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Syncope: Risk Stratification And Clinical Decision Making

Abstract

Syncope is a common occurrence in the emergency department, accounting for approximately 1% to 3% of presentations. Syncope is best defined as a brief loss of consciousness and postural tone followed by spontaneous and complete recovery. The spectrum of etiologies ranges from benign to life threatening, and a structured approach to evaluating these patients is key to providing care that is thorough, yet cost-effective. This issue reviews the most relevant evidence for managing and risk stratifying the syncope patient, beginning with a focused history, physical examination, electrocardiogram, and tailored diagnostic testing. Several risk stratification decision rules are compared for performance in various scenarios, including how age and associated comorbidities may predict short-term and long-term adverse events. An algorithm for structured, evidence-based care of the syncope patient is included to ensure that patients requiring hospitalization are managed appropriately and those with benign causes are discharged safely.

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Authors

Suzanne Y. G. Peeters, MD
Emergency Medicine Residency Director, Haga Teaching Hospital, The Hague, The Netherlands

Amber E. Hoek, MD
Attending Emergency Physician, Erasmus Medical Centre, Department of Emergency Medicine, Rotterdam, The Netherlands

Susan M. Mollink, MD
Emergency Medicine Resident, Haga Teaching Hospital, The Hague, The Netherlands

J. Stephen Huff, MD
Professor of Emergency Medicine and Neurology, University of Virginia, Charlottesville, VA

Peer Reviewers

Andy Jagoda, MD, FACEP
Professor and Chair, Department of Emergency Medicine, Icahn School of Medicine at Mount Sinai, Medical Director, Mount Sinai Hospital, New York, NY

Scott Silvers, MD, FACEP
Chair, Department of Emergency Medicine, Mayo Clinic, Jacksonville, FL

Prior to beginning this activity, see "Physician CME Information" on the back page.

Case Presentations

It is a busy day in your ED when 3 patients arrive within minutes of each other. A 51-year-old woman arrives by EMS. She felt faint while riding her racing bicycle and got off just before losing consciousness. EMS found her conscious, but pale, with heart rate, 50 beats/min; blood pressure, 90/50 mm Hg; respiratory rate, 25 breaths/min; and oxygen saturation, 98% on room air. EMS provided 1 liter of normal saline without a change in her vital signs. In the ED, her BP is still 90/60 mm Hg. She tells you that just before she got off her bike, she experienced pain in her throat, but she denies chest pain, shortness of breath, or headache. She appears uncomfortable and complains of persisting throat pain and states she is afraid of dying. Her initial ECG shows a sinus bradycardia but is otherwise normal. Her past medical history is not significant. She takes no medications. She is an experienced marathon runner and has never had similar complaints. You wonder what could have caused the syncope and persistent bradycardia.

A short time later, a 19-year-old woman presents to the ED after fainting in the park while attending a party. She tells you she suddenly felt light-headed, warm, and sweaty, and then passed out. According to her friends, she had a brief period of her arms jerking. When she came to, she felt very tired. Her vital signs are: respiratory rate, 18 breaths/min; oxygen saturation, 99% on room air; heart rate, 85 beats/min; blood pressure, 110/70 mm Hg; and temperature, 36.6°C. There is no evidence of tongue biting, and her neurologic examination is normal. Though she says she does not believe she is pregnant, you perform an hCG test, which is negative. You wonder about the significance of her arm jerking and whether she might have had a seizure.

In the next room is a 77-year-old man brought in by his daughter-in-law. He had a brief loss of consciousness, without sustaining an injury, and is now fully recovered, feels fine, and states he wants to leave. His daughter-in-law, however, does not want to take him home "like this." His vital signs are: respiratory rate, 16 breaths/min; oxygen saturation, 96% on room air; heart rate, 75 beats/min; blood pressure, 150/90 mm Hg; and temperature 37.2°C. His ECG shows a left bundle branch block that is unchanged compared with his old ECG. His past medical history is significant for an acute myocardial infarction, a CABG, hypertension, and diabetes. His medications include a diuretic, aspirin, metoprolol, an ACE inhibitor, and metformin. His bedside glucose is within normal limits.

He looks so well that you are tempted to follow his wishes and send him home, but something just doesn't seem right…

Introduction

Syncope is a temporary loss of consciousness and posture, often described as "fainting" or "passing out." Near-syncope is defined as a patient almost losing consciousness, and it is approached in the same way as syncope. A 2012 prospective cohort study comparing 244 patients with near-syncope and 293 with syncope showed that patients with near-syncope are as likely as those with syncope to experience critical interventions or adverse events. However, patients with near-syncope were less likely to be hospitalized, 49% versus 69% respectively, which may be a potential risk-management issue.[1]

Syncope accounts for 1% to 3% of all emergency department (ED) visits.[2-7] The incidence of syncope in the ED increases with age, with a sharp rise in patients older than 70 years.[8,9] The overall incidence of syncope is 2.6 per 1000 person-years, with an incidence of 1.6 per 1000 person-years for the first episode.[8] Syncope is reported as the primary presenting complaint in 75% of syncope patients seen in the ED, and, in 45%, it was the only complaint.[6]

Patients presenting to the ED likely represent a different population from those seen in other clinical settings, with a higher pretest probability for significant underlying etiology.[10,11] In the Framingham study, the incidence for the first syncope in the general population was 6.2 per 1000 person-years, with only 56% of patients reporting having consulted a physician for evaluation.[9]

Syncope is a symptom with a wide range of possible underlying causes. The most effective diagnostic tools in evaluating a patient with syncope are history, physical examination, and electrocardiogram (ECG).[8,12-15] Multiple studies in Europe and North America have shown that unstructured evaluations for syncope result in high costs and low diagnostic yield when compared to evaluations that follow a standardized protocol.[2-4,7,13,16-23] The use of algorithms, guided by clinical findings, resulted in a reduction of undiagnosed cases from 50%-70% down to 17%-25%.[4,7,8-12-14,17,21,24-39]

This issue of *Emergency Medicine Practice* presents the best available evidence for the diagnostic strategy and risk stratification of patients with syncope presenting to the ED and provides guidance for differentiating patients who can be safely discharged from those who are at risk for an adverse outcome and need to be hospitalized.

Critical Appraisal Of The Literature

A literature search from 1945 through January 2014 was performed using Ovid MEDLINE®, Embase, and the Cochrane Database of Systematic Reviews. Search terms included *syncope, transient loss of consciousness, collapse, risk stratification, emergency department,* and synonyms. The National Guideline Clearinghouse (www.guideline.gov) was searched with equivalent search terms for syncope management guidelines on risk stratification in the ED published in the last decade.

Clinical guidelines regarding the evaluation and

diagnosis of syncope have been published by many organizations, including the American College of Emergency Physicians (ACEP), the European Society of Cardiology (ESC), the National Institute for Health and Care Excellence (NICE), and the Canadian Cardiovascular Society (CCS). **(See Table 1.)**

There were 1310 English language articles retrieved, selected, and graded using standardized grading forms by 2 independent reviewers. Inclusion criteria were risk stratification, management of syncope in the ED, risk factors of syncope, and articles most relevant to emergency medicine. Studies of populations hospitalized for syncope were included to draw a complete image of the etiology, diagnostic strategies, and outcomes. Case reports, letters, editorials, and nonsystematic reviews (expert opinion) were excluded. Systematic review and guideline references were checked for relevant articles missing in the search. A total of 172 articles were used as best available evidence for this issue.

Syncope and related conditions proved to be infrequently and inconsistently defined in the current medical literature.[44] Some study populations included patients with seizures and hypoglycemia. The terms *vasovagal, neurocardiogenic, neurogenic,* and *reflex syncope* are inexactly defined in different papers but are generally synonymous. This article will use the term *neurally mediated syncope.*

The syncope literature consists mainly of prospective and retrospective cohort studies, case reports, nonsystematic reviews, and expert opinion. Most studies have small sample sizes and are thus assigned a low level of evidence.

Etiology And Epidemiology

The etiology of syncope is divided into 3 major categories, listed here in decreasing incidence. **(See Table 2, page 4).**

- Neurally mediated syncope
- Orthostatic hypotension-mediated syncope
- Cardiovascular-mediated syncope

In a prospective cohort study evaluating patients presenting with transient loss of consciousness admitted to the hospital, neurally mediated syncope was the most frequent diagnosis (60.2%) in patients of all ages.[45] A meta-analysis of 43,315 patients with syncope presenting to the ED reported that neurally mediated syncope and orthostatic hypotension accounted for 29% of the cases (95% confidence interval [CI], 12-47); 33% were discharged without a diagnosis. The hospital admission rate was 42% (95% CI, 32-52). There was a 4.4% mortality rate at 1 month (CI 95%, 3.1-5.1), 1.1% from a cardiovascular etiology (95% CI, 0.7-1.5). Cardiovascular-related syncope accounted for 10.4% of the cases (95% CI, 7.8-16), with 4.8% due to bradydysrhythmias (95% CI, 2.2-6.4), and 2.6% due to tachydysrhythmias (95% CI, 1.1-3.1).[32] A prospective cohort study of 1418 patients reported that, of the deaths in patients with syncope at 1 year, 37% were cardiac related. The all-cause mortality rate after an ED visit for syncope increased from 1.4% at 30 days to 4.3% at 6 months, and 7.6% at 1 year.[46]

Neurally Mediated Syncope

Neurally mediated syncope results when the reflexes that control circulatory homeostasis become dysfunctional, causing vasodilatation and/or bradycardia and a fall in blood pressure. Neurally mediated syncope is classified according to the following physiologic mechanisms:

- Vasodepressor type; characterized by loss of upright vasoconstrictor tone
- Cardioinhibitory type; characterized by bradycardia
- Mixed type; characterized by occurrence of both mechanisms

Typical neurally mediated vasovagal syncope is precipitated by a trigger event such as fear, severe pain, strong emotion, or instrumentation (eg, having blood drawn). Situational syncope occurs during or directly after specific events, including micturition, coughing, defecation, vomiting, or swallowing. Carotid sinus syncope occurrs during carotid sinus stimulation.

A prospective study of 280 patients with neurally mediated syncope identified 14% of the cases with typical neurally mediated (vasovagal) syncope, 12% with situational syncope, and 12%

Table 1. Relevant Practice Guidelines For Syncope

Organization	Title	Year Published
American College of Emergency Physicians[40]	Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with syncope	2007
European Society of Cardiology[41]	Guidelines for the diagnosis and management of syncope	2009
National Institute of Health and Care Excellence[42]	Transient loss of consciousness ('blackouts') management in adults and young people	2010
Canadian Cardiovascular Society[43]	Standardized approaches to the investigation of syncope: Canadian Cardiovascular Society position paper	2011

Table 2. Classification Of Syncope By Cause

Neurally Mediated

- Typical neurally mediated (vasovagal)
  - Fear
  - Severe pain
  - Strong emotion
  - Instrumentation
  - Valsalva (weight lifters)
  - Breath-holding spell
- Situational
  - Postexercise
  - Coughing
  - Micturition
  - Defecation
  - Vomiting
  - Swallowing
  - Carotid sinus stimulation / hypersensitivity

Orthostatic Hypotension-Mediated

- Volume depletion
  - Hemorrhage
  - Dehydration
  - Diarrhea
  - Vomiting
  - Septic/distributive shock
- Primary autonomic failure
- Secondary autonomic failure
  - Drug-induced autonomic failure

Cardiovascular-Mediated

- Dysrhythmias
  - Second- or third-degree AV block
  - Atrial fibrillation/flutter
  - Ventricular tachycardia
  - Sick sinus syndrome
  - Torsades de pointes
  - Supraventricular tachycardia
  - Pre-excitation
  - Long QT syndrome
  - Brugada syndrome
  - Pacemaker malfunction
- Structural cardiovascular disease
  - Valvular heart disease
    - Aortic stenosis
    - Mitral stenosis
    - Tricuspid stenosis
  - Cardiomyopathy
  - Congenital heart disease
  - Myxoma
  - Pericardial tamponade
  - Aortic dissection
  - Myocardial infarction
  - Severe congestive heart failure
  - Pulmonary hypertension
  - Pulmonary embolism
  - Subclavian steal syndrome

Abbreviation: AV, atrioventricular.

with carotid sinus syncope. Typical neurally mediated syncope occurs more often in younger age groups (with a lower prevalence of cardiovascular disease) and it is characterized by more prodromal symptoms, longer duration of symptoms, more symptoms during recovery, and a lower prevalence of sustained injury, compared to other forms of neurally mediated syncope.[47]

Postexercise-related syncope occurs in young athletes as a form of situational syncope. Nonexertional and postexertional syncope in young athletes is almost always neurally mediated and has a low recurrence rate. However, exertional syncope, though infrequent (1.3% of athletes with syncope), may be caused by cardiovascular abnormalities.[48] Recurrent exercise-related syncope in young athletes without cardiovascular disease (after exclusion by a negative cardiac evaluation including echocardiography) is not associated with adverse outcome.[48,49]

Orthostatic Hypotension-Mediated Syncope

Orthostatic hypotension is defined as an abnormal fall in systolic blood pressure (SBP) (> 20 mm Hg) after standing, that results in global cerebral hypoperfusion and symptoms (eg, dizziness, light-headedness, and near-syncope). Orthostatic hypotension is common in patients with syncope and is detected in the vast majority of patients (89%) by 2 minutes after standing.[50] Causes of orthostatic hypotension include:

- Volume depletion by hemorrhage or volume loss.
- Autonomic nervous dysfunction in which the sympathetic nervous system is unable to adequately produce sufficient peripheral vasoconstriction after standing up. It can be caused by a primary dysfunction or a secondary process (eg, diabetes or drugs).

A prospective cohort study of syncope patients in the ED found that orthostatic hypotension was considered the cause in 24% of cases; 37% had drug-induced hypotension; 21% had hypovolemia; 12% had postprandial hypotension; and 29% had idiopathic hypotension. Asymptomatic orthostatic hypotension was found in 10% of patients with syncope attributed to other causes. Compared to patients with neurally mediated syncope, those with orthostatic hypotension were significantly older, had more comorbidities, and were more frequently hospitalized. Drug-related hypotension was the most frequent cause for this disorder.[51]

Cardiovascular-Mediated Syncope

Cardiovascular causes are the third most common reasons for syncope, and are due primarily to dysrhythmias or structural cardiovascular disease. Obstruction of blood flow may be one of the mecha-

nisms involved in syncope associated with pulmonary embolism and aortic dissection.

Dysrhythmias are the most frequent cause of syncope due to cardiovascular causes. They can be due to intrinsic cardiac factors such as conduction disturbances or extrinsic factors such as drugs. Causes include ischemia, sick sinus, long QT, preexcitation, and Brugada syndrome.

Structural cardiovascular diseases are diseases of the myocardium, heart valves, or pericardial/vascular wall linings that directly cause fixed or dynamic obstruction to forward flow or that indirectly impede flow by myocardial ischemia, resulting in acute or chronic compromise of cardiac output.

Syncope has been observed in patients with pulmonary embolism, and up to 20% of patients with a massive pulmonary embolism will have syncope.[41] Subclavian steal syndrome is a rare vascular cause of brain hypoperfusion, leading to syncope. It is caused by reversed blood flow in the vertebral artery due to a proximal narrowing of the subclavian artery. With movement of the ipsilateral arm, blood is shunted from the vertebrobasilar system to the arm musculature, resulting in cerebral hypoperfusion.

Differential Diagnosis

One of the first steps in approaching the patient with syncope is to distinguish it from other causes of transient loss of consciousness (eg, vertebrobasilar transient ischemic attack, seizure, or metabolic disorder). Any pathological process with pain may cause neurally mediated syncope. Any disease process accompanied by hypovolemia, shock, or autonomic dysfunction can have orthostatic symptoms and result in syncope. **Table 3** presents conditions that may mimic syncope but are not due to transient global cerebral hypoperfusion.

Stroke Or Transient Ischemic Attack

Neurologic disorders are rarely the primary cause of syncope. A few stroke syndromes (such as brain stem ischemia) can have brief episodes of transient loss of consciousness as a symptom of decreased blood flow to the reticular activating system. The episodes are typically associated with other neurologic symptoms of posterior circulation ischemia.[52] Subarachnoid hemorrhage is a consideration in some cases of syncope, and is usually accompanied by other symptoms such as sudden headache, altered mental status, or focal neurologic deficits. The assumed mechanism of subarachnoid hemorrhage resulting in syncope is decreased brain perfusion caused by a temporary rise in intracranial pressure.

Seizures

Transient cerebral hypoperfusion with neurally mediated syncope may cause brief, jerking limb movements that may be mistaken for a tonic-clonic seizure. Convulsive-like movements or myoclonic activity occurs in 28% to 90% of patients with neurally mediated syncope.[53,54] One study of patients diagnosed with epilepsy reported a misdiagnosis rate of 13%.[55] Prodromal symptoms consistent with neurally mediated syncope make the diagnosis of epileptic seizure less likely. Unconsciousness lasting more than 5 minutes, unusual posturing, tonic-clonic limb movements, a bite on the lateral aspect of the tongue, and a slow return to full alertness or prolonged confusion after the event are suggestive of a seizure. A meta-analysis reported a specificity of 96% and a sensitivity of 33% for lateral tongue biting in differentiating between seizures and syncope.[56]

Metabolic Disorders

Hypoglycemia in known diabetic patients may rarely cause transient loss of consciousness by mechanisms not fully understood. Autonomic mechanisms may be part of the pathophysiology. It is unlikely that hypoglycemia causing transient loss of consciousness will resolve without intervention.

Toxins

A variety of agents can cause transient loss of consciousness by central nervous system and respiratory depression. Agents with a short onset of action and short half-life may mimic syncope, though most toxins will cause prolonged loss of consciousness.

Table 3. Conditions That May Mimic Syncope

TLOC Without Global Cerebral Hypoperfusion

- Neurologic
  - Seizures
  - Vertebrobasilar transient ischemic attack
  - Subarachnoid hemorrhage
  - Subdural/epidural hemorrhage
  - Traumatic brain injury
- Metabolic disorders
  - Hypoglycemia
  - Hypoxia
  - Hyperventilation
- Intoxication
  - Drug exposure
  - Chemical/toxic gas exposure

Disorders Without TLOC

- Cataplexy
- Drop attacks and falls
- Psychogenic
  - Somatization disorder
  - Anxiety disorder
  - Conversion

Abbreviation: TLOC, transient loss of consciousness.

Psychiatric Conditions

Psychiatric conditions can mimic syncope; however, they are always a diagnosis of exclusion. Presentations can range from fully conscious actions for secondary gain to dissociative states where the patient has no conscious control over the activity. Hyperventilation associated with panic disorder can cause syncope by hypocarbia and subsequent cerebral vasoconstriction. Various psychiatric drugs can cause orthostatic hypotension and prolonged QT intervals, and thus, a risk for a dysrhythmia as the cause for the syncopal event. A prospective cohort study found that 20% of patients with syncope met the diagnostic criteria for at least 1 major psychiatric or drug/alcohol disorder, and 20% of patients were twice as likely to have recurrent syncope and have more prodromal symptoms.[57] Other studies confirmed a positive association between psychiatric disorders or substance abuse with syncope of unclear etiology.[58,59]

Prehospital Care

Prehospital care of a patient who has suffered a temporary loss of consciousness starts with assessing and stabilizing the airway, evaluating breathing and circulation, and assessing blood glucose. In true syncope, the patient will, typically, have regained consciousness before the ambulance arrives. Assessment for life-threatening causes of syncope is the first priority for the prehospital provider. When traumatic head injury is suspected as a complication of syncope, the cervical spine should be evaluated and immobilized, as appropriate, according to the National Emergency X-Radiography Utilization Study (NEXUS) Criteria, Canadian C-spine Rule, or other local emergency medical services (EMS) protocols. Intravenous access should be obtained if the patient is hypotensive or symptomatic. Generally, an ECG should be obtained, and, in cases of suspected myocardial infarction, the ECG should be transmitted to the base station/cardiac center, if possible.

EMS personnel should be aware of risk factors associated with adverse outcome in patients who have experienced syncope and ensure the immediate transport of any high-risk patient to the ED. Transport to a regional center should be provided for patients who have clinical findings suggestive of stroke, trauma, or ST-segment elevation myocardial infarction.

Emergency Department Evaluation

The approach to the patient with syncope has 3 steps: (1) identify life-threatening conditions; (2) perform a systematic evaluation to determine the etiology of the syncope; and (3) perform risk stratification for possible adverse (cardiac) outcomes when the etiology is unclear.

History

Patients presenting with a history of syncope have a potentially life-threatening process until proven otherwise, and rapid triage with stabilization is essential. Start with a broad differential that includes all causes of transient loss of consciousness before assuming that the patient has experienced true syncope. If possible, interview witnesses for important details occurring just prior to or during the event, since the patient might not have an accurate recollection of the event. See **Table 4 (page 7)** for a list of important historical facts. See **Table 5 (page 7)** for a list of symptoms that may suggest a life-threatening cause.

If no life-threatening cause is suspected, make a judgment as to whether the event was truly syncope. Perform a careful history and determine whether there was a brief loss of consciousness and loss of postural tone. If a patient has not spontaneously recovered to his baseline level, the episode was not a true syncope. In patients with true syncope, attempt to discover if it was cardiovascular-mediated, neurally mediated, orthostatic hypotension-mediated, or due to some other cause. Ask about a family history of sudden cardiac death.

Inquire whether symptoms such as dizziness/near-syncope were present after standing up from a sitting or a supine position. Review the patient's medication list, including over-the-counter and recreational drugs. Drug-related hypotension is a frequent cause of orthostatic hypotension (37%).[51] Ask about new medications and changes in medication dose or frequency. Check for possible drug interactions. **Table 6 (page 8)** lists clinical features suggesting a diagnosis of syncope.

The most common prodromal symptoms of neurally mediated syncope are pallor, dizziness, and diaphoresis.[45] Other predictors of neurally mediated syncope include syncope immediately after standing up, blurred vision, nausea, warmth, light-headedness, prolonged sitting or standing prior to syncope, duration of recovery more than 1 minute, or fatigue following syncope.[60,61]

Predictors of cardiovascular-mediated syncope include: older age, presence of structural heart disease, syncope occurring in supine position or with exertion, absence of or short prodromal symptoms, and chest pain preceding syncope. Other features suggestive of cardiovascular syncope include the presence of suspected or established heart disease after the initial evaluation, palpitations, and absence of nausea, vomiting, diaphoresis, and blurred vision preceding syncope.[3,15,35,60-65]

Physical Examination

Abnormal vital signs may be the key in identifying the etiology of syncope. Hypotension and tachycardia are suggestive of hypovolemia and persistent

Table 4. Important Historical Facts For Syncope

Prior to the Episode

- Activity
  - During or after exercise; during or after standing up; while in supine position; during or immediately after micturition/defecation, coughing, or swallowing
- Prodromal signs
  - Dizziness, pallor, diaphoresis, blurred vision, warmth, light-headedness
- Circumstances
  - Prolonged standing, warm or crowded environment, postprandial, experiencing fear or pain, neck movements, instrumentation

At Onset of the Episode

- Associated symptoms
  - Palpitations; chest pain; radiating pain to arms, jaw, or back; ripping/tearing back pain; abdominal pain; dyspnea; pleuritic chest pain; sudden headache; neck pain; paralyses; melena; diarrhea; fever; weakness
- Timing of symptoms
  - Prolonged, sudden

Witness Information

- Fall/injury
  - Mechanism of falling (sudden, slumping, or kneeling over), losing consciousness first, head trauma
- Duration of loss of consciousness
  - Seconds or minutes
- Movements
  - No movement; jerking or tonic/clonic movements; duration of movements
- Associated symptoms
  - Skin color (pallor, cyanosis, flushing), breathing pattern (snoring)

After the Episode

- Mental status
  - Confusion, length of recovery time
- Associated symptoms
  - Palpitations; chest pain; radiating pain to arms, jaw, or back; ripping/tearing back pain; abdominal pain; dyspnea; pleuritic chest pain; sudden headache; paralyses, melena
  - Diarrhea, fever, weakness, incontinence of urine or feces, tongue bite
  - Diaphoresis, nausea, vomiting, fatigue, muscle aches, injury

Past Medical History

- Family history
  - Sudden death, fainting, congenital heart disease
- Cardiovascular history
  - Structural heart disease, coronary artery disease/myocardial infarction, dysrhythmias
- Neurological history
  - Parkinsonism, epilepsy
- Metabolic disorders
  - Diabetes
- Medications (including drugs of abuse)
  - Prescribed, over-the-counter, and recreational
- Previous events
  - Previous syncope, associated symptoms, and diagnosis

tachypnea and/or low oxygen saturation may suggest pulmonary embolism.

A drop in blood pressure with recognizable symptoms following 1 to 3 minutes standing, preceded by a drop in blood pressure after 5 minutes in a supine position is considered diagnostic for orthostatic hypotension. Interestingly, 1 study reported asymptomatic changes in SBP in 10% of patients with syncope that was ultimately attributed to other causes.[51]

The cardiac examination focuses on detecting outflow obstruction and valvular regurgitation. Check for signs of (right-sided) outflow obstruction and heart failure by looking for distended neck veins. Listen for murmurs suggesting valvular diseases (such as aortic stenosis). Check capillary refill, and peripheral pulses, and assess for edema and cyanosis.

Table 5. Typical Clinical Characteristics Related To Possible Life-Threatening Causes Of Syncope

Life-Threatening Etiology	Clinical Characteristics
Subarachnoid hemorrhage	Sudden headache Worst headache ever Neurologic deficit
Cerebrovascular accident	Neurologic deficit
Acute myocardial infarction	Chest pain Radiating pain to back/arms/jaw
Aortic stenosis	Chest pain Dyspnea Syncope on exertion
Thoracic aortic aneurysm and dissection	Chest pain Ripping pain between shoulder blades Radiating pain/symptoms: • Ascending aorta (throat/jaw) • Descending aorta (back) Neurologic deficit in case of involvement of carotid artery or lumbar artery Chest pain with or without radiation in case of involvement of coronary artery
Massive pulmonary embolism	Dyspnea Pleuritic chest pain associated with breathing Syncope on exertion
Abdominal aortic aneurysm and dissection	Abdominal pain with or without radiation to back Neurologic deficit in case of lumbar artery involvement
Gastrointestinal bleed	Melena
Ruptured ectopic pregnancy	Abdominal pain
Sepsis	Fever Signs consistent with specific infectious sources (eg, headache, confusion, cough, dysuria, abdominal pain)

Syncope rarely has a neurologic etiology; however, a systematic neurologic examination with attention to the cranial nerves and a survey for focal neurologic findings should be done.

Syncope patients who fall may experience significant trauma. Consider the possibility of head and neck trauma and immobilize the cervical spine, when appropriate. An observational cohort study found that 29% of patients with syncope sustained an injury, though the characteristics of the trauma were of little value in determining the specific cause of the syncope.[66]

Examine the patient for possible infection sources, palpate the abdomen for a pulsating mass suggesting an aortic aneurysm, and perform a rectal examination to look for gastrointestinal bleeding.

Diagnostic Studies

A variety of diagnostic tests are used in syncope; however, the overall yield is low, and testing must be done judiciously. An estimated pretest probability for the etiology and an understanding of test sensitivity and specificity are key in deciding which diagnostic tests may be useful.

Table 6. Clinical Features Suggesting A Diagnosis Of Syncope

Cardiovascular-Mediated Syncope
• Structural heart disease
• Family history of sudden cardiac death
• Syncope during exertion or while supine
• Palpitations associated with syncope
• Abnormal ECG suggesting dysrhythmic syncope
▪ Any nonsinus rhythm
▪ LBBB
▪ Left axis deviation
▪ Bifascicular block
▪ RBBB with first-degree AV block
▪ RBBB with LAFB or LPFB
▪ Mobitz type I second-degree or third-degree AV block
▪ Nonsustained VT
▪ Pre-excitation (delta wave)
▪ Prolonged QT or Brugada pattern
▪ Signs of AMI and new ischemia
Neurally Mediated Syncope
• Precipitated by prolonged standing or trigger event
• Prodrome with nausea, vomiting, blurred vision, feeling warm, diaphoresis
• During a meal; postprandial; during or directly after micturition, defecation, coughing, or swallowing
• With head rotation or pressure on carotid sinus
• After exertion
Orthostatic-Mediated Syncope
• After standing up
• A change in vasodepressive drugs
• Autonomic dysfunction (Parkinsonism)

Abbreviations: AMI, acute myocardial infarction; AV, atrioventricular; ECG, electrocardiogram; LAFB, left anterior fascicular block; LBBB; left bundle branch block; LPFB, left posterior fascicular block; RBBB, right bundle branch block; VT, ventricular tachycardia.

Electrocardiogram

An ECG is recommended in every patient with syncope except for patients with a clearly identified etiology/trigger (eg, a noxious stimulus like a blood draw). The overall diagnostic yield of an ECG in a syncope patient is 2% to 9%.[3,21,67] In patients aged < 40 years without evidence of heart disease, the diagnostic yield is 0% to 3%.[39,67] Key features to focus on when reading the ECG are: (1) evidence of ischemia, (2) conduction disturbances, (3) a pre-excitation pattern (delta wave), (4) prolonged corrected QT interval, and (5) a Brugada pattern. A normal ECG has a high negative predictive value.[62] Abnormal initial ECG findings are well correlated with potential dysrhythmic causes of syncope.[68,69] In a prospective cohort of 1474 patients with syncope and near-syncope, 3.1% of patients were diagnosed with an acute myocardial infarction. The initial ECG was abnormal in 80% of patients with acute myocardial infarction.[70]

An abnormal ECG is a tool for risk stratification and guides more specialized cardiovascular tests. Several studies have associated the following conditions with adverse cardiac outcome in 30 days: left bundle branch conduction abnormalities, any nonsinus rhythm during ED stay, a second-degree Mobitz type II or third-degree atrioventricular block, bundle branch block with first-degree atrioventricular block, right bundle branch with left anterior or posterior fascicular block, new ischemic changes, left axis deviation, or ED cardiac monitor abnormalities.[71,72] In a prospective study where patients with bundle branch block and syncope followed an extensive cardiovascular diagnostic workup, 83% received a definitive diagnosis.[29] A study of patients with an unclear cause of syncope after initial evaluation found that frequent or repetitive premature ventricular contractions and sinus pauses (compared to rare premature ventricular contractions) were independent ECG predictors of sudden death and mortality at 2 years (28.3% vs 10.8%).[73]

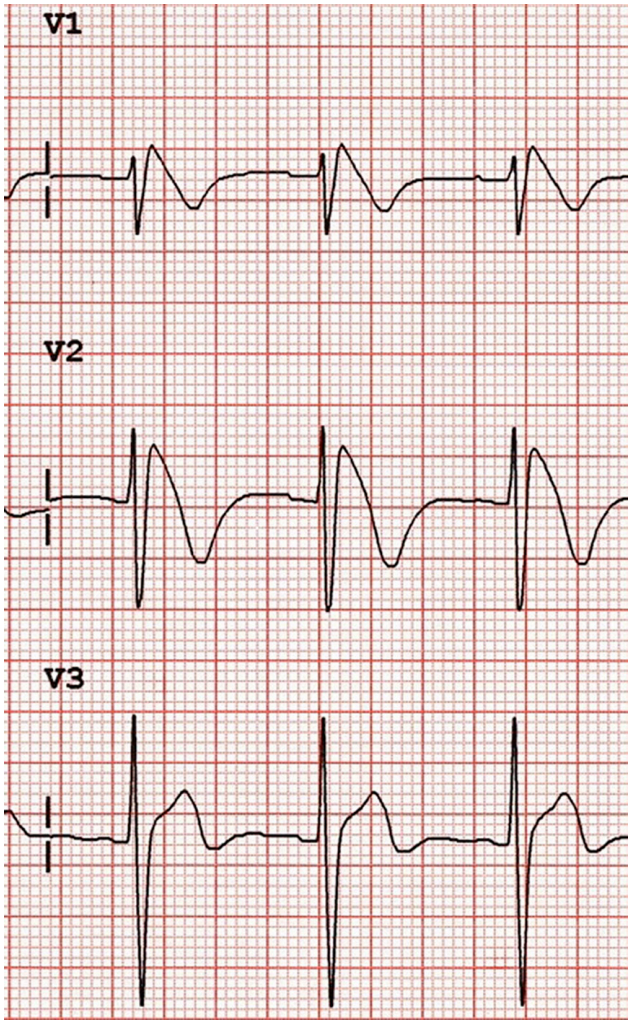
Brugada Syndrome

Brugada syndrome is a genetic disease that is characterized by abnormal ECG findings and an increased risk of sudden cardiac death. About 1 in 3 patients with Brugada syndrome present with syncope as the first manifestation. A retrospective study reported that more than one-third of patients with Brugada syndrome have a normal ECG at first evaluation.[74] The classic ECG pattern in Brugada syndrome is a coved ST-segment elevation > 2 mm followed by a negative T wave in precordial leads V_1 through V_3 (pseudo right bundle branch block). **See Figure 1 (page 9)**.

Prolonged Monitoring

Several studies have looked at the usefulness of prolonged monitoring in patients with unexplained syncope. The highest yield was in patients with positive cardiac history and an abnormal ECG. The ideal duration for prolonged monitoring is unclear, but it seems reasonable to monitor high-risk patients for 24 to 72 hours. One study of patients with a positive cardiac history and abnormal ECG undergoing 24-hour Holter monitoring reported a 12% diagnostic yield.[75] The diagnostic yield is highest within 24 hours and much lower after 48 hours.[76] However, another study found that 24-hour Holter monitoring was too brief to identify all potentially important dysrhythmias; the yield was 15% for the first 24 hours, 11% for 24 to 48 hours, and 4.2% for 48 to 72 hours.[77] The best cut-off time seems to be 72 hours (especially in older patients presenting with heart failure [sensitivity 73%, specificity 86%]).[78]

Figure 1. Brugada Type I On Electrocardiogram



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Echocardiography

Echocardiography can provide information on left ventricular function (a predictor of dysrhythmias). Routine echocardiography in syncope patients has a very low yield, but it can be useful in patients with unexplained syncope and a cardiac history or abnormal ECG, with a yield up to 27%. It is useful in confirming an unknown aortic stenosis in patients with suggestive signs and symptoms. It rarely provides an unsuspected diagnosis.[39,79] A study on patients with syncope and coronary artery disease with a nondiagnostic electrophysiological evaluation revealed that, in patients with a reduced ejection fraction, the risk of sudden death and ventricular dysrhythmias is up to 10% per year.[80]

Carotid Sinus Massage

Carotid sinus massage is diagnostic for carotid sinus hypersensitivity when resulting in a ventricular pause lasting > 3 seconds and/or a fall in SBP of > 50 mm Hg. If accompanied by syncope, it is diagnostic of carotid sinus syndrome. Carotid sinus massage is recommended by the European Society of Cardiology guideline on syncope in patients > 40 years of age with unexplained syncope after initial evaluation.[41] Carotid sinus massage should be avoided in patients with previous transient ischemic attack, stroke within the past 3 months, or carotid bruits (except if carotid Doppler studies excluded significant stenosis). Neurologic complications occurred in 0.29% of studied patients who underwent carotid sinus massage.[41] It should be performed while monitoring the patient and with resuscitation equipment close at hand. Carotid sinus hypersensitivity, orthostatic hypotension, and neurally mediated syncope are common conditions affecting older patients with and without syncope, and falls are likely to coexist.[81,82] The finding of a hypersensitive response should not necessarily preclude further investigation for other causes of syncope, however.

For patients with any history of falls, syncope or dizziness, and carotid sinus hypersensitivity, the sensitivity of carotid sinus massage is 41% and specificity is 64%. When carotid sinus hypersensitivity is accompanied by symptoms of syncope, near-syncope, or dizziness, the sensitivity is 17% and specificity is 86%.[82] Other studies of very elderly patients with syncope reported that carotid sinus syndrome is the most common etiology of syncope in this group and it is significantly more common in subjects aged > 80 years, with a diagnostic yield of 34% to 48%.[83,84] A prospective observational cohort study recommended carotid sinus massage as the first diagnostic maneuver after a nondiagnostic initial evaluation for older patients with syncope complicated by a severe trauma.[66]

Chest X-Ray

Unless guided by specific symptoms, a routine chest x-ray in patients with syncope has a very low diagnostic yield and is not recommended.[3]

Head Computed Tomography

Head computed tomography (CT) is rarely helpful unless neurologic signs and symptoms are present.[21] Multiple studies concluded that routine head CT does not yield relevant clinical findings in syncope patients.[3,85-87]

Electroencephalography

Routine electroencephalography has an extremely low diagnostic yield in syncope and is not recommended.[88-91]

Laboratory Testing

Several studies have shown low yield of laboratory testing unless guided by history and physical examination. Medication use is important to consider in suspected electrolyte disturbances. Abnormal results in complete blood count, electrolytes, and serum glucose range from 0% to 5% in patients with syncope. It is unclear wehther the results provide a cause for syncope.[2,14,16,56]

Serum/Urine Pregnancy Test

One systematic review stated that a pregnancy test has a very low yield in finding the cause of syncope.[14] However, a urine human chorionic gonadotropin (hCG) test is inexpensive, noninvasive, and should be obtained in women of child-bearing age.

Biomarkers

Cardiac biomarkers may be useful in select cases of syncope. One small study on troponin I in the ED concluded that acute myocardial infarction is infrequent (1.4%), and troponin I determination provides little additional benefit to the initial ECG in identifying patients with syncope due to acute myocardial infarction. Troponin I is not recommended to rule out acute myocardial infarction in adult patients presenting with isolated syncope.[92] However, elevated troponin predicts adverse cardiac outcome in syncope and may be useful for risk stratification.[92-94]

Four small prospective studies on the usefulness of N-terminal pro-brain natriuretic peptide (NT-pro BNP) in discerning cardiac from noncardiac syncope found promising results, with sensitivities around 90% and specificities ranging from 51% to 93%. These studies were done in highly selected groups of hospitalized patients and 1 was done in children.[95-98] The use of NT-pro BNP in the ED remains unclear.

A meta-analysis of serum prolactin measurement within 1 hour of syncope for differentiating between seizures and syncope showed that a positive result (> 3 times baseline) was highly predictive of generalized tonic-clonic seizures; however, a negative result did not exclude a seizure.[99] A small study looked at serum creatinine kinase and myoglobin for differentiating between syncope and seizure and found they were not useful in the ED.[100]

Management Of Syncope

The wide range of causes of syncope results in an even wider range of possible management strategies.

Risk Factors

Analyzing the literature to determine predictors of adverse outcomes after syncope is challenging because of the large variability in the definition of an "adverse event" and the timing of the event. Outcome determinations range from 7 days to 5 years. Clearly, a 1-year outcome risk is not as relevant as a 3-day to 7-day risk in the ED determination on whether to admit or discharge a patient.

Cardiovascular findings and evidence of bleeding are the 2 most powerful predictors of an adverse outcome after syncope.[32] The risk of short-term adverse outcome after an ED visit for syncope declines sharply after 7 days. A retrospective study of more than 35,000 patients reported short-term adverse cardiac outcomes in 3% of syncope patients.[101]

The presence of multiple potential causes for syncope is an independent predictor of increased mortality.[102] Conversely, in patients with identified benign etiologies for their syncope or near-syncope (neurally mediated or from dehydration), an adverse outcome within 30 days is unlikely, despite the presence of risk factors.[103,104]

Risk factors for short-term and longer-term outcomes consistently reported in the literature include: cardiovascular diseases or structural heart disease, congestive heart failure,[10,12,35,37,101,105-113] older age,[101,111,113] male sex,[10,101,106,107,114-117] and abnormal ECG.[106,111,113,117]

Risk Stratification Decision Rules

There is no single decision rule that is sufficiently sensitive and specific to use in the ED setting. However, decision rules do provide a framework for clinical decision making. The challenge of developing risk scores is in making them reliable. Since adverse outcomes are relatively rare, syncope studies must recruit large numbers of patients in order to be sufficiently powered to derive and validate a decision rule. In all studies reviewed, the sample sizes were too small to do this. Variation of inclusion criteria and definitions adds to the complexity of interpreting the many studies in the literature and precludes performing good-quality meta-analyses.

Several studies have been performed deriving and validating risk scores or decision rules for syncope patients, including the San Francisco

Syncope Rule (SFSR),[30,113,118-127] the Osservatorio Epidemiologico sulla Sincope nel Lazio (OESIL) risk score,[93,113,128,129] the Risk stratification Of Syncope in the Emergency Department (ROSE) decision instrument,[130-132] the Boston Syncope Criteria,[133,134] and the Evaluation of Guidelines in SYncope Study (EGSYS) score.[113,135] **Table 7** shows the SFSR and OESIL risk factors. The SFSR attempts to predict short-term adverse outcome within 7 days, and it may help with physician decision making and decrease hospital admissions.[124] In most external validation studies, sensitivity and specificity were lower than in the derivation studies (as expected), but correlated well with adverse outcome.[30,119-123,127]

A systematic review of the SFSR reported a sensitivity of 87% (95% CI, 79%-93%), and a specificity of 52% (95% CI, 43%-62%). There was substantial heterogeneity among the studies. The probability of a serious outcome when given a negative score with the SFSR was < 5%. The probability was < 2% when the rule was applied only to patients for whom no cause of syncope was identified after initial evaluation in the ED. Missed cardiac disease was the most common cause of a false-negative classification.[118] Another meta-analysis reported a sensitivity of 86% (95% CI, 83%-89%) and a specificity of 49% (95% CI, 48%-51%).[136] Differences in study design and ECG interpretation may account for the variable prognostic performance of the SFSR when validated in different practice settings.

In several external validation studies, the OESIL risk score was found to be predictive of adverse cardiac outcome and mortality and useful in reducing unnecessary hospital admissions. Unfortunately, the OESIL score has not performed with consistently high sensitivities among studies.[93,113,129,137] A meta-analysis of 3 studies reported a sensitivity of 95% (95% CI, 88%-98%) and specificity of 31% (95% CI, 29%-34%).[136]

Table 7. The SFSR And OESIL Decision Risk Factors For Syncope

SFSR	OESIL
C - History of congestive heart failure	Age > 65 years, 1 point
H - Hematocrit < 30%	History of cardiovascular disease, 1 point
E - Abnormal ECG	Syncope without prodrome, 1 point
S - Shortness of breath	Abnormal ECG, 1 point
S - Triage systolic blood pressure < 90 mm Hg	
A patient with any of the above measures is considered at high risk for a serious outcome	A score ≥ 2 points implies an increased risk of cardiac death

Abbreviations: ECG, electrocardiogram; OESIL, Osservatorio Epidemiologico sulla Sincope nel Lazio; SFSR, San Francisco Syncope Rule.

The ROSE rule consists of the following risk factors: brain natriuretic peptide (BNP) ≥ 300 pg/mL, bradycardia ≤ 50 beats/min, a rectal examination showing fecal occult blood, anemia ≤ 90 g/L, chest pain associated with syncope, ECG showing Q-wave (not in lead III), and oxygen saturation ≤ 94% on room air. Outcome was all-cause mortality at 1 month. The recommendation is that if 1 of these items is positive, the patient needs to be admitted. The rule has not been adequately externally validated and does not perform well at predicting 1-year adverse outcome of ED syncope patients.[130-132]

The Boston Syncope Criteria encompass signs and symptoms of coronary artery disease, signs of conduction disease, worrisome cardiac history, valvular heart disease by history or physical examination, family history of sudden cardiac death, persistent abnormal vital signs in the ED, volume depletion, and primary central system nervous event. The primary outcome is either a critical intervention or an adverse outcome within 30 days. This rule includes patients with transient loss of consciousness.[133,134]

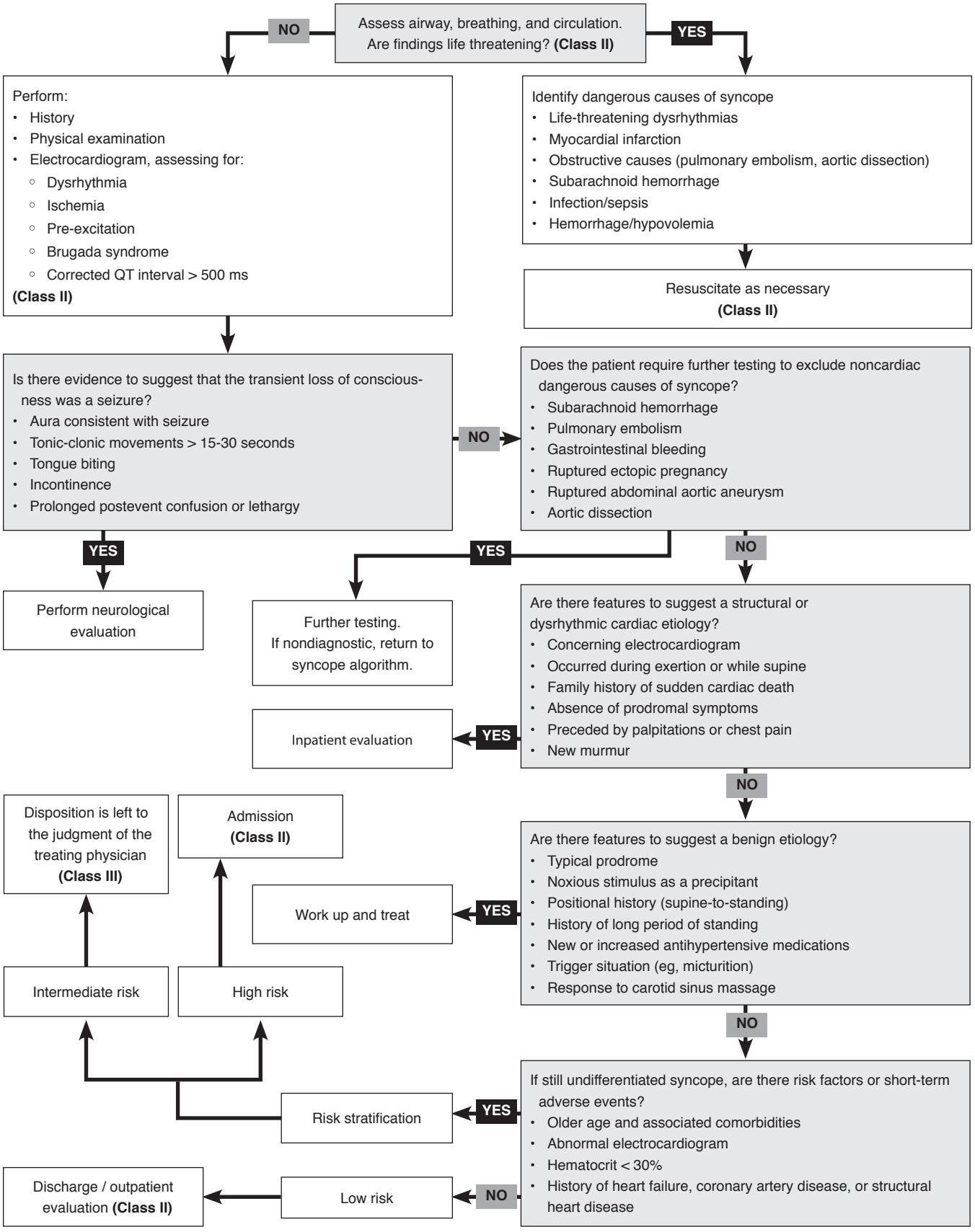
The EGSYS score consists of an abnormal ECG and/or heart disease, palpitations before syncope, syncope during effort or in supine position, absence of autonomic prodromes, and absence of predisposing and/or precipitating factors. The EGSYS score attempts to predict cardiac syncope.[135] Neither rule has been adequately externally validated.

An interesting study comparing physician judgment and decision making with the SFSR showed that physician judgment is good when predicting which patients with syncope will develop serious outcomes, but contrary to their judgment, physicians still admit a large number of low-risk patients.[125] A comparable study on clinical judgment versus the OESIL score and SFSR showed that, with only clinical judgment, fewer patients would have been admitted; however, sensitivity would be lower (77%, 88%, and 81% for clinical judgment, OESIL score, and SFSR, respectively).

What Do The Guidelines Say?

The ACEP clinical policy on syncope gives a level B recommendation to admitting patients with syncope who have high risk factors for adverse outcomes, including: older age and associated comorbidities, abnormal ECG (including acute ischemia, dysrhythmias, or significant conduction abnormalities), hematocrit < 30% (if obtained), history or presence of heart failure, coronary artery disease, or structural heart disease. A study testing these recommendations found high sensitivity (100%) and specificity (81%) in identifying patients with cardiac syncope and a significant reduction in the hospital admission rate.[138] Another study confirmed this sensitivity; however, the low specificity

Clinical Pathway For Syncope



Please see Class of Evidence Definitions on page 13.

(26%) led to unnecessary admissions.[137] A comparison of preadmission and postadmission rates after implementation of the ACEP guideline showed a decline in admission rates.[139]

The Canadian Cardiovascular Society concluded that there is little persuasive evidence that ED syncope rules and diagnostic syncope units provide efficient care and improved outcomes, but that formal diagnostic algorithms with specialist support show promise.[43] The ESC, NICE, and Canadian Cardiology Society guidelines do not give recommendations on using decision rules and provide a list of known risk factors to decide on admission.[41-43]

Special Circumstances

Pediatric Syncope

Syncope in adolescents and children is generally a benign event. In a large cohort of ED visits of patients aged 7 to 18 years, 0.9% were for syncope.[140] The approach to pediatric patients with syncope is the same as in adults. History, physical examination, and ECG are most helpful in determining a diagnosis and in guiding testing. The yield of unguided diagnostics (laboratory tests, head CT) is low.[141-144] The most common diagnosis in pediatric groups is neurally mediated syncope (65%-80%), distantly followed by orthostatic hypotension and cardiac syncope.[141,144,145]

Several studies of syncope in children show the same characteristics in neurally mediated and cardiovascular-mediated syncope as in adults. Cardiovascular syncope is mostly triggered by exercise, less frequently has prodromes, and occurs more often in a supine position. Children with cardiac syncope more often have a family history of syncope, sudden death, myocardial disease or dysrhythmias, a history of cardiac disease or an abnormal ECG. Neurally mediated syncope is triggered by fear (or other emotion), pain, prolonged standing, or being in a warm, crowded place.[145-147] Breath-holding spells are considered a form of neurally mediated syncope.

In a prospective cohort study of high-risk children with exercise-related syncope and an abnormal ECG, neurally mediated syncope was still established in 51% of patients. Cardiac syncope was diagnosed in 11%. The cause of syncope remained unexplained in 5.5% of patients. History of ECG abnormalities and exertional syncope were independent predictors of cardiac syncope. The sensitivity of history of an abnormal ECG for predicting cardiac syncope was 93.5%, with a specificity of 90.9%. The sensitivity of an exertional syncope for predicting cardiac syncope was 61%, with a specificity of 85.5%.[146]

Geriatric Syncope

The approach to elderly patients with syncope is the same as in all other adults. The differential diagnoses are comparable, though there is a higher incidence of cardiovascular causes and other comorbidities, and orthostatic hypotension.[64] Syncope and near-syncope in the elderly often results from polypharmacy or adverse drug reactions.[21,148]

Several studies have shown a higher mortality, morbidity, and recurrence rate of syncope in the elderly. One prospective cohort study with 2-year follow-up found a total mortality of 17% and a 32.5% recurrence rate.[149] Another showed a mortality of 30% compared to 8% in the young.[115] Cardiac syncope was significantly more frequent in deceased than in surviving patients (21.7% vs 12.3%), whereas neurally mediated and unexplained syncope did not differ.[149]

In patients with a noncardiovascular cause or unknown cause of syncope, a history of congestive heart failure, older age, and male sex are important prognostic factors.[115] Other risk factors for an adverse 30-day outcome in the elderly are age > 90 years, history of dysrhythmia, a SBP > 160 mm Hg, an abnormal ECG, and an abnormal troponin I level. A low-risk predictor was a complaint of near-synco-

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

pe rather than syncope.[107] Elderly women (despite being less likely to have cardiovascular comorbidities) are significantly more likely to present to an ED with syncope, yet less likely to be discharged with a defined etiology.[150]

Disposition

High-Risk Patients

In patients with life-threatening etiologies (eg, aortic dissection) or an unclear cause of syncope and assessed by risk stratification as high-risk, admission is advised. There is no clear evidence on how many risk factors a patient needs to require admission. A history or findings consistent with structural cardiac disease or heart failure alone would place the patient at high risk. Most guidelines recommend admission if 1 of these or if additional risk factors are present.[21,40,41,43] **Table 8** lists risk factors for an adverse outcome.

High-risk patients with unexplained or recurrent syncope are candidates for an electrophysiological evaluation. A prospective study detected electrophysiological abnormalities in 35% of patients with unexplained syncope.[151] In another study, the electrophysiological evaluation was positive in 44% of high-risk patients.[152] A history of injury related to loss of consciousness, ejection fraction ≤ 40%, a PR interval > 200 ms, bundle branch block, coronary artery disease, remote myocardial infarction, use of type I antiarrhythmic drugs, and male sex were independent predictors of a positive electrophysiological study.[151,153] An implantable loop recorder may also be of value in select patients.

Table 8. Risk Factors For An Adverse Outcome

- Syncope while supine, during exercise, or without prodromal symptoms
- Structural cardiac disease: ischemic, dysrhythmic, obstructive, valvular
- Abnormal ECG
- Heart failure in past history or current state, diminished left ventricular function
- Dyspnea
- Hypotension
  - SBP < 90 mm Hg
- Older age
- Anemia
  - Hematocrit < 30% (if obtained)
- Evidence of hemorrhage
  - Occult blood on rectal exam
- Male sex
- Family history of early sudden death aged < 50 years

Abbreviations: BP, blood pressure; ECG, electrocardiogram; SBP, systolic blood pressure.

Low-Risk Patients

Low-risk patients with a clear benign cause (eg, neurally mediated syncope) and patients with an unclear cause without risk factors are safe to discharge. Patients < 40 years of age with an isolated syncopal event who also have a normal physical examination, a normal ECG, and no evidence of structural or ischemic heart disease can safely be discharged.

Intermediate-Risk Patients

Intermediate-risk patients are the patients that are neither high-risk nor low-risk (eg, a 75-year-old patient with syncope of unclear etiology and, besides age, no other risk factors; or a patient with neurally mediated syncope with cardiovascular disease). This group of patients is deemed intermediate-risk by default, and their management remains unclear. The decision to admit is left to the treating physician. Different studies use different ages as the threshold for decision making. Age is a continuous variable that reflects the cardiovascular health of the individual rather than an arbitrary value.[40] One study reported that without other risk factors, age > 65 years alone was not a predictor of adverse outcome.[154] Another study found that patients > 50 years of age with a negative ED evaluation and no risk factors are safe to discharge.[155] Patients with benign etiologies for syncope, even with risk factors, do not benefit from hospitalization based on risk factors alone and are safe to discharge.[103]

Intermediate-risk patients may be good candidates for an observation unit. Consider the social situation of the patient, the ability to have a timely follow-up appointment, and the patient's wishes. All of these factors influence disposition. A study investigating predictors of hospitalization found that predictors of inhospital care include factors unrelated to the prognosis, such as unexplained etiology of syncope and the need for assistance with everyday activities.[156]

Consider referral for tilt-table testing for patients with recurrent syncope in whom heart disease is not suspected.[21] A meta-analysis showed good performance of tilt-table testing in discriminating between symptomatic patients with neurally mediated syncope and asymptomatic controls with a diagnostic odds ratio of 12 ($P < 0.001$).[157]

Discharge Instructions

Discharge instruction should provide clear direction on when and with whom to follow up. The instructions should include safety and prevention strategies.

Driving Recommendations

There is little evidence to support driving restrictions for patients with syncope. Syncope while driving could obviously lead to serious consequences for the patient and his surroundings. Depriving

patients of necessary transport has also negative consequences, so it depends on what is deemed an acceptable risk. Two studies reported a syncopal event while driving occurring in 10% of patients experiencing syncope, most commonly neurally mediated syncope (37%) followed by cardiac dysrhythmias (12%). Syncope recurrence rate during driving was < 1%.[158,159] Long-term survival in these patients was comparable to that of an age-matched and sex-matched cohort%.[158] It seems reasonable to not restrict driving in patients with a clearly identified benign cause.

Summary

The approach of the syncope patient in the ED has 3 steps: (1) determine if a life-threatening condition is present; (2) attempt to determine the etiology of the syncope if no life-threatening condition is found; and (3) in cases with unclear etiology, perform risk stratification for possible adverse outcomes.

A focused history and physical examination, including an ECG, will provide the clues to most life-threatening causes in patients presenting with syncope. Additional diagnostic testing is tailored to the individual patient and guided by history, physical examination, and ECG. After initial evaluation, if the cause is uncertain, a disposition decision is directed by risk stratification.

Several decision rules for syncope have been developed, though none have been shown to be sufficiently sensitive or specific to use in the ED setting. The decision rules provide an overview of existing risk factors that predict short-term and longer-term adverse events. Risk factors include syncope while supine, during exercise, or without prodromal symptoms; structural cardiac disease, heart failure in past history or current state, diminished left ventricular function, dyspnea, abnormal ECG, hypotension, older age, anemia, male sex, and family history of early sudden death. In patients with life-threatening etiologies or an unclear cause of syncope with high-risk factors, admission and monitoring are advised. There is no clear evidence on how many risk factors a patient needs to have to be admitted. Low-risk patients with identified benign etiologies or with unclear etiology without risk factors are safe to discharge. In intermediate-risk patients, the decision to admit is left to the treating physician. These patients may be good candidates for admission to an observation unit.

Acknowledgement

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Case Conclusions

The 51-year-old bicyclist who was also a marathon runner did not have improvement of her SBP, which remained at 90 mm Hg. Furthermore, she had throat pain, which could have been an angina equivalent. Your primary concern was that she had a cardiac outflow problem because of an aortic dissection or a pulmonary embolism. A neurally mediated component to her syncopal event could not be excluded. A CT aortogram was ordered to assess for dissection. It showed a type A aortic dissection starting in the ascending aorta extending to just above her renal arteries. Her spinal cord arteries originated from the true lumen, explaining why she had no neurologic or other symptoms. The throat pain was attributed to radiating pain from the intimal tear in her ascending aorta. She developed pain between her shoulder blades later during her stay in the ED while awaiting surgical intervention. She made a full recovery after surgery.

Time- And Cost-Effective Strategies

The strategies for time- and cost-effective strategies that best serve the patient are:

1. **Limit testing.**
   History, physical examination, and ECG should lead the diagnostic strategy. Unguided diagnostics and routine tests have low yield and increase costs. Concentrate on the most specific, sensitive, and cost-effective diagnostics.[13,18,143,160]
2. **Limit admissions.**
   Admission of patients with syncope results in a 50% discharge rate without a definitive diagnosis. There is no clear evidence that an adverse outcome is prevented by hospital admission. It is clear that unnecessary hospital admissions increase costs and patient risk for acquiring infections while in the hospital. Use guidelines or risk assessment tools to make an informed decision.[13,23,34,160,161]
3. **Follow a standardized protocol.**
   Many studies have shown that following a standardized protocol/algorithm based on current guidelines where history, physical examination, and ECG guide the diagnostic process reduces inappropriate admissions, increases diagnostic efficacy and accuracy, and reduces costs.[8,22,24,27,94,133,139,161-169]
4. **Use syncope observation units.**
   Syncope units use a standardized approach for diagnosing and treating patients. Several studies have shown improvement of diagnostic yield, reduction of hospital admission, and length of hospital stay without affecting recurrent syncope or all-cause mortality.[22,25,26,170-172]

In the second case, the patient had neurally mediated syncope with brief, rhythmic jerking movements of her extremities caused by cerebral hypoperfusion. She was not postictal and had no tongue bite. Her ECG was normal. She had no other life-threatening causes or risk factors and was safely discharged.

In the third case, the patient had syncope of unclear etiology. Detailed history revealed a short prodrome of light-headedness, no chest pain, and no palpitations. The syncope did not occur after standing up, and there was no orthostatic hypotension, so you performed risk stratification using the recommendations from the ACEP syncope policy. His risk factors were his age, ECG abnormalities with a left bundle branch block, and structural heart disease. He was admitted to the hospital for cardiac monitoring, and he turned out to have ventricular tachycardias, for which he received an implantable cardiac defibrillator.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

Risk Management Pitfalls For Syncope (Continued on page 17)

1. **"It didn't even occur to me that a patient with syncope might have a dissection of the thoracic aorta."**
   Syncope is generally a benign process. However, one must be proactive in trying to identify life-threatening causes. History, physical examination, and ECG findings are most helpful, but keep your differential large or you may miss the rare life-threatening conditions.

2. **"I sent the patient home after syncope with a history suggestive for cardiac syncope. There were no abnormalities on physical examination or on the ECG. The patient returned because of an accident with his truck after syncope."**
   People with occupations that are high risk for disastrous outcomes include truck drivers, bus drivers, airplane pilots, and heavy equipment operators. In particular, they need counseling about the risks of driving after syncope. Instructions should be provided for paying attention for prodromal symptoms.

3. **"I sent a patient home with the diagnosis 'syncope based on orthostatic hypotension.' After a few days the patient returned with another episode of syncope and on the monitor a dysrhythmia was seen."**
   There may be multiple causes of a syncopal episode, especially in the elderly. Even if a patient had an obvious stressor prior to the syncopal episode, or had orthostatic hypotension, other causes are still possible.

4. **"I obtained an ECG in a patient with syncope that showed a sinus rhythm with no conduction abnormalities. The patient died of a sudden cardiac arrest the next day."**
   A normal ECG has a high negative predictive value, but it does not completely rule out future cardiac events. Obtain an ECG in every patient with syncope (with, perhaps, the exception of syncope in a young person with a clearly identified trigger) to assess rhythm and conduction abnormalities. Assess for evidence of pre-excitation, prolonged corrected QT time (> 500 ms), and Brugada pattern. When checking the patient's medication list, be alert for drugs known to cause prolonged QT syndrome.

5. **"I did a complete workup in a 48-year-old patient with syncope, including ECG, laboratory tests, and a chest X-ray, before discharging him. A few hours later, he returned with a hemiparesis from a subarachnoid hemorrhage. He didn't mention he had a sharp headache just before the event."**
   The most important step in obtaining an accurate diagnosis is the history of present illness. Invest the time to get all the facts from the patient, family, and bystanders. This investment will yield more efficient ED diagnostic workup, more accurate diagnosis, and a higher quality of emergency care.

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6. "A patient came in after syncope and had another episode in the ED. I think the underlying problem might be a dysrhythmia that we didn't capture on the ECG."
Continuous ECG monitoring increases the likelihood of capturing an intermittent dysrhythmia. All patients with a possible cardiac cause of syncope should be placed on continuous ECG monitoring in the ED.

7. "I ordered a CT of the thorax because the patient complained of dyspnea and hemoptysis after syncope, and I was concerned for a pulmonary embolism. The patient became more dyspneic and tachypneic, and, while being transported to radiology, he arrested."
If you recognize a potential life-threatening cause in a patient, consider starting aggressive treatment before getting diagnostics.

8. "I discharged a patient from the ED after his first episode of syncope. He had a second episode of syncope and didn't go to see a doctor because 'it was nothing the last time.'"
Patients who suffer from syncope and are discharged from the ED should seek follow-up with their primary care physician, especially if they are at the extremes of age. It is necessary to explicitly instruct or arrange this for your patients; otherwise, they may assume it is not important. Make sure they understand the importance of seeking attention with additional symptoms or events.

9. "After a discussion with the cardiologist, I discharged the 78-year-old syncopal patient. A detailed history did not identify any new worrisome symptoms. Even though he had a coronary artery bypass graft 3 years prior, there were no abnormalities on physical examination and no ECG changes. Two days later the patient returned with a cardiac arrest."
Factors associated with higher risk for an adverse event after syncope are advanced age, cardiovascular disease, and an abnormal ECG. Patients with these and other risk factors may require admission for observation and further evaluation. Outpatient follow-up may be inadequate when the patient is risk stratified as high-risk.

10. "A 85-year-old patient was sent home after syncope based on orthostatic hypotension. A few weeks later she returned to the ED because she sustained a head injury during syncope."
Take the time to inform your patients about the possible dangers of syncope. Patients should be warned about possible trigger events for syncope, associated signs and symptoms, and the risk of a sudden attack. Particularly in the elderly, instructions should be provided for procedures to decrease the risk of falls, such as using a cane or walker, taking extra time to equilibrate when changing position, and paying attention to symptoms that may precede the syncopal attack.

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CME Questions

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1. Which of the following is unlikely to cause cardiac syncope?
   a. Prolonged QT interval
   b. Atrial fibrillation
   c. Bradycardia associated with venipuncture
   d. Tachyarrhythmias

2. Which of the following is the most discriminating between transient loss of consciousness caused by seizure and neurally mediated syncope?
   a. Jerking limb movements and confusion during and after the transient loss of consciousness
   b. Incontinency of urine and/or feces
   c. Lateral tongue biting
   d. Prodromal symptoms

3. **Which of the following historical features is most likely to be associated with a dangerous cause of syncope?**
   a. Syncope while upright
   b. Syncope during exercise
   c. Syncope during micturition
   d. Syncope while experiencing pain

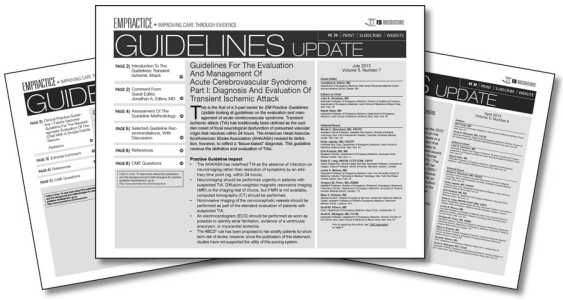
4. **A 49-year-old man presented after syncope while running on a treadmill. For the past few months he has had dyspnea on exertion and angina. Which of the following is the most likely cause of his symptoms?**
   a. Aortic stenosis
   b. Atrial septum defect
   c. Mitral valve insufficiency
   d. Pulmonary stenosis

5. **What is the only diagnostic test recommended in every patient with syncope except those with a clearly benign etiology or trigger?**
   a. Electrolytes
   b. Electrocardiogram
   c. Head CT
   d. Chest x-ray

6. **An incomplete right bundle branch block and persisting downsloping ST-segment elevation with negative T in V_1 to V_3 matches with:**
   a. Torsades de pointes
   b. Wolff-Parkinson-White syndrome
   c. Brugada syndrome
   d. Sick sinus syndrome

7. **All of the following regarding evaluation and diagnostics of syncope are true, EXCEPT:**
   a. Event recorders and Holter monitoring have a low diagnostic yield.
   b. An electroencephalogram is a good screening tool and should be performed in all syncope patients to rule out epilepsy.
   c. Patients with structural heart disease and/or ECG abnormalities often do have an abnormal electrophysiologic study.
   d. Tilt-table testing is suggested for patients with recurrent, unexplained syncope in which cardiac cause is very unlikely.

8. **In patients with syncope, prolonged ECG monitoring may be indicated if the following is present:**
   a. History of coronary artery disease and abnormal ECG
   b. Onset of symptoms after emotional event
   c. Age < 18 years
   d. Early repolarizarion



In The March/April 2014 Issue Of *EM Practice Guidelines Update*

The March/April 2014 issue of *EM Practice Guidelines Update* reviews 2 recently updated practice guidelines on the diagnosis, treatment, and prevention of *Clostridium difficile* infection (CDI) in the ED: the 2013 guideline from the American College of Gastroenterology and the 2014 guideline from the European Society of Clinical Microbiology and Infectious Diseases.

Seth Gemme, MD and Brian Clyne, MD review these guidelines for applicability in the ED and answer these questions:

- How are "mild," "moderate," "severe," and "severe-complicated" CDI defined?
- What are the most effective tests for CDI that can be used in the ED?
- Should the emergency clinician treat suspected CDI empirically while awaiting test results?
- What is the first-line medication choice for each type of CDI? Recurrent CDI?

All subscribers to *Emergency Medicine Practice* have free access to this online publication at www.ebmedicine.net/Cdiff, and each issue also includes 2 hours of CME. For more information on this publication and to see the full archive of *EM Practice Guidelines Update* issues, go to "What Is EM Practice Guidelines Update All About?" or http://www.ebmedicine.net/content.php?action=showPage&pid=94&cat_id=16.

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Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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Upon completing this article, you should be able to:

1. Describe the neurally mediated, orthostatic, and cardiovascular causes of syncope.
2. Discuss how history, physical examination, and electrocardiogram guide the diagnostic process for patients with syncope.
3. Demonstrate an evidence-based process for diagnostic strategy in the emergency department that can risk stratify for disposition of patients with syncope.

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Fax: 1-770-500-1316
5550 Triangle Parkway, Suite 150
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E-mail: **ebm@ebmedicine.net**
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