic syncope. While a large portion of the population meets the definition of orthostasis, they do not necessarily have syncope. Older adults, pregnant women, and patients taking drugs with vasodilating effects are predisposed to develop symptomatic orthostasis.

- **Medications** – The effects of medications account for 5 to 15 percent of syncopal events. The mechanism can be orthostasis or cardiotoxicity. Medications often implicated include calcium channel blockers, beta blockers, alpha blockers, nitrates, antiarrhythmics, diuretics (affecting volume status and electrolyte concentrations), and medications affecting the QTc interval (eg, antipsychotics and antiemetics) (table 2) [23].

Other conditions

- **Neurologic syncope** – True syncope is defined by an immediate, spontaneous return to baseline function following loss of consciousness, without new focal neurologic findings. Thus, true neurologic syncope reflecting underlying neurovascular disease is rare.

  Examples of neurologic syncope include subarachnoid hemorrhage, transient ischemic attack, subclavian steal syndrome, and complex migraine headache. Stroke and transient ischemic attacks generally cause focal neurologic deficits that do not recover rapidly or completely. Often, patients with cerebrovascular disease convey a history of nonsyncopal episodes featuring neurologic deficits, possibly including diplopia, vertigo, focal weakness, or numbness.

  Syncope may be misconstrued as seizure because many patients with transient loss of consciousness have brief convulsive episodes secondary to cerebral hypoperfusion, particularly if bystanders or objects keep them upright. Syncope can usually be differentiated from seizure by the brevity of the convulsions; the absence of epileptic aura, urinary or fecal incontinence, and tongue biting; and the lack a true postictal phase (typically five minutes or longer). (See "Syncope in adults: Epidemiology, pathogenesis, and etiologies".)

- **Psychiatric syncope** – Anxiety and panic disorders can cause situational syncope. Emergency clinicians must be cautious when attributing syncope to psychiatric causes. Patients with hypoxia, inadequate cerebral perfusion, or other medical conditions may

appear confused or anxious. Patients with psychiatric syncope are generally young, without cardiac disease, and complain of multiple episodes [24].

- **Drug-induced loss of consciousness** – Drugs of abuse and alcohol may cause a transient loss of consciousness, but generally, these patients manifest signs of toxicity and do not spontaneously return to normal neurologic function immediately after regaining consciousness. Alcohol can also cause symptomatic orthostasis by impairing vasoconstriction [25].

- **Metabolic** – Metabolic causes of syncope include hypoglycemia and hypoxia. Electrolyte abnormalities secondary to renal injury or other conditions may precipitate syncope due to dysrhythmia.

- **Rare causes** – Rare causes of syncope include atrial myxoma, Takayasu arteritis, systemic mastocytosis, and carcinoid. Anaphylaxis can involve syncope and loss of consciousness, and both patients and witnesses sometimes overlook or forget the more subtle, earlier symptoms, such as flushing, itching, hives, cough, bronchospasm, or abdominal cramping. In addition, these less dramatic symptoms may have resolved by the time the patient is evaluated. (See "Anaphylaxis: Emergency treatment".)

HISTORY

A thorough history is essential to determine accurately the underlying cause of syncope. Several studies suggest that history and physical examination lead to the diagnosis in approximately half of patients [13,14]. Other studies have assessed specific signs and symptoms to determine which patients are at increased risk for serious outcomes, particularly sudden death [18,26-29].

- **Age** – Young patients are more likely to experience vasovagal syncope. Nevertheless, the emergency clinician must consider the possibility of dysrhythmia, particularly if other concerning factors exist (eg, exertional syncope, family history of sudden death). Electrocardiogram (ECG) findings consistent with dysrhythmia include a short or prolonged QTc or short PR interval.

  Older adult patients appear to be at greater risk for adverse outcomes following syncope [30]. Falls and associated fractures can occur. Aortic stenosis is of particular concern as patients get older and it is the most common obstructive cardiac lesion. Older adult patients are more likely to have autonomic dysfunction, orthostasis, and multiple medications, increasing the risk for syncope [31]. Progression of diabetes can cause

autonomic dysfunction and orthostasis, as can some other conditions such as Parkinson disease [32].

However, several studies suggest that while age correlates with death and other adverse outcomes, age itself is nonspecific, and a history of underlying heart disease is more predictive [33]. As an example, one prospective observational study of 45 consecutive patients 50 years and older with syncope found no significant events at one month among those whose ED evaluation was unremarkable [34]. The use of a number of different age thresholds to define the "at-risk" age makes it difficult to interpret the data. One study used 45 years as a cutoff, another 75 years [18], while consensus opinions have used 60 or 70 years [13,35].

We believe there is little utility in using any absolute age threshold to determine increased risk. Risk of adverse outcomes after syncope gradually increases with age and should be considered in the context of other risk factors, particularly those associated with heart disease.

- **Associated symptoms and triggers** – Concomitant symptoms can provide important diagnostic clues. As an example, chest pain may indicate an acute coronary syndrome or pulmonary embolism. Palpitations suggest an arrhythmia. Dyspnea raises concern for pulmonary embolism or heart failure. Abdominal or low back pain associated with syncope raises the possibility of a rupturing abdominal aortic aneurysm. Headache raises the possibility of subarachnoid hemorrhage. Symptoms such as headache, paresthesias, or weakness may suggest a neurologic cause.

  The emergency clinician should seek a history consistent with vasovagal syncope. If this diagnosis can be made, the patient is low risk. A gradual prodrome generally precedes vasovagal syncope and often includes a sense of warmth, nausea and vomiting, diaphoresis, changes in vision, and pallor, either just prior to or shortly after the event. Inquiring about potential vasovagal triggers is also helpful. Triggers commonly associated with vasovagal syncope include visual stressors (eg, seeing blood during phlebotomy or watching a childbirth), strong physical or emotional stress, micturition, defecation, coughing, swallowing, and prolonged standing in a warm environment.

- **Position** – Patients who lose consciousness with prolonged standing (ie, minimum of 15 to 20 minutes) are more likely to have vasovagal syncope [30]. Patients who lose consciousness while moving from a lying to a standing position are more likely to have orthostasis. Syncope while sitting or supine is suspicious for arrhythmia [30,36].

- **Onset** – Sudden loss of consciousness without warning or prodrome suggests arrhythmia [30]. A prospective observational study of patients with recurrent syncope found that 64 percent of patients sustained an arrhythmia at the time of their sudden loss of consciousness when studied with a loop recorder [37]. Injury from falls associated with an abrupt loss of consciousness can occur. Patients with prodromes are more likely to have vasovagal syncope and have repeatedly been shown to be low risk [10].

- **Duration of symptoms** – The duration of a syncopal event is difficult to quantify. Patients are generally unaware of the duration of their loss of unconsciousness, and events are often unwitnessed or poorly quantified if they are witnessed. As a rough guide, an "event" or loss of consciousness persisting for more than four or five minutes should raise concerns for seizure or other causes of altered mental status.

- **Exertional syncope** – Syncope with exertion raises the possibility of arrhythmia or cardiac outflow obstruction (eg, aortic stenosis, hypertrophic cardiomyopathy, or pericardial tamponade). These patients warrant a thorough cardiac evaluation, including chest radiograph, ECG, and echocardiography.

- **Seizure versus syncope** – Often, clinicians have difficulty determining whether their patient suffered a seizure or syncope. Patients with certain seizure disorders do not manifest generalized convulsions, and patients with syncope may have brief tonic/clonic episodes. Approximately 5 to 15 percent of patients thought to have syncope may have a seizure disorder [38].

  Factors suggestive of seizure include [38-40]:

  - Prodrome (aura) different from that described for vasodepressor syncope
  - Eye deviation, usually superiorly and/or laterally
  - Episode of abrupt onset associated with injury
  - Presence of a tonic phase before the onset of rhythmic clonic activity
  - Head deviation or unusual posturing during the episode
  - Tongue biting (particularly involving the lateral aspect of the tongue)
  - Loss of bladder or bowel control
  - Prolonged post-event (postictal) phase during which the patient is confused and disoriented

  Despite these guidelines, the differentiation of seizures from syncope is sometimes difficult, especially in the patient with a brief seizure where the postictal phase is minimal or the patient with syncope who takes more time than is typical to return to baseline.

However, a postictal phase and confusion without spontaneous return to baseline mentation within minutes is more suggestive of seizure [41].

- **Medications** – A review of the patient's medications may reveal the cause of syncope. This is particularly important with older adult patients. Medications often implicated include calcium channel blockers, beta blockers, alpha blockers, nitrates, antiarrhythmics, diuretics (affecting volume status and electrolyte concentrations), and medications affecting the QTc interval (eg, antipsychotics and antiemetics) (table 2) [23].

- **Prior episodes** – A history of syncopal episodes may be of value. A single episode or multiple episodes over many years suggests a benign etiology. Several episodes over a short period of time in someone with no history of syncope suggest a more significant cause, such as dysrhythmia.

- **Family history** – A family history of unexplained sudden death, dysrhythmia, or early cardiovascular disease (ie, in close relatives less than 50 years old) places patients at increased risk for cardiac syncope [42].

- **Associated injury** – Acute loss of consciousness may result in significant injury or events that predispose to injury. Motor vehicle accidents, hip fractures, and subdural hematomas can result. Emergency clinicians should assess the patient for potential injuries. Although patients with prodromal symptoms have less risk of death and other adverse outcomes following syncope, there is no evidence that they have less risk of acute injury from syncope (eg, from falls). Such patients may ignore warning signs and may be just as likely to incur injury as patients without a prodrome [18].

PHYSICAL EXAMINATION

The physical exam should focus on vital signs and a focused cardiac and neurologic exam, as well as any specific complaints.

- **Vital signs** – Transient hypotension or bradycardia occur during most syncopal events. Abnormal vital signs generally normalize by the time of evaluation in the ED. Persistently abnormal vital signs are concerning and must be investigated. Discrepancies between upper extremities in pulse or blood pressure may reflect aortic dissection or subclavian steal syndrome and should be investigated (see "Clinical features and diagnosis of acute aortic dissection" and "Subclavian steal syndrome", section on 'Physical examination' and "Upper extremity atherosclerotic disease", section on 'Presentations' and "Upper extremity

atherosclerotic disease", section on 'Physical examination'). Low oxygen saturation or tachypnea may be a sign of heart failure or pulmonary embolism.

Orthostatic vital signs should be obtained. The patient should be supine for five minutes before the initial set is obtained. Vital signs are retaken after the patient has been standing for three minutes and compared with initial measurements. The following changes are considered abnormal and may reflect hypovolemia or autonomic dysfunction [20]:

- Drop in systolic blood pressure of 20 mmHg or more.
- Increase in heart rate of 20 beats per minute or more.

  Many asymptomatic patients meet these criteria for orthostasis, but a drop of blood pressure below 90 mmHg associated with symptoms can be diagnostic in itself. Keep in mind that syncope from orthostatic hypotension is a diagnosis of exclusion in the ED, reserved for low-risk patients who have symptoms consistent with the diagnosis. Orthostatic hypotension may be related to serious conditions, including myocardial ischemia or acute blood loss (eg, gastrointestinal bleeding, ruptured abdominal aortic aneurysm, or ectopic pregnancy). (See 'Common conditions' above.)

  There are reports of using wearable technology (eg, Fitbit, Apple watch) to determine whether a change in heart rate correlated with symptoms [43,44]. ED physicians should consider interrogating wearable devices if present on a patient with syncope just as they should interrogate a pacemaker if present.

- **Cardiac examination** – Auscultation of the heart may reveal a rate that is either abnormal or irregular (eg, atrial fibrillation). The clinician should listen for murmurs, specifically for aortic and mitral stenosis. Extra heart sounds, either an S3 or S4, can often be heard in patients with heart failure. Findings on cardiac exam suggesting structural heart disease should be investigated. (See "Auscultation of cardiac murmurs in adults" and "Auscultation of heart sounds".)

  The presence of an implantable pacemaker should be noted as malfunction may lead to syncope.

- **Pulmonary examination** – Auscultation of the lungs may reveal abnormal sounds (eg, crackles, wheezes) consistent with heart failure or other pathology (eg, pulmonary embolus, cardiac ischemia).

- **Neurologic examination** – Patients with syncope by definition return to baseline neurologic function. A thorough exam should be done to identify any subtle focal abnormality suggestive of stroke. (See "The detailed neurologic examination in adults".)

- **Neck examination** – Clinicians should listen for a carotid bruit. Murmurs of aortic stenosis may also radiate to the neck. Examine the neck for elevated jugular venous pressure, a possible sign of heart failure.

  Some groups suggest that carotid massage be performed as part of the evaluation of syncope. We feel this test lacks sufficient sensitivity and specificity to play a meaningful diagnostic role in the ED. We advise caution when considering whether to perform carotid massage in patients with potential carotid artery disease. (See "Reflex syncope in adults and adolescents: Clinical presentation and diagnostic evaluation" and "Vagal maneuvers", section on 'Carotid sinus massage'.)

- **Rectal examination** – A rectal exam with a stool guaiac test can identify some patients with gastrointestinal hemorrhage, which may present with syncope.

- **Intraoral examination** – Lacerations to the lateral aspect of the tongue are suggestive of seizure [39]. (See 'History' above.)

- **Injury assessment and general examination** – The emergency clinician should perform a head to toe exam (ie, secondary survey) looking for evidence of trauma. Common injuries associated with falls following syncope include facial fractures, hips fractures, wrist fractures, and subdural hematomas. A general examination, guided by patient complaints, may reveal important findings such as papilledema or a pulsatile abdominal mass.

DIAGNOSTIC TESTING

Electrocardiogram — Practice guidelines suggest that **all** patients presenting with syncope should receive an electrocardiogram (ECG) [13,45,46]. Although the diagnostic yield of the ECG is low (2 to 7 percent may reveal a significant abnormality), the test is inexpensive, easy to perform, and included in most risk stratification decision tools. An abnormal ECG suggests an underlying cardiac problem, and further investigation is needed.

The clinician should assess the ECG looking for evidence of cardiac arrhythmia or ischemia as the cause of syncope. Concerning ECG findings include [47-49]:

- Non-sinus rhythm
- Left bundle branch block (LBBB)

- Signs of acute myocardial ischemia or infarction

A more comprehensive list of clinical and ECG features associated with syncope from arrhythmia is found in the following table (table 4). Significant ECG findings include prolonged intervals (QRS, QTc), severe bradycardia, preexcitation, low voltage in the standard limb leads, suggesting pericardial effusion, and abnormal conduction syndromes (eg, Wolf-Parkinson-White and Brugada). A short QT interval has also been associated with significant arrhythmias that could result in syncope [50,51]. (See "Syncope in adults: Clinical manifestations and initial diagnostic evaluation", section on 'Electrocardiogram'.)

Of note, there is no consensus about what constitutes significant ECG findings in the setting of syncope. The ECG criteria in some decision tools are complex and lack validation by bedside physicians. Even when apps and calculators are used for complex criteria, most require some interpretation of findings that lack agreement or validation.

Multiple studies have assessed ECG criteria linked to dysrhythmia or ischemia with variable results. In one prospective observational study, patients with an ECG showing sinus rhythm and no new abnormal morphologic changes compared with prior ECGs had substantially lower risk of adverse events during the week following their syncope [18]. According to a subsequent prospective study, high-risk findings associated with adverse cardiac outcomes include any non-sinus rhythm identified from any source (ie, standard 12-lead ECG or cardiac monitor tracings obtained in the ambulance or the ED) and any abnormal conduction of the left bundle (ie, LBBB, left anterior or posterior fascicular block, prolonged QRS duration) [47,52]. A paced rhythm on ECG should prompt the clinician to ask cardiology or the local manufacturer's representative to interrogate the pacemaker to determine the rhythm at the time of the syncopal event.

Cardiac monitoring — While in the ED, patients should be placed on a cardiac monitor. Numerous studies suggest that cardiac monitoring, in addition to the 12-lead ECG, is useful for identifying dysrhythmias while the patient is in the ED [9,53-55]. In one prospective observational study of 95 consecutive syncope patients, major abnormalities identified by Holter monitoring included significant bradycardia (heart rate <30 beats per minute), sinus pauses (particularly those >2 seconds), Mobitz II block, complete heart block, ventricular tachycardia, and frequent premature ventricular contractions (PVCs) [56]. Such findings alert the clinician to an arrhythmogenic cause of syncope. Atrial tachydysrhythmias may cause a syncopal event but usually do not in patients with structurally normal hearts [57].

Laboratory evaluation — Routine laboratory screening in patients with syncope is not supported by evidence and seldom aids management [8,58,59]. Hypoglycemia may rarely

explain an acute syncopal event but should be performed on all patients with altered mental status. Clinicians should obtain other tests based on the clinical circumstances.

Electrolytes may be beneficial in critically ill patients or patients thought to have electrolyte abnormalities from volume loss, diuretic use, or comorbidities such as renal failure. In patients with active bleeding or suspected anemia, a hematocrit should be obtained, and coagulation studies may be useful. A hematocrit less than 30 increases the risk of adverse short-term events in patients with syncope and predicts the need for transfusion [18,60]. A urine pregnancy test should be performed in any female of child-bearing age.

Elevated measurements of natriuretic peptides (ie, brain natriuretic peptide [BNP] or pro-BNP) appear to be predictive of those at risk for adverse outcomes following syncope [61-66]. A systematic review of 11 studies (4246 patients) assessing the predictive value of cardiac biomarkers in adults with syncope concluded that natriuretic peptides and high-sensitive troponin were useful for identifying patients who developed adverse cardiac events following a syncopal episode [67]. (See "Natriuretic peptide measurement in heart failure" and "Troponin testing: Clinical use".)

Some decision tools have incorporated laboratory tests. The FAINT Score includes BNP and troponin, while the Canadian Syncope Risk Score (CSRS) includes troponin [68,69]. Natriuretic peptides and troponin are markers, respectively, for heart failure and ischemic heart disease, both of which are established risk factors for adverse events following syncope. Both of the above scores include a history of heart disease, in particular heart failure and arrhythmia, in addition to these biomarkers, which have become more sensitive and useful for risk stratification.

Neurologic studies — Patients with a history of or physical exam suspicious for a transient ischemic attack, stroke, or new onset seizure need further evaluation. Patients without historical or examination features suggestive of neurologic disease need no further neurologic imaging. Despite the low diagnostic yield of brain imaging, clinicians continue to overuse head computed tomography (CT) and magnetic resonance imaging (MRI) in the evaluation of syncope patients [59,70]. An electroencephalogram may be useful in some cases where it is clinically difficult to distinguish syncope from seizure [71].

Echocardiography — Although not readily available in some EDs, echocardiography is helpful for determining the presence of structural heart disease and is being performed by more emergency physicians at the bedside. Echocardiography can show valvular anomalies, wall motion abnormalities, elevated pulmonary pressure (seen with pulmonary embolism), and pericardial effusions. It is most useful in patients with a known history of cardiac disease or

abnormal ECG findings for further investigation of these at-risk patients. However, routine bedside echocardiography for unexplained syncope in patients without clinical risk factors has no demonstrated benefit [72].

APPROACH TO DIAGNOSIS

The most important tasks for the emergency clinician faced with a syncope patient are to identify and manage life-threatening problems and to differentiate between patients safe for discharge and those who require immediate investigation and in-hospital management [9,45]. In making such determinations, clinicians should consider presyncope (ie, near loss of consciousness) and true syncope a spectrum of the same disease process, with no difference in management [1]. An algorithm outlining ED management (algorithm 1) and tables listing dangerous causes of syncope and high-risk features (table 1 and table 3) are provided. ED clinicians should keep in mind that despite exhaustive testing, a clear diagnosis will ultimately not be found for many patients with syncope. A subset of these patients may have occult cardiac syncope.

When evaluating the syncope patient, emergency clinicians should keep in mind three questions:

- Is this true syncope, or does some other serious condition account for the patient's loss of consciousness (eg, stroke, seizure, head injury)?
- If this is true syncope, is there a clear life-threatening cause?
- If this is true syncope and the cause is not clear, is the patient at high risk?

Life-threatening causes of syncope include hemorrhage (eg, gastrointestinal, subarachnoid), pulmonary embolism, and cardiac syncope from arrhythmia, acute coronary syndrome, or structural heart disease.

Patients with syncope from major hemorrhage usually have a low hematocrit. This may not be the case if bleeding is severe and acute, in which case the hematocrit can be misleading. Gastrointestinal bleeding is the most common cause, and often the rectal exam or a stool guaiac test will be positive for blood. The emergency clinician must consider other sources of hemorrhage, including ruptured aortic aneurysm, ruptured ectopic pregnancy, ruptured ovarian cyst, and ruptured spleen. Appropriate diagnostic testing should be performed. Obtain a pregnancy test in all women of child-bearing age. Bedside ultrasound can be invaluable in determining the presence of abdominal aortic aneurysm, ectopic pregnancy, and intraabdominal blood. (See "Approach to acute lower gastrointestinal bleeding in adults" and

"Approach to acute upper gastrointestinal bleeding in adults" and "Ectopic pregnancy: Clinical manifestations and diagnosis" and "Ultrasonography of pregnancy of unknown location" and "Evaluation and management of ruptured ovarian cyst".)

Pulmonary embolism (PE) is an uncommon but potentially dangerous cause of syncope. Patients with PE often present with dyspnea and chest pain and may be hypoxic. An electrocardiogram (ECG) with evidence of right heart strain is suggestive. Most often, the diagnosis is made by computed tomography (CT) scan. When suspecting PE, clinicians should perform a work-up based on the patient's baseline risk for PE. Evaluation and management of PE are discussed separately. Patients whose presenting symptom of PE is syncope are not at increased risk of death compared with patients whose syncope is not caused by PE [73,74]. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism" and "Epidemiology and pathogenesis of acute pulmonary embolism in adults".)

Although the incidence of PE was purported to be as high as 17 percent among patients hospitalized with syncope (n = 230) in a controversial study published in 2016 by the PESIT investigators [75], subsequent studies have refuted this finding [76-78]. According to a retrospective review of five databases from four countries involving over 1.6 million adults who presented to the ED for syncope, the prevalence of PE ranged from 0.5 to 2.1 percent among patients who were hospitalized, and from 0.06 to 0.55 percent for all patients [76].

Syncope associated with a significant headache suggests possible subarachnoid hemorrhage, and evaluation with head CT and lumbar puncture may be necessary. Should historical features or examination findings suggest transient ischemic attack or stroke, clinicians should obtain a CT with angiography or magnetic resonance imaging (MRI) and neurologic consultation. Suspected seizure should be evaluated with neuroimaging (head CT or MRI) and electroencephalogram (EEG), as an in-patient or out-patient. A less extensive evaluation may be appropriate for patients with an established seizure diagnosis and no concerning features in their presentation. (See "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis" and "Initial assessment and management of acute stroke".)

Diagnosing cardiac syncope is especially important because the one-year mortality of such patients approaches 30 percent, significantly higher than noncardiac syncope or syncope of unknown etiology [6]. The mortality of syncope patients with heart failure is even higher [19]. We suggest clinicians obtain an ECG in all syncope patients. The emergency clinician should study the ECG for evidence of ischemia, arrhythmia, and conduction or electrolyte abnormalities. An abnormal ECG or concerning history should prompt further workup, including echocardiography and cardiac monitoring as indicated. (See 'Electrocardiogram'

above and "Syncope in adults: Clinical manifestations and initial diagnostic evaluation", section on 'Electrocardiogram'.)

Arrhythmia is the most common serious cause of cardiac syncope but may not manifest during the course of an ED evaluation. Syncope patients should be placed on a cardiac monitor while evaluated in the ED. Myocardial infarction (MI) is a rare but important cause of syncope. Most such patients have atypical presentations without ST segment elevations on initial ECGs. MI occurs in about 3 percent of patients with syncope, and the negative predictive value of a "normal" ECG is greater than 99 percent [79].

Several historical factors raise concern for cardiac syncope, including a strong family history (eg, close relative with sudden death or MI before 50 years old), a history of heart disease (eg, coronary artery disease, heart failure, MI, valvular disease, arrhythmia), and symptoms consistent with heart disease (eg, chest pain, palpitations, shortness of breath).

Examination findings suggestive of cardiac syncope include abnormal vital signs, including orthostatic changes and discrepant pulses or blood pressure, and abnormal heart sounds. If the history or physical examination suggests structural heart disease, an echocardiogram should be obtained. Review the patient's medication list for drugs that may affect the heart rate or rhythm or blood pressure. The elderly and patients taking multiple medications are at greater risk of syncope from medication effects [23]. (See 'Physical examination' above and 'History' above.)

Patients diagnosed with vasovagal (vasovagal) syncope are at very low risk of adverse outcomes. Historical factors, including a prodrome (eg, sense of warmth, dizziness, pallor, diaphoresis, abdominal pain, changes in vision, or nausea), may suggest the diagnosis. Clinicians may be able to identify potential triggers, such as micturition, defecation, cough, prolonged standing, or a stressful event (eg, blood draw). A history suggestive of vasovagal syncope coupled with an unremarkable physical examination and a normal ECG constitute a reassuring presentation, with little risk of life-threatening complications.

Note that determining the cause of syncope in patients presenting to the ED can be difficult [60]. Using a careful history, physical examination, and an ECG, along with selective ancillary testing based on clinical findings, clinicians can identify the cause in approximately 50 percent of patients in the ED [13,14]. Even with intensive inpatient and outpatient testing (eg, loop recorders, Holter monitors, long-term continuous rhythm monitors [80], tilt table testing, and electrophysiology studies), clinicians remain unable to determine the cause of syncope in many patients.

In addition, studies of long-term monitoring suggest that a brief period of in-hospital cardiac monitoring is likely insufficient to diagnose patients with arrhythmias causing syncope. Even 24

to 72 hours of assessment with a Holter monitor, while it diagnoses more arrhythmias, misses important arrhythmias that can only be detected with long-term monitors. Newer, portable, longer-wear cardiac monitors are available and can easily be applied in the ED. The results from prospective observational studies suggest that the time needed to detect an arrhythmia is often longer than that provided by a standard 48-hour Holter monitor and that these newer monitors may improve diagnostic accuracy [81,82].

RISK STRATIFICATION

Risk stratification tools should be used to assist clinical judgment but cannot replace it. Emergency clinicians will be unable to determine an exact cause in a significant number of patients who present to the ED with syncope. In such instances, using risk stratification to guide management and disposition represents a practical approach. A treatment algorithm is provided (algorithm 1).

Using the risk factors identified in the studies described below can help clinicians determine patient risk and appropriate disposition (table 3). While individual studies may be limited by such factors as the size of the cohort, the number of adverse events, and the definition of an event, a consistent theme emerges: patients with an abnormal ECG on presentation or a history of heart disease, particularly structural heart disease (eg, heart failure), are at greater risk for adverse outcomes.

When applying risk stratification tools, clinicians must be careful to include only appropriate patients. Patients with significant underlying pathology and patients who do not reflect the population in which the risk stratification tool was studied are not appropriate subjects. As examples, a syncope patient with associated acute coronary syndrome may not meet any high-risk criteria but obviously requires admission, while patients with loss of consciousness from head trauma or illicit drug use cannot be assessed using a decision rule if such patients were excluded from the rule's derivation and validation studies.

Over the past couple of decades, several research groups have identified factors associated with increased risk for short-term and one-year morbidity and mortality following a syncopal episode. According to systematic reviews, each approach has limitations, and no one decision rule should be used in isolation to determine patient risk or disposition [83,84]. That said, the groups have identified several common risk factors of importance, and these should be incorporated into the clinician's assessment [9,83-86]. The findings of major research groups are described here and have been incorporated into guidelines as outlined below:

- One research group performed derivation and validation studies on cohorts of consecutive ED patients with syncope to identify predictors of arrhythmia and death at one year [27]. Significant risk factors were a history of arrhythmia, an abnormal electrocardiogram (ECG), a history of decompensated (ie, congestive) heart failure, and age over 45 years.

- Another research group assessed adverse outcomes at 7 and 30 days in their derivation and validation of The San Francisco Syncope Rule (SFSR) [18,87,88]. Significant predictors of adverse events (primarily arrhythmia) included a history of acute decompensated (ie, congestive) heart failure, abnormal ECG (non-sinus rhythm or new changes), hematocrit less than 30, shortness of breath, and systolic blood pressure of less than 90 mmHg at triage.

  A systematic review of external validation studies of the SFSR found that it is the most thoroughly investigated prediction rule for the assessment of syncope and that validation studies have reported inconsistent results [89]. Such inconsistencies may be due to the use of different definitions for arrhythmia, different definitions for an abnormal ECG, and differences in the clinicians interpreting the tracing [89].

- The Osservatorio Epidemiologico sulla Sincope nel Lazio (OESIL) study group developed a risk score based on predictors of death at one year, which they found to be an abnormal ECG, a history of cardiovascular disease (including heart failure), age over 65, and syncope without prodrome [28].

- Researchers in Switzerland developed and validated a prediction score for subsequent arrhythmia in patients with unexplained syncope following a standard ED evaluation [29]. They found the significant variables to be an abnormal ECG, a history of heart failure, and age above 65 years. The prevalence of arrhythmia was nearly identical in each cohort, though each phase of the study was performed in a different country (Switzerland and the United States).

- The Short-Term Prognosis of Syncope (STePS) trial identified an abnormal ECG, a history of cardiac disease, absence of a prodrome, and concomitant trauma as indicators of high risk in the short term (within 10 days of presentation) [90].

- The researchers who created the Canadian Syncope Risk Score (CSRS) assessed adverse short-term outcomes (within 30 days) and identified the following high-risk features: abnormal ECG, history of cardiac disease, lack of vasovagal etiology, elevated troponin, and systolic blood pressure <90 mmHg or >180 mmHg [68,91]. Using nine variables, patients were stratified **after** an initial ED evaluation where no obvious cause for syncope

was identified into risk categories ranging from very low to high. The CSRS has been validated in 3819 patients in several sites across Canada.

The CSRS was also validated in an international, multicenter cohort that included 2283 patients 40 years or older who presented to an ED with syncope [92]. In patients classified as low risk (1388 patients) by the CSRS, only 1 percent had a serious clinical event or required a procedure at 30 days. A multivariate regression analysis of the nine individual items of the CSRS found that ED clinician gestalt for the classification of syncope (eg, cardiac syncope, vasovagal syncope, or other) had the largest odds ratio for predicting the primary composite outcome, and a simplified model including only the clinician classification achieved similar test characteristics compared with the entire CSRS.

- The FAINT Score is intended to determine risk in older adults (≥ 60 years) with unexplained syncope [69]. The risk factors assessed are history of heart failure, history of cardiac arrhythmia, initial abnormal ECG result, elevated pro B-type natriuretic peptide (BNP), and elevated high-sensitivity troponin T. The score awaits external validation.

The American College of Emergency Physicians (ACEP) has published evidence-based guidelines on the management and disposition of patients with syncope, but these have not been updated since 2007. This policy statement is based primarily on the risk stratification data presented in earlier studies and suggests admission for patients with evidence of acute decompensated (ie, congestive) heart failure or structural heart disease and patients at high risk for adverse outcomes (table 3) [45]. The American College of Physicians, European Society of Cardiology, American College of Cardiology, and American Heart Association have also established guidelines and algorithms for management and disposition [13,46]. A group of international experts created guidelines for the ED management of syncope patients that generally reinforces these guidelines and algorithm [9]. We suggest that emergency clinicians use the ACEP guidelines and the attached ED syncope algorithm, which are better suited to the conditions of EM practice (algorithm 1).

In a study from 2004 to assess their decision-making, emergency clinicians demonstrated excellent judgment regarding patient risk but often did not determine disposition based on their judgment, choosing to admit close to 30 percent of patients who they felt had a less than 2 percent chance of a serious adverse outcome [60]. The researchers concluded that using clinical judgment augmented by risk stratification may reduce hospital admissions and save some of the estimated two billion dollars spent on syncope each year in the United States. Incorporating risk factors into a clinical pathway that placed low-risk patients with syncope in an ED observation unit reduced hospital admissions and patients who returned to the ED without a change in 30-day outcomes [93]. Since 2004, the advent of risk stratification research, electronic

health records with clinical decision support and online calculators have improved the management of patients with syncope. A large cohort study found ED admission rates of low-risk syncope patients decreased from 28 to 14 percent over a 10-year period from 2008 to 2017, correlating with this period of research and dissemination [94].

PATIENT DISPOSITION

Patients with obvious cardiac or neurologic causes of syncope, as well as those with concerning symptoms or signs, should be investigated thoroughly in the ED and likely admitted for further in-hospital testing or treatment. To some degree, clinician judgment determines which symptoms or signs are "concerning," but such findings may include:

- Syncope accompanied by chest pain or shortness of breath
- Exertional syncope
- Abnormal vital signs
- Abnormal findings on cardiac, pulmonary, or neurologic examination

For well-appearing asymptomatic patients with an unclear cause, clinicians should determine the risk for adverse outcome. Clinicians determine risk on the basis of patient history, examination and electrocardiogram (ECG) findings, and evidence-based guidelines. The presence of any of the following features determines high risk (table 3). (See 'Risk stratification' above.)

- Abnormal ECG
- History of structural heart disease or clinical findings suggestive of heart failure
- Persistently low blood pressure (systolic <90 mmHg)
- Shortness of breath with event or during evaluation
- Elevated troponin or BNP (if obtained)
- Hematocrit <30 (if obtained)
- Older age and associated comorbidities
- Family history of sudden cardiac death

High-risk patients should be investigated in the ED and likely admitted for inpatient monitoring and with a prolonged monitoring device if no arrhythmia is found during admission [95]. A significant portion of these patients will have arrhythmias detected within six hours of ED arrival. Patients with significant arrhythmias should be admitted to cardiology for pacemaker or automatic implantable defibrillators as indicated. Low-risk asymptomatic patients may be discharged, provided out-patient follow-up can be arranged and the clinician has no other

concerns. Patients with obvious vasovagal syncope who have returned to baseline and are asymptomatic without injury should be discharged. Outpatient evaluation of syncope, including the use of ambulatory monitors, tilt table testing, and other diagnostic techniques, is discussed in detail separately. (See "Syncope in adults: Clinical manifestations and initial diagnostic evaluation" and "Syncope in adults: Epidemiology, pathogenesis, and etiologies" and "Upright tilt table testing in the evaluation of syncope".)

Patients without any feature associated with high risk for an adverse event but with some concerning clinical finding are generally deemed to be at intermediate risk. The disposition of intermediate-risk patients will vary depending upon local health care practice and resources, including the availability of consultants, in-hospital or observation unit beds, and timely outpatient follow-up, including the ability to initiate ambulatory monitoring from the ED. The clinician and patient should discuss the clinical findings and possible approaches, and the patient's goals and preferences should be incorporated into the determination of disposition for those deemed to be at intermediate risk. Of note, most arrhythmias can be detected with prolonged ambulatory monitoring of up to 15 days with the incidence being greatest during the first 24 to 48 hours after the event [81,95-98]. There are many potential long ambulatory monitoring devices with various features, and their use to diagnose arrhythmia in syncope is recommended in the new American Heart Association (AHA) guidelines. The evaluation of intermediate risk patients is reviewed in greater detail separately. (See "Syncope in adults: Risk assessment and additional diagnostic evaluation", section on 'Assessment based on the initial evaluation'.)

In some EDs, patients identified as intermediate risk are managed in a syncope observation unit [99-101]. In one observational study, 103 consecutive patients presenting to an ED with syncope were randomly assigned to standard care or care in a syncope observation unit [102]. Patients in the observation unit required fewer admissions, and no significant differences in mortality or recurrent syncope were detected [100]. Further studies are needed to determine if such units provide a cost benefit without missing significant outcomes. There continues to be tremendous variability in admission patterns for patients with syncope and controversy surrounding who benefits from admission. As more sophisticated ambulatory monitoring is developed, the need for admission of intermediate-risk patients will continue to evolve [9,103-106]. Pathways based on risk, use of observation units, and outpatient follow-up have improved management [93].

PREVENTION

One prospective randomized multicenter study found that instructing patients diagnosed with vasovagal syncope in the use of physical counterpressure maneuvers (PCM) decreased the

incidence of recurrent episodes of syncope from 51 percent to 32 percent compared with conventional therapy [107]. Conventional therapy consisted of explaining the mechanism underlying fainting and providing advice about lifestyle modifications (eg, avoiding known triggers, increasing fluid and salt intake, lying down when symptoms occur). PCM consisted of one or more of the following: (1) leg crossing combined with tensing of the muscles of the legs, abdomen, and buttocks; (2) arm tensing by grasping one hand with the other and forcibly abducting the arms; or (3) squeezing an object clasped in the dominant hand.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Syncope".)

SUMMARY AND RECOMMENDATIONS

- **Overview** – When evaluating the patient with syncope, the primary responsibility of the emergency clinician is to assess whether a life-threatening cause is present and to provide appropriate management and disposition. An algorithm outlining emergency department (ED) management (algorithm 1) and a table listing dangerous causes of syncope (table 1) are provided. (See 'Approach to diagnosis' above.)

  When evaluating the patient with syncope, try to answer the following questions:

  - Is this true syncope, or does some other serious condition account for the patient's loss of consciousness (eg, stroke, seizure, head injury)?
  - If this is true syncope, is there a clear life-threatening cause?
  - If this is true syncope and the cause is not clear, is the patient at high risk?

- **Differential diagnosis**

  - **Life-threatening conditions** – The most important causes to consider are cardiac syncope, blood loss, pulmonary embolism, and subarachnoid hemorrhage. (See 'Life-threatening conditions' above.)

  - **Common conditions** – Common but less dangerous causes of syncope include vasovagal, carotid sinus sensitivity, orthostasis, and medication-related (table 2). (See 'Common conditions' above.)

- **Other conditions** – Seizure, stroke, and head injury do not meet the technical definition of syncope but should be considered during the initial assessment. (See 'Other conditions' above.)

- **History** – Historical features that reflect increased risk of a dangerous cause of syncope include concomitant symptoms (eg, shortness of breath, headache, chest pain), sudden loss of consciousness without prodrome, exertional syncope, older age, and family history of sudden death. Review which medications the patient is taking since medication reactions account for a significant percentage of syncopal episodes. (See 'History' above.)

  Syncope and seizure can be difficult to differentiate. Certain seizure disorders do not manifest generalized convulsions, and syncope can cause brief tonic/clonic episodes. A postictal phase and confusion without spontaneous return to baseline mentation within minutes is more suggestive of seizure. (See 'History' above.)

- **Physical examination** – Focus examination on vital signs and the neurologic and cardiac examinations. Patients with syncope often fall and sustain secondary injuries, which may include the head, wrist, or hip, thus warranting a symptom-guided examination looking for injuries. (See 'Physical examination' above.)

- **Diagnostic testing** – All patients presenting with syncope should receive an electrocardiogram (ECG). While in the ED, patients should be placed on a cardiac monitor. Routine non-targeted laboratory testing rarely aids management. Patients with history or examination features suggestive of neurologic disease should have central nervous system imaging. Although not readily available in some EDs, echocardiography is helpful for determining the presence of structural heart disease. (See 'Diagnostic testing' above.)

- **ECG** – Obtain an ECG on all patients with syncope. Significant findings include prolonged intervals (QRS, QTc), severe bradycardia, pre-excitation, and evidence of myocardial infarction (table 4). Other notable findings include low voltage in the standard limb leads, suggesting pericardial effusion; and abnormal conduction syndromes (eg, Wolf-Parkinson-White, Brugada, and short QT syndromes). (See 'Electrocardiogram' above.)

- **Obvious cardiac or neurologic causes of syncope** – Patients with obvious cardiac or neurologic causes of syncope, as well as those with concerning symptoms or signs (table 3), should be investigated thoroughly in the ED and likely admitted to inpatient or observation units. Patients with obvious vasovagal syncope who have returned to baseline and are asymptomatic without injury can be discharged. (See 'Patient disposition' above.)

- **Asymptomatic patients with an unclear cause** – Emergency clinicians will be unable to determine an exact cause in a significant number of patients with syncope. In such instances, risk stratification tools help guide management and disposition. Clinicians determine risk on the basis of patient history, physical examination, ECG findings, laboratory studies if indicated (eg, brain natriuretic peptide [BNP], troponin), evidence-based guidelines, and validated decision tools. A composite table of high-risk features based on findings from several studies is provided (table 3). (See 'Risk stratification' above.)

    - High-risk patients should be investigated in the ED and likely admitted. Highest-risk features include a history of congestive heart failure and an abnormal ECG.

    - The disposition of intermediate-risk patients will vary depending upon local health care practice and resources, including the availability of consultants and timely outpatient follow-up, the ability to initiate ambulatory monitoring from the ED, and risk tolerance. Patient goals and preferences should be incorporated into the determination of disposition. (See 'Patient disposition' above.)

    - Low-risk asymptomatic patients may be discharged with appropriate outpatient follow-up.

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Topic 293 Version 39.0

GRAPHICS

Major life threatening causes of syncope

Cardiovascular syncope
Arrhythmia
Ventricular tachycardia
Long QT syndrome
Brugada syndrome
Bradycardia: Mobitz type II or 3rd degree heart block
Significant sinus pause >3 seconds
Ischemia
Acute coronary syndrome, myocardial infarction
Structural Abnormalities
Valvular heart disease: aortic stenosis, mitral stenosis
Cardiomyopathy (ischemic, dilated, hypertrophic)
Atrial myxoma
Cardiac tamponade
Aortic dissection
Significant hemorrhage
Trauma with significant blood loss
Gastrointestinal bleeding
Tissue rupture: aortic aneurysm, spleen, ovarian cyst, ectopic pregnancy, retroperitoneal hemorrhage
Pulmonary embolism
Saddle embolus resulting in outflow tract obstruction or severe hypoxia
Subarachnoid hemorrhage

Graphic 81820 Version 1.0

Common causes of syncope

Neurocardiogenic syncope
Micturition
Defecation
Cough mediated
Deglutition
Glossopharyngeal nerve
Situational
Carotid sinus hypersensitivity
Head turning
Circumferential neck compression (neck tie)
Shaving
Orthostatic syncope
Volume loss
Autonomic dysfunction
Deconditioning, prolonged bed rest
Medication related syncope
Vaso active medications
Alpha and beta blockers, calcium channel blockers, nitrates, antihypertensive medications, diuretics, erectile dysfunction medications
Medications affecting conduction
Antiarrhythmics, calcium channel and beta blockers, digoxin,
Medications affecting the QT interval
Antiarrhythmics, antiemetics, antipsychotics/depressants
Diuretics

Graphic 62074 Version 2.0

High- and low-risk factors in syncope patients

Low-risk factors	High-risk factors
Characteristics of the patients	
Young age (<40 years)	
Characteristics of syncope	
Only while standing	During exertion
Standing from supine/sitting position	In supine position
Nausea/vomiting before syncope	New onset chest discomfort
Feeling of warmth before syncope	Palpitations before syncope
Triggered by painful/emotionally distressing stimulus	Associated with dyspnea
Triggered by cough/defecation/micturition	
Factors present in the history of the patient	
Prolonged history (years) of syncope with same characteristics as current episode	Family history of sudden death
	Decompensated (congestive) heart failure
	Aortic stenosis
	Left ventricular outflow tract disease
	Dilated cardiomyopathy
	Hypertrophic cardiomyopathy
	Arrhythmogenic right ventricular cardiomyopathy
	Left ventricular ejection fraction <35%
	Documented ventricular arrhythmia
	Coronary artery disease/Myocardial infarction
	Congenital heart disease
	Pulmonary hypertension
	ICD implantation
Symptoms, signs, or variables associated with the syncopal episode	
	Anemia (Hb <9 g/dL)
	Lowest systolic blood pressure in the emergency department <90 mmHg

	Sinus bradycardia (<40 bpm)
ECG features*	
	New (or previously unknown) left bundle branch block
	Bifascicular block + first degree AV block
	Brugada ECG pattern
	ECG changes consistent with acute ischemia
	Non-sinus rhythm (new)
	Bifascicular block
	Prolonged QTc (>450 ms)

According to characteristics of the patient and the syncopal episode, the subject can be defined as low, high or indeterminate risk. Low risk: patients with one or more low-risk characteristics and without any high-risk characteristics. High risk: patients with at least one high-risk characteristic. Intermediate or indeterminate risk: patients without any high- or low-risk characteristics, or patients with only low-risk factors and some co-morbidities such as chronic renal failure, respiratory failure, hepatic failure, neoplasm, cerebrovascular disease or previous history of heart disease. Note that finding any of these abnormalities does not always lead to a definite diagnosis.

ICD: implantable cardioverter defibrillator; AV: atrioventricular; bpm: beats per minute; ECG: electrocardiogram.

* Note that not all the ECG patterns are covered by the table, and some other ECG patterns could be considered in stratifying patient risk such as short QT syndrome, early repolarization, ECG findings indicating hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and incidental finding of Q wave.

Graphic 108983 Version 20.0

Clinical and electrocardiographic (ECG) features of patients with syncope at high risk of an arrhythmic cause

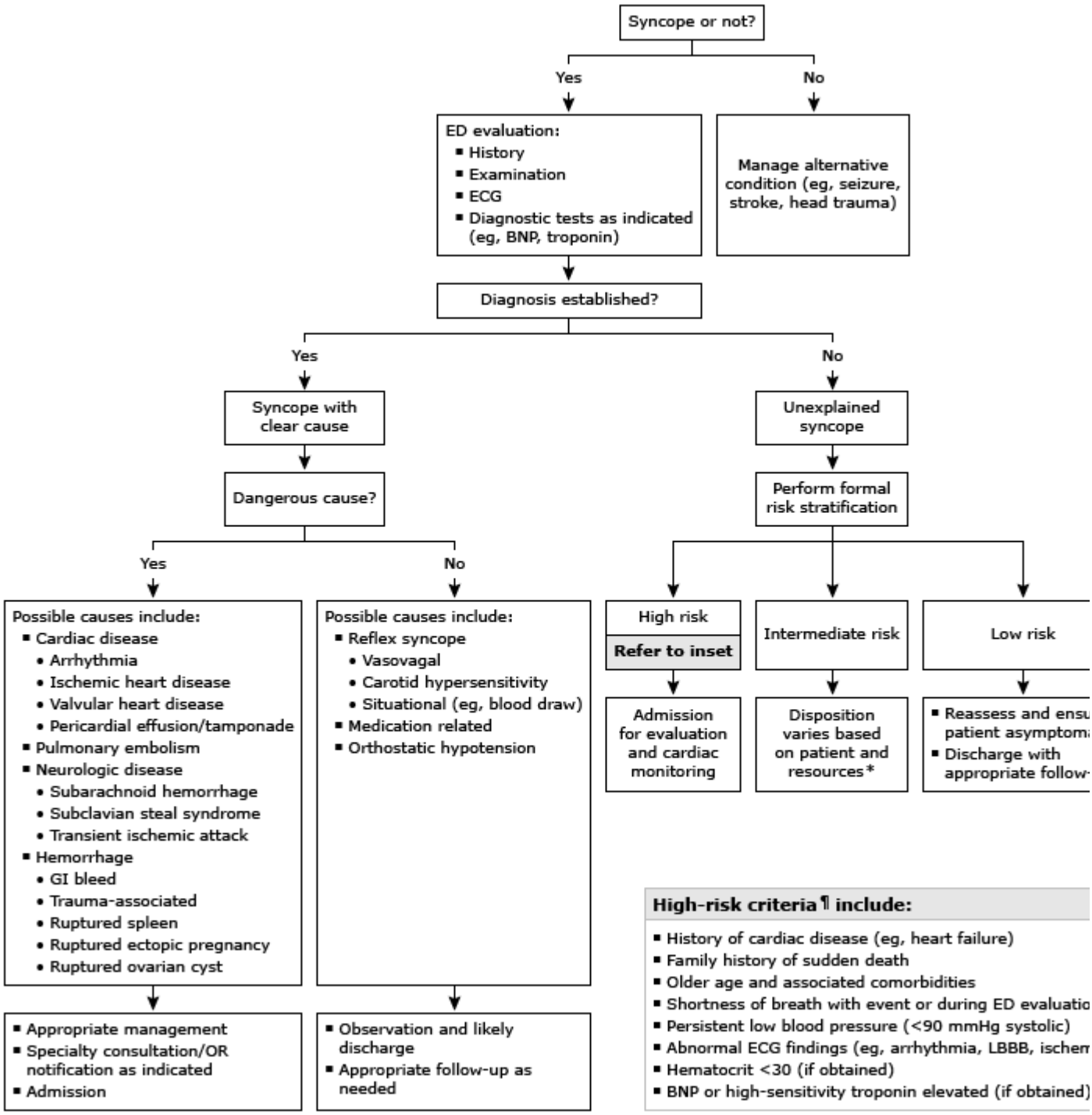
▪ Significant structural heart disease or CAD (including reduced LVEF, heart failure, CAD with prior MI, severe aortic or mitral stenosis, hypertrophic cardiomyopathy)
▪ Persistent sinus bradycardia <40 beats per minute or sinus pauses >3 seconds in an awake patient
▪ Third-degree (complete) AV block
▪ Mobitz II second-degree AV block
▪ Preexcited QRS complexes, suggesting Wolff-Parkinson-White syndrome
▪ Alternating left and right bundle branch block
▪ VT or paroxysmal supraventricular tachycardia with rapid ventricular rate
▪ Nonsustained polymorphic VT with long or short QT interval
▪ Long or short QT intervals
▪ Right bundle branch block pattern with ST elevation in leads V1 to V3 (Brugada syndrome)
▪ Negative T waves in right precordial leads and epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy
▪ Pacemaker or implantable cardioverter-defibrillator malfunction with cardiac pauses

CAD: coronary artery disease; LVEF: left ventricular ejection fraction; MI: myocardial infarction; AV: atrioventricular; VT: ventricular tachycardia.

Adapted from: Brignole M, Moya A, de Lange FJ, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J 2018; 39:1883.

Graphic 118883 Version 3.0

Emergency department approach to an adult patient with syncope



ED: emergency department; ECG: electrocardiogram; BNP: brain natriuretic peptide; GI: gastrointestinal; OR: operating room; LBBB: left bundle branch block.

* The disposition of patients at intermediate risk varies depending on local practice and resources, including availability of consultants and hospital or observation unit beds, and the availability of timely out-patient follow-up with ambulatory cardiac monitoring.

¶ For details about high-risk criteria, including tables summarizing high-risk features of the history, examination, and ECG, refer to the UpToDate topic covering assessment of syncope in the ED.

Δ BNP and high-sensitivity troponin testing is most useful in older adults and patients with heart disease. It is not needed in all patients.

Graphic 69774 Version 3.0