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STATEMENT OF FINANCIAL DISCLOSURE

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, Dr. Wise (editor) reports he is on the speakers bureau for the Medicines Company. Dr. Judith Toski Welsh (author), Dr. Todd Welsh (author), Dr. Emerman (peer reviewer), Ms. Coplin (executive editor), and Ms. Mark (executive editor) report no financial relationships relevant to this field of study.

AHC Media

Evaluation of Syncope

Introduction

Syncope is a sudden, transient loss of consciousness associated with loss of postural tone and subsequent spontaneous, complete recovery. Although the symptoms of syncope can appear concerning, most patients are not at risk of significant morbidity or mortality. The abrupt and dramatic nature of a syncopal event, however, frequently results in an urgent office or emergency department evaluation because it is so frightening to both victims and bystanders. Determining appropriate treatment and disposition outcomes in the most time- and cost-effective manner is an important goal.

A recent review of the National Hospital Ambulatory Medical Care Survey revealed that 1% of all emergency department visits were for syncope, and that the admission rate ranged from 27% to 35%. Among admitted patients, no additional discharge diagnosis was more common than that of "syncope and collapse."[1] Admission rates vary by practice but are not decreasing despite the low diagnostic and therapeutic yield of most hospital stays. Medicare expenditures for the Diagnosis-Related Group (DRG) of syncope in the fiscal year that ended after the second quarter of 2012 were in excess of $512 million. Ninety percent of inpatient admissions for syncope were determined to be medically unnecessary, with most errors related to inpatient rather than observation status. The diagnosis of syncope has become a target for Medicare recovery audit contractors interested in denying claims payment for physician and hospital services.[2]

Epidemiology

Syncope is a common complaint in adult patients. In the Framingham Study, adults aged 30 to 62 years at the time of enrollment were monitored for 26 years. Three percent of the men and 3.5% of the women experienced at least one episode of syncope during the study. The women tended to be younger, while more of the men were older adults at the time of onset of symptoms.[3] The true incidence of syncope is difficult to assess since many episodes go unreported, and may be characterized erroneously as falls.[4] In one observational study of medical students, 39% admitted to at least one episode of syncope in their lives, and women were more likely than men to report a fainting spell.[5] The elderly have the highest incidence of syncope, are more likely to be injured as a consequence, come to medical attention more

EXECUTIVE SUMMARY

- Obtain an ECG on all patients with transient loss of consciousness.
- The three historical factors that suggest a high-risk cardiac cause are: syncope occurred during exertion, syncope occurred during supine position, and syncope occurred suddenly and without warning (no prodrome).
- Routine laboratory testing has a low yield — but consider point-of-care testing of serum glucose when recovery of alertness is slow, hemoglobin when history suggests potential for anemia, serum electrolytes when history suggests possibility of abnormality, and pregnancy test in reproductive-age women.
- Routine CT scanning of the head is not recommended for unselected syncope patients.
- Orthostatic vital signs may be helpful as a test to reproduce symptoms when the history notes premonitory lightheadedness prior to the loss of consciousness, but variation in reproducibility makes presence or absence of vital sign changes less than perfectly reliable.

often, and are more frequently hospitalized than younger people.

Etiology and Pathophysiology

The age range of patients with syncope has a bimodal distribution with a peak in the second decade of life and then a sharp rise after the age of 70 years.[6] Most young patients are determined to have syncope of benign origin, such as a vasovagal spell. Older adults are at higher risk of syncope of cardiac origin than their younger counterparts. Syncope in the patient with heart disease is especially concerning due to the increased risk of death from any cause.[7] Carotid sinus hypersensitivity is exclusively diagnosed in older adults. Medication effects, especially those from antihypertensives and diuretics, can cause syncope in patients of all ages.[8] (*See Table 1.*)

Neurally mediated syncope (vasovagal, vasodepressor, neurogenic) is the most common cause of fainting spells among the general population. It is triggered by a noxious stimulus, and causes autonomic dysfunction resulting in increased parasympathetic tone and sympathetic outflow inhibition. Peripheral vasodilation and bradycardia lead to cerebral hypoperfusion and loss of postural tone and consciousness. Common triggers are listed in Table 2.

Carotid sinus syncope is the result of an excessively sensitive response to neck stimulation. The carotid baroreceptor reflex is triggered inappropriately, and transient cerebral hypoperfusion results. The vast majority of patients are elderly men. Triggers include tightening a tie around the neck, shaving, head turning, or wearing tight shirt collars. Brief (less than 3 seconds) periods of asystole, hypotension, or both may occur. There is no increased risk of sudden death with carotid sinus syncope, but there is an increased risk of recurrence and falls, which can be mitigated by permanent pacemaker placement in symptomatic individuals.

Syncope caused by cardiovascular disease increases the patient's risk of sudden death. In the year following an episode of cardiac syncope, the rate of death is six times higher than for non-cardiac syncope causes.[9] Individuals with heart failure and syncope have a one-year mortality of 45%.[10] Cardiac syncope is the result of inadequate cardiac output, either from mechanical or electrical (arrhythmia) causes. Ventricular tachycardia accounts for 11% of all syncope cases in adults and is the most common arrhythmia associated with the condition.[9] Poor left ventricular function is associated with ventricular tachycardia. Elderly patients are at increased risk of sick sinus syndrome and reduction in cardiac output due to bradycardia. Ventricular arrhythmias are also associated with hypertrophic cardiomyopathy in younger adults. Hypertrophic cardiomyopathy is the most common cause of sudden cardiac death in adolescents, and syncope may precede the event. Syncope with exertion is an especially significant occurrence and should prompt an appropriate evaluation.[11]

Orthostatic hypotension is the second most common cause of syncope. Volume depletion, medications, autonomic dysfunction, or a combination of factors all contribute to loss of consciousness. The American Academy of Neurology and American Autonomic Society guidelines define orthostatic hypotension as the reduction of systolic blood pressure by at least 20 mmHg or diastolic blood pressure by 10 mmHg within 3 minutes of standing.[12] Orthostatic vital signs are neither sensitive nor specific for assessing volume status or ruling out a high-risk syncope diagnosis, but are inexpensive to perform and may affect management in selected patients.[13] Orthostasis can result from volume depletion or instability of the autonomic system. Postural vital sign abnormalities may suggest the need for medication adjustments or intravenous fluids.[14]

Medications can cause syncope by causing a decrease in blood pressure, inducing an arrhythmia, or by suppressing cardiac output. Five percent to 15% of all syncopal events are believed to be related to medications.[15] Diuretics, beta blockers, calcium channel blockers,

Table 1. Common Causes of Syncope in Adults

Reflex-mediated Syncope

- Neurocardiogenic
- Situational
- Carotid sinus hypersensitivity

Cardiac

- Bradyarrhythmias
- Tachyarrhythmias
- Myocardial ischemia
- Valvular disease/insufficiency
- Cardiac tamponade
- Pacemaker dysfunction
- Hypertrophic cardiomyopathy
- Heart failure

Neurologic

- Vertebrobasilar insufficiency
- Psychiatric disease

Orthostatic Hypotension

- Dehydration
- Anemia
- Autonomic dysfunction
- Immobility
- Pregnancy

Medication/Drug-induced

- Antihypertensives
- Diuretics
- Alcohol
- Anticholinergics
- Narcotics

Metabolic Disorders

- Hypoglycemia
- Hypoxia

Hemorrhage/Anemia

Unexplained

angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs), levodopa, alpha blockers, nitrates, tricyclic antidepressants, antiarrythmics, and medications that prolong the QT interval (antiemetics and antipsychotics) have been implicated in inducing syncope. Narcotics and alcohol are also responsible for syncopal events. A recent meta-analysis of cholinesterase inhibitors used in patients with dementia showed an increase in the risk of syncope without increasing the risk of fractures, falls, or significant injury.[16]

Psychiatric causes are thought to account for about a quarter of all unexplained syncope cases.[17] Major depressive disorder, anxiety, and alcohol dependence were highly correlated with syncopal events. Vasovagal spells can be brought on by stress or fear. Episodes of hyperventilation and hypocapnia resulting in peripheral vasodilation and cerebral hypoperfusion also result in syncope. Heavy alcohol consumption results in orthostasis due to impairment of vasoconstriction.[18]

Insufficent energy substrates — a relative or absolute deficiency of oxygen or glucose — can cause loss of consciousness due to impaired brain metabolism. Hypoglycemia occurs in diabetics on oral hypoglycemic agents or insulin, or in patients with glycogen storage disease. Initial symptoms include shakiness, sweating, hunger, and dizziness and can progress to confusion or coma. Administration of glucose aborts the episode. Spontaneous recovery from hypoglycemia is sometimes seen due to the counter-regulatory hormones (epinephrine, glucagon) released in response to hypoglycemia, but the recovery of alertness is slower than typically seen in syncope. Hypoxia results from respiratory or cardiac causes or as a result of ascent to altitude. Blood loss and other causes of anemia induce anoxic syncope with exertion due to lack of oxygen-carrying capacity to the brain and vital organs.

Clinical Features

Initial evaluation of a patient with suspected syncope includes a careful history and physical exam following acute stabilization, and, along with electrocardiography, are the highest yield interventions in the diagnosis of syncope.[19] Determining that a syncopal event actually occurred and identifying high-risk features of the event are also critical tasks during the evaluation. (*See Table 3.*)

A complete physical assessment should be undertaken, with careful

attention paid to vital signs and the cardiovascular and neurologic exams. Abnormal vital signs, including elevated or low blood pressure and heart rate, need to be managed and fully evaluated. Hypoxia associated with syncope should raise concern for pulmonary embolism. Pulsus paradoxus is associated with cardiac tamponade. Systolic murmurs in a patient with syncope are worrisome for hypertrophic cardiomyopathy or aortic stenosis, both of which increase the risk of death. Lateral tongue biting suggests that the spell was a seizure rather than true syncope. Abdominal pain or tenderness should be fully evaluated and may include a rectal exam to check for an occult gastrointestinal (GI) bleed. Evidence of a head injury may result from the loss of postural tone related to syncope, but it might also indicate a traumatic loss of consciousness from a mechanical fall or assault. In the latter case, the patient may not recall the circumstances surrounding the event due to a concussion, and this could be mistaken for a lack of prodrome.

Controversy exists as to the value of carotid examination and carotid sinus massage (CSM) as part of the evaluation of syncope patients. The European Society of Cardiology guidelines recommend against carotid ultrasound due to the low yield of the test in "typical" patients, but that those with neurologic findings may benefit.[21] Syncope brought on by neck turning suggests carotid sinus hypersensitivity and is a consideration in patients aged 40 years and older. Carotid sinus hypersensitivity is diagnosed in part by carotid sinus massage. The CSM is considered positive if the test results in asystole for 3 seconds and/or a fall in systolic blood pressure of more than 50 mmHg. European Society of Cardiology guidelines recommend that CSM should not be performed in patients with a previous stroke or transient ischemic attack within the past 3 months, and in those patients with carotid bruits unless ultrasound excluded carotid stenosis (Class III, Level C evidence).[21] Positive findings on the CSM test are fairly common but do not exclude other causes of syncope.[19]

Table 2. Common Triggers of Neurocardiogenic Syncope

- Situational: fear, pain
- Micturition
- Defecation
- Coughing/sneezing
- Swallowing
- Diving

Orthostatic challenge is another physical exam maneuver that is recommended by both European and American guidelines as a best practice. Blood is displaced from the thorax to the legs with a change from the supine to an upright position, which decreases cardiac output by decreasing venous return. Without adequate compensation for the drop in venous return, blood pressure drops, resulting in syncope. European Society of Cardiology guidelines recommend measurement of the blood pressure manually while the patient is supine and after 3 minutes of standing when orthostatic hypotension is suspected (Class I, Level B evidence). The test is diagnostic when there is a symptomatic decrease in systolic blood pressure greater than 20 mmHg or a drop in diastolic blood pressure greater than 10 mmHg from baseline, or a decline in systolic blood pressure to less than 90 mmHg (Class I, Level C evidence).[21] Orthostatic hypotension can exist along with a serious arrhythmia, however, so this finding does not exclude another diagnosis.

Diagnostic Studies

Much of the routine testing that is performed on syncope patients is unnecessary. Electrocardiography (ECG), telemetry, cardiac enzymes, and computed tomography (CT) are the most frequently obtained tests.[14] Of these, only ECG is routinely recommended. Electrocardiography is a critical part of every assessment of syncope in adults, and is a level A recommendation in the American College of Emergency Physicians (ACEP) guidelines.[22] Cardiac syncope is diagnosed in only 3% of patients without a known history of heart disease.[23] Abnormalities suggestive of arrhythmogenic syncope include:

- sinus bradycardia or pauses;
- bi- or trifascicular block;
- Mobitz I second-degree or third-degree atrioventricular block;
- prolonged or shortened QT interval;
- ventricular hypertrophy;
- ST elevation or depression, Q waves (acute or chronic ischemic changes);
- ventricular tachycardia or rapid paroxysmal supraventricular tachycardia (SVT);
- Brugada pattern;
- evidence of arrhythmogenic right ventricular dysplasia/early repolarization.[24]

Patients with these ECG features and syncope need an urgent consultation with a cardiologist. Electrophysiologic testing is recommended in patients with syncope associated with impaired left ventricular function or known structural heart disease, but is not routinely recommended. Continuous cardiac monitoring can detect arrhythmia missed on ECG, and either ambulatory or inpatient

monitoring may be indicated if an abnormal heart rhythm is suspected, and 24 hours is usually enough to accomplish this task. Extensive monitoring (up to 72 hours) is not associated with an increased yield of an abnormal finding in most patients.[22]

Blood tests have limited value in uncomplicated syncope. The complete blood count may have some utility in patients because anemia, a hematocrit less than 30%, is associated with an increased risk for adverse outcomes (see below).[25] The yield of routine blood tests in syncopal patients is estimated at 2-3%. Cardiac markers are often obtained in patients with syncope in the ED.[26] A prospective observational study of high-sensitivity cardiac troponin T measurement failed to demonstrate a relationship between increased troponin T levels and cardiac causes of syncope, and that elevated troponin levels did not increase the risk of an adverse outcome within 180 days of follow up.[27]

In a prospective, observational study of echocardiography in a population of patients with syncope, clinically important findings were present only in those with suspected aortic stenosis, a history of heart disease, or an abnormal ECG.[28] In a prospective study of the use of routine echocardiography in patients with an uncertain diagnosis of syncope, only 15% had clinically significant findings.

Brain imaging is performed in up to 50% of patients presenting with syncope to the ED. Head CT is not recommended as a routine study in uncomplicated syncope due to its low diagnostic yield; only 5% of patients with transient loss of consciousness (LOC) had positive findings in the largest study performed on the subject.[29] Limiting testing to those patients with a focal neurologic deficit, age older than 60 years, taking anticoagulants, or history/physical exam findings consistent

Table 3. Historical Details Important in the Evaluation of Syncope

History	Important Features
Activity at the time of onset, precipitating factors	Exertion at the time of onset suggests cardiac disease.
Aura or warning symptoms	Absence of prodrome is high risk for arrhythmia.
Loss of consciousness (LOC)	Long duration of LOC is concerning for alternative diagnosis.
Witnessed abnormal motor activity	Myoclonic jerks can occur with syncope, but prolonged abnormal movements suggest an alternative diagnosis such as seizure.
Pain before or after event	Chest pain before the event is concerning for cardiac etiology. Injuries can occur related to falls or LOC during driving or other activity. Abdominal pain may indicate internal hemorrhage from a ruptured AAA or ectopic pregnancy, sepsis from an intestinal perforation or infectious process, or a benign but painful condition causing increased vagal tone. Headaches could be from an intracranial hemorrhage.
Incontinence, tongue biting	Can occur during syncope, but far more common during seizure.
Neurologic dysfunction following episode	Focal weakness, confusion, coma more likely to be related to seizure or CVA.
Duration of event	Syncopal events are generally brief (less than 1 minute). Seizures and the subsequent postictal period are often longer.
Medication	Careful review of the patient's medications may reveal potential precipitants.
Drug or alcohol use	Rule out intoxication.
Past medical history and history of other similar episodes	Seizures and syncope both can be recurrent events. History of heart disease suggests cardiac syncope.
Family history	Sudden cardiac death episodes in family members are high risk for Brugada syndrome, long QT syndrome, or cardiomyopathy.[20]
Any recent changes in stool color, consistency, or frequency	Bloody or black/tarry stools may indicate acute blood loss anemia from GI bleeding. Diarrhea could result in hypovolemia.

Table 4. Factors Differentiating a Seizure from Syncope

Characteristic	Syncope	Seizure
Preceding symptoms	Nausea, vomiting, feeling cold, sweating, blurred vision, lightheadedness	Aura
During the episode	Very brief if any tonic-clonic movements and they start after the loss of consciousness	Prolonged tonic-clonic movements coinciding with the loss of consciousness. Chewing, lip smacking, frothing, tongue biting, incontinence
Post event	Generally rapid and full recovery	Prolonged confusion Muscle pains Headache Feeling sleepy

with acute head trauma would reduce the number of scans by 24%. There is, however, no prospectively derived clinical decision rule in place to help guide the decision to obtain CT in syncope patients.

Carotid duplex ultrasound (CDUS) is another test that is frequently ordered to evaluate patients with syncope and which is not recommended in uncomplicated cases. American and European guidelines recommend that CDUS be performed only in patients with history suggestive of stroke or transient ischemic attack (TIA), or presenting with focal neurological deficits or carotid bruits on examination.[21]

Differential Diagnosis

Transient loss of consciousness (T-LOC) occurs in many different disease processes, and differentiating syncope from other causes can be difficult. The European Society of Cardiology guidelines divide T-LOC into traumatic and non-traumatic forms. T-LOC related to trauma is usually the result of concussion. Non-traumatic T-LOC is more challenging to differentiate. Asking the following questions can help determine whether the cause of the T-LOC episode was syncope:

- Was there complete loss of consciousness?
- Did it occur before the fall and not as a result of it?
- Was the episode transient, rapid in onset, and short in duration?
- Did the patient recover without need for intervention, and come back completely to baseline?
- Was there loss of postural tone?

If all the questions have a "yes" answer, then syncope is the most likely cause of the episode. With one or more "no" answers, undertake a search for another cause.[30] Other non-traumatic disorders that can be misdiagnosed as syncope include:

- seizure (*see Table 4*);
- intoxication;
- transient ischemic attack/cerebrovascular accident;
- metabolic disorders: hypoglycemia, hypoxia, hyperventilation with hypocapnia;
- cataplexy;
- drop attacks;
- pseudosyncope/psychogenic causes.

In addition, true syncope can be associated with significant underlying disorders, many of which are life-threatening. Volume depletion can be related to medications, GI losses, or inadequate intake. Pulmonary embolism is a rare but serious cause of syncope that should be considered in patients with hypoxia, tachycardia, unexplained shortness of breath, pleuritic chest pain, or hemoptysis.[31] Blood loss can also result in syncope. Important sources of hemorrhage that may not be clinically obvious include:

- acute or chronic GI bleeding;
- aortic aneurysm rupture;
- ectopic pregnancy;
- ruptured ovarian cyst;
- ruptured spleen;
- occult trauma;
- retroperitoneal hemorrhage.

Management and Disposition

The initial goal of management of a patient with syncope is stabilization. Most patients with true syncope will be back to baseline without intervention and will present in stable condition. Unstable patients present with chest pain related to ischemia, hypotension, or an abnormal heart rhythm. Patients with an arrhythmogenic cause of syncope may require an emergent intervention with medication, cardioversion, or defibrillation. Patients with acute symptomatic hemorrhage will require detection of the site of bleeding and appropriate care of their underlying disease process. Myocardial ischemia is a relatively rare cause of syncope, present in 0.2%-4% of patients.[32] Patients older than 75 years with myocardial ischemia or infarction

Table 5. San Francisco Syncope Rule — High-risk Criteria "CHESS" Mnemonic

C: Congestive heart failure history
H: Hematocrit < 30%
E: ECG abnormalities (new changes on ECG or nonsinus rhythm)
S: Shortness of breath
S: Systolic blood pressure < 90 mmHg at triage

are more likely to present with "atypical" symptoms like faintness or a fall. Obtaining an ECG rapidly in these patients is critical to avoid missing the diagnosis.[33]

Risk assessment is a secondary goal following stabilization, and is the key to appropriate testing, treatment, and disposition for the vast majority of patients. Several clinical decision tools have been developed and evaluated to aid in risk assessment and to guide further management and the decision to admit. The intent of clinical decision rule implementation is to ensure that all patients with high-risk diagnoses are identified and appropriately managed, while avoiding inappropriate interventions and admissions in patients who are predicted to have a benign course. Another goal of protocol implementation is to reduce practice variation, which results in increased cost of care.

Controversy exists regarding use of risk-stratification tools and their value in practice compared to clinical judgment. Two commonly referenced clinical decision scores are the San Francisco Syncope Rule (SFSR) and Osservatorio Epidemiologico sulla Sincope nel Lazio (OESIL) risk score. The ability of the rules to predict their specified outcomes was variable, and there is currently no single decision tool recommended for use by either the American or European guidelines.

In 2010, Serrano et al performed a meta-analysis of 18 studies and found that methodological flaws in the studies and their validation limited their utility. The authors concluded that further refinement of the clinical decision scores was needed before they could be part of routine practice.[34] Another meta-analysis of five different tools published in 2014 confirmed this assessment, and determined that the clinical decision rules did not predict adverse outcome or reduce unnecessary admissions any better than the application of clinical judgment alone.[35] A 2011 meta-analysis to assess the San Francisco Syncope Rule (*see Table 5*), the most-investigated clinical decision rule for syncope, also noted the inconsistent results of the validation trials. The authors concluded that the SFSR should be used only in patients without an evident cause of syncope following evaluation, and that if the patient fails to meet any high-risk criteria, their risk of a serious outcome is 2% or less.[36]

Although no one risk stratification tool has been shown to be superior to clinical judgment, emergency clinicians need to be aware of factors that suggest either a benign course or a more serious danger to the patient. Younger age (younger than 40 years) is usually associated with a better outcome. Patients with the same symptoms occurring over many years also are more likely to be suffering from reflex syncope. Patients with significantly abnormal vital signs (systolic blood pressure less than 90 mmHg or sinus bradycardia less than 40 bpm) are at higher risk of an adverse outcome. Important historical features of low- and high-risk syncope are included in Table 6.[22]

The decision to admit a patient or to refer for outpatient management can be challenging to make. ACEP guidelines recommend that patients with syncope and evidence of heart failure or structural heart disease should be admitted, as well as those "at high risk for adverse outcome" (Level B recommendation).[37] Few admissions, even of patients with high-risk characteristics, yield much in the way of outcome. A current research initiative is underway in several centers to assess the use of dedicated syncope observation protocols and centers dedicated to rapid, cost-saving care.

A prospective, randomized controlled trial of syncope unit admission for patients of intermediate risk determined that with a multidisciplinary approach, diagnostic yield of testing can be improved and hospital admission rates and length of stay can be reduced without increasing mortality. Intermediate risk was defined as: previous history of cardiac disease (MI, congestive heart failure, cardiomyopathy, or cardiac device) without acute decompensation or active symptoms, family history of premature sudden death, bundle branch block or Q waves without acute ischemic changes on ECG, symptoms not consistent with vasovagal cause, or physician concern of possible cardiac syncope.[38]

A 2014 randomized controlled trial comparing routine inpatient admission with ED observation protocol similarly found that intermediate-risk patients with syncope left the hospital 18 hours earlier than admitted patients, saving costs of $629 per visit. There was no significant difference in safety events (death, pulmonary embolism, MI, stroke, fall/trauma following index visit, or need for acute cardiac intervention), patient ratings of care, or quality-of-life scores between the treatment arms. Protocol-driven testing included at least two

Table 6. Important Historical Features of Low- and High-risk Syncope[22]

Low-risk History	High-risk History
Occurs only while standing	Occurs in supine position
Occurs following position change from supine or sitting to standing	Present during exertion
Nausea or vomiting prior to episode	Chest discomfort associated with spell
Feeling warm or "light-headed" before episode	Present with palpitations
Triggered by pain or fear	Absence of a prodrome (occurred suddenly and without warning)
Triggered by coughing/voiding/defecating	

troponin tests to exclude acute MI, an echocardiogram if a murmur was heard on exam, and any additional testing considered to be necessary by the treating physician.[39]

Cost savings were estimated to be $108 million (± $89 million) in a simulation in which all intermediate-risk patients (age older than 50 years) were admitted to an observation unit utilizing a syncope protocol instead of an inpatient ward.[40] The simulation assumed a reduction of 235,000 (± 13,900) admissions on an annual basis. Reduction of low-value resource utilization and waste is a significant goal of health care systems in the United States. Although patients admitted to a dedicated syncope unit were more likely to receive a definitive diagnosis at discharge, readmission rates are high for syncope patients regardless of the site of initial admission.[41]

Patient Concerns

A major concern of patients with syncope is whether or not they may continue to drive, and the risk of recurrence of syncope. Most patients do not want to lose the independence associated with driving, but public safety concerns need to be addressed before allowing a patient with syncope to undertake potentially injurious activity. The most common type of syncope is neurally mediated syncope, and this is also the most common cause of syncope while driving.[42] The Canadian Medical Association, American Heart Association, and Heart Rhythm Society have made recommendations regarding restrictions on private and commercial driving for patients with neurally mediated syncope and with specific arrhythmias. There are differences between the guidelines in terms of timeframes, but in general, mild neurally mediated syncope does not lead to driving restriction for private (non-commercial) driving. Episodes in patients with syncope during driving, a documented symptomatic arrhythmia, structural heart disease, or in a professional driver require driving restrictions pending further assessment or effective treatment.[43]

Recurrence of syncope is common, and even relatively benign events like vasovagal syncope can cause difficulty with functional status and psychological functioning.[44] A recent meta-analysis of prospective observational studies demonstrated a very low rate of mortality. In the first 30 days post-event, the rate of death from all causes was 1.6%, while the chance of death or a "major event" like stroke or MI was about 9%. There is a linear increase in the recurrence of syncope, from 0.3% at 30 days to 22% at 2 years.[45]

Conclusion

Syncope is a common, potentially high-risk complaint among patients. It occurs as a result of transient, spontaneously reversible cerebral hypoperfusion events causing loss of postural tone. There are many syncope mimics that must be differentiated from true syncope to establish the diagnosis. Most patients with syncope will not suffer an untoward outcome, but appropriate detection of patients at risk of death or disability due to an undiagnosed underlying disease process is an important task for the emergency clinician. There are multiple guidelines available through American and European medical societies, but no single clinical decision rule has been proven to be superior to a clinician's own judgment in identifying high-risk patients. ED observation protocols have been shown to reduce the cost and to increase the diagnostic yield of testing in those patients who fall into the intermediate-risk group.

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CME Questions

1. Which of the following tests should be performed routinely on all adults presenting with syncope?
   A. echocardiography
   B. CT brain
   C. electrocardiography
   D. serum electrolytes

2. Which of the following patients is at low risk of cardiac syncope?
   A. a 20-year-old man who collapsed while running a marathon but is back to baseline at triage
   B. a 66-year-old man with a history of an MI 20 years ago
   C. a 24-year-old woman who became dizzy and fainted while having her blood drawn in the hospital laboratory; she has had episodes like this in the past
   D. a 78-year-old woman who passed out at church; her ECG shows a right bundle branch block, and the ED tech cannot find a comparison ECG

3. Which of the following accurately describes syncope related to carotid sinus hypersensitivity?
   A. It is a common diagnosis in adolescents and young adults.
   B. It occurs as a result of defecation or micturition.
   C. Carotid sinus hypersensitivity is a high-risk cardiac diagnosis that is routinely managed with cardiac pacing.
   D. Shaving, turning the neck, or tightening a tie precipitates symptoms.

4. Which of the following classes of medication is most commonly associated with syncope in adult patients?
   A. diuretics
   B. beta-lactam antibiotics
   C. serotonin reuptake inhibitors
   D. coenzyme Q supplements

5. Which of the following statements is true regarding clinical decision rules (CDR) and syncope?
   A. American and European guidelines recommend the application of different CDRs.
   B. There is no single CDR that has been shown to be superior to clinician judgment.
   C. The San Francisco Syncope Rule recommends an orthostatic challenge as part of the assessment.
   D. Hematocrit > 30% is associated with increased clinical risk.

6. Which of the following ECG abnormalities is associated with a low risk of cardiac syncope?
   A. Mobitz type I atrioventricular block
   B. bifascicular block
   C. shortened QT interval
   D. sinus bradycardia (rate = 39 bpm)

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7. Which of the following etiologies of syncope is the most common diagnosis in the ED?
   A. orthostatic hypotension
   B. cardiac
   C. vasovagal
   D. carotid sinus hypersensitivity

8. Which of the following arrhythmias is most commonly associated with cardiac syncope?
   A. ventricular fibrillation
   B. atrial fibrillation
   C. ventricular tachycardia
   D. paroxysmal atrial tachycardia

9. Which of the following patients with a normal ECG and unremarkable physical examination may continue to drive immediately following a syncopal episode?
   A. a 19-year-old woman who had a syncopal event while running track
   B. a 24-year-old man who fainted during a dermatologic procedure in the office
   C. a 65-year-old woman with a history of coronary artery disease and smoking who drives a tractor-trailer for a living
   D. a 73-year-old man with a history of aortic stenosis

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