

Evaluation of Syncope in the Emergency Department

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Introduction

Syncope is a symptom complex composed of a transient loss of consciousness associated with an inability to maintain postural tone, secondary to a brief decrease in cerebral blood flow that spontaneously and completely resolves and that requires no resuscitation.¹ Accounting for 3% of emergency department (ED) visits and 1% to 6% of all hospital admissions,² syncope presents a challenge to emergency practitioners: to differentiate those patients safe for discharge from those who require emergent evaluation and in-hospital management for potentially life-threatening etiologies. The precise cause of syncope can be identified during the initial evaluation in only 20% to 50% of patients.³ Of note, it is estimated that up to 80% of the causes of syncope that *are* identified during a hospital admission are determined in the emergency department.⁴

While most potential causes of syncope are benign and self-limited, some etiologies are associated with significant morbidity and mortality. Approximately 4% of patients discharged from the ED with syncope return within 72 hours and are admitted or die.⁵

Cardiac arrhythmias and sudden death are the chief short-term complications to be avoided in syncope. In one population-based study, patients with cardiac causes of syncope had double the mortality rate of patients without syncope. The average cost of care per hospital admission for syncope is approximately \$5,000, and more than \$2 billion a year is spent in the United States on such hospitalizations.⁶ The emphasis in the evaluation of the patient who presents to the ED with syncope is on risk stratification and on doing so in an expeditious, cost-effective manner, and in a medico-legally defensible manner. This article will attempt to simplify the clinical approach to the patient with syncope based upon the current literature.

Differential Diagnosis

The differential diagnosis of syncope is extensive (Table 1). In addition, other syncope-like conditions, such as seizure, stroke, and head injury, should be considered during the initial evaluation of a patient with transient loss of consciousness. Seizures may be difficult to distinguish from syncope. Seizure is suggested by: a history of seizure disorder, an abrupt onset associated with head injury, tongue biting (particularly involving the lateral aspect of the tongue), the presence of a tonic phase preceding the onset of clonic activity, unusual posturing or head deviation, loss of bladder or bowel control, age less than 45 years, medication noncompliance, a preceding aura, and prolonged confusion and disorientation after the event.⁷

In contrast to seizure, syncope is often preceded by sweating or nausea and by sitting or standing and has rapid return of orientation upon awakening. Syncope more often occurs in patients older than 45 years, and it is associated with a history of congestive heart failure (CHF) and coronary artery disease (CAD).

Life-threatening causes of syncope include cardiovascular causes, hemorrhage, and subarachnoid hemorrhage (SAH). A “rule of 15s” for syncope reminds us that approximately 15% of the following life-threatening conditions present with syncope: SAH, acute coronary syndrome (ACS), aortic dissection, leaking aortic aneurysm (AAA), and ruptured ectopic pregnancy.⁴

Many of the missed diagnoses of these five conditions that resulted in medico-legal action involved presentations that included syncope. The physician evaluating a patient with brief loss of consciousness should be vigilant for the possibility of carbon monoxide toxicity, SAH, carotid dissection, vertebrobasilar transient ischemic attack, leaking abdominal aortic aneurysm, gastrointestinal hemorrhage, and ruptured ectopic pregnancy.

Table 1: Differential Diagnosis of Syncope

NEURALLY-MEDIATED (REFLEX)	CARDIOGENIC
Carotid sinus hypersensitivity <ul style="list-style-type: none"> • Head turning • Circumferential neck compression (neck tie) • Shaving 	Cardiac arrhythmia <ul style="list-style-type: none"> • Amiodarone toxicity • Atrial fibrillation with Wolff-Parkinson-White syndrome • Atrial flutter • Atrial surgery • AV block • AV canal defects • AV conduction system disease • Sinus node dysfunction • Supraventricular tachycardia • Ventricular tachycardia • Pacemaker or automated internal cardiac defibrillator dysfunction • Brugada syndrome • Catecholaminergic tachycardia • Long QT syndrome
Glossopharyngeal neuralgia	Structural cardiac obstructive lesions <ul style="list-style-type: none"> • Acute coronary syndrome • Aortic valve stenosis • Atrial myxomas • Hypertrophic cardiomyopathy <ul style="list-style-type: none"> ■ Cardiac tamponade ■ Aortic dissection
Idiopathic postural hypotension	Significant hemorrhage <ul style="list-style-type: none"> • Trauma with significant blood loss • Gastrointestinal bleeding • Tissue rupture <ul style="list-style-type: none"> ■ Aortic aneurysm ■ Spleen ■ Ovarian cyst ■ Ectopic pregnancy ■ Retroperitoneal hemorrhage
Peripheral neuropathy <ul style="list-style-type: none"> • Alcoholic • Amyloid deposition • Diabetes • Malnutrition 	Pulmonary embolism <ul style="list-style-type: none"> • Saddle embolus resulting in outflow tract obstruction or severe hypoxia
Situational <ul style="list-style-type: none"> • Cough • Swallow, defecation • Micturition • Post-exercise • Post-prandial • Others (e.g., brass instrument-playing, weightlifting) 	Subarachnoid hemorrhage
Vasovagal (common faint)	Cerebrovascular <ul style="list-style-type: none"> • Vascular steal syndromes
MEDICATION-RELATED	Orthostatic hypotension <ul style="list-style-type: none"> • Drug side effects • Dysautonomias <ul style="list-style-type: none"> ■ Multiple system atrophy ■ Parkinson's disease ■ Postural orthostatic tachycardia syndrome ■ Pure autonomic failure <ul style="list-style-type: none"> • Shy-Drager syndrome • Volume loss • Autonomic dysfunction • Deconditioning, prolonged bed rest
Vasoactive medications <ul style="list-style-type: none"> • Alpha and beta blockers • Calcium channel blockers • Nitrates • Antihypertensive medications • Diuretics • Erectile dysfunction medications 	
Medications affecting conduction <ul style="list-style-type: none"> • Antiarrhythmics • Calcium channel blockers • Beta blockers • Digoxin 	
Medications affecting the QT interval <ul style="list-style-type: none"> • Antiarrhythmics • Antiemetics • Antipsychotics/depressants 	

Cardiovascular causes are the most common life-threatening conditions associated with syncope, and these can be divided into arrhythmogenic, structural, and ischemic.⁸ Syncope from a sudden disruption in cardiac output is the most deadly form of syncope. Arrhythmogenic causes of syncope can include ventricular tachycardia, long QT syndrome, Brugada syndrome, bradycardia (e.g., Mobitz type II or 3rd degree heart block), and significant sinus pauses (i.e., >3 seconds). Lyme disease is a cause of conduction defects that cause bradydysrhythmia and that present as syncope. Ischemia includes acute myocardial infarction and coronary syndromes. Among structural abnormalities are: valvular heart disease, such as aortic or mitral stenosis, cardiomyopathy (e.g., ischemic, dilated, hypertrophic), aortic dissection, atrial myxoma, and cardiac tamponade.

Non-life-threatening causes of syncope include neurocardiogenic syncope, carotid sinus hypersensitivity, orthostatic syncope, and medication-related syncope. Neurocardiogenic syncope,

also known as neurally-mediated, vasovagal, and vasodepressor syncope, is a reflex-mediated bradycardia and hypotension that leads to a brief decrease in cerebral perfusion. Such episodes usually last less than 30 seconds and may be accompanied by tonic-clonic movements, known as brainstem release phenomena, or myoclonus. In contrast to seizures, sphincter control is maintained in vasodepressor syncope. Neurocardiogenic causes of syncope include micturition and defecation, cough, swallowing, glossopharyngeal nerve, pain, heat, breath-holding, and situ-

ational. These events are due either to increased vagal tone or to inappropriately decreased sympathetic tone.

Medication effects are contributory in 5% to 15% of events, and many common medications can contribute to syncope. These include: alpha and beta blockers, antiarrhythmics, antihypertensive medications, antiemetics, antipsychotics, antidepressants, calcium channel blockers, digoxin, diuretics, erectile dysfunction medications, nitrates, medications affecting conduction and those prolonging the QT interval (Table 2).⁹

QT prolongation is also associated with hypokalemia, hypomagnesemia, hypocalcemia, elevated intracranial pressure, ACS, hypothermia, and hereditary causes. Alcohol is another substance that frequently contributes to syncope. It will be noted that many patients with syncope are taking several classes of these medications at the same time.

Carotid sinus hypersensitivity is typically seen in men older than 40 years and leads to syncope associated with head turning, neck compression, and shaving.

Orthostasis may be responsible for up to one-quarter of the episodes seen in the ED, and it is due to circulating blood volume loss, autonomic dysfunction, deconditioning, and prolonged bed rest. Peripheral autonomic neural dysfunction is seen in elderly patients and in patients with Parkinson's disease, diabetes, multiple sclerosis, and spinal cord injury. The Shy-Drager syndrome is a rare disorder causing recurrent syncope secondary to damage in the autonomic nervous system.

History

Historical features to be elicited in patients with syncope are age, associated symptoms and triggers, position at the time of syncope, onset and duration, exertion as a precursor, presence of seizure activity, medications, prior episodes, family history, and associated injury. Patients and their families will often use vernacular to describe syncope, such as "passed out," "fell out," or "blacked out."

It has been observed that the risk of adverse outcomes after syncope is directly correlated with age.¹⁰ Although risk-stratification schema have used various specific age cut-offs to define a high risk group, age is optimally interpreted within the context of other independent risk factors, such as structural heart disease, in order to define risk. Up to 20% of syncope in older adults is related to cardiac arrhythmia.

Associated symptoms at the time of syncope should direct further investigations. Chest pain suggests ACS or PE, while headache or specific weakness implies a neurological cause of syncope. Acute shortness of breath or leg pain would prompt an evaluation for PE. Headache might suggest SAH or carbon monoxide exposure, while menstrual irregularity or vaginal bleeding might lead to a workup for ectopic pregnancy. Flank or abdominal pain with syncope suggests leaking AAA.

A history of a strong emotional or situational trigger suggests neurocardiogenic causes. Physical or emotional distress, cough,

Table 2: Partial List of Drugs that Prolong the QT syndrome

Generic Name	Brand Name	Class/Clinical Use
Amiodarone	Cordarone®	Anti-arrhythmic / abnormal heart rhythm
Amiodarone	Pacerone®	Anti-arrhythmic / abnormal heart rhythm
Arsenic trioxide	Trisenox®	Anti-cancer / leukemia
Astemizole	Hismanal®	Antihistamine / allergic rhinitis
Bepidil	Vasacor®	Anti-anginal / heart pain
Chloroquine	Aralen®	Anti-malarial / malaria infection
Chlorpromazine	Thorazine®	Anti-psychotic/ anti-emetic / schizophrenia/ nausea
Cisapride	Propulsid®	GI stimulant / heartburn
Clarithromycin	Biaxin®	Antibiotic / bacterial infection
Disopyramide	Norpace®	Anti-arrhythmic / abnormal heart rhythm
Dofetilide	Tikosyn®	Anti-arrhythmic / abnormal heart rhythm
Domperidone	Motilium®	Anti-nausea / nausea
Droperidol	Inapsine®	Sedative; anti-nausea / anesthesia adjunct, nausea
Erythromycin	Erythrocin®	Antibiotic; GI stimulant / bacterial infection; increase GI motility
Erythromycin	E.E.S.®	Antibiotic; GI stimulant / bacterial infection; increase GI motility
Halofantrine	Halfan®	Anti-malarial / malaria infection
Haloperidol	Haldol®	Anti-psychotic / schizophrenia, agitation
Ibutilide	Corvert®	Anti-arrhythmic / abnormal heart rhythm
Levomethadyl	Orlaam®	Opiate agonist / pain control, narcotic dependence
Mesoridazine	Serentil®	Anti-psychotic / schizophrenia
Methadone	Dolophine®	Opiate agonist / pain control, narcotic dependence
Methadone	Methadose®	Opiate agonist / pain control, narcotic dependence
Pentamidine	Pentam®	Anti-infective / pneumocystis pneumonia
Pentamidine	NebuPent®	Anti-infective / pneumocystis pneumonia
Pimozide	Orap®	Anti-psychotic / Tourette's tics
Procainamide	Pronestyl®	Anti-arrhythmic / abnormal heart rhythm
Procainamide	Procan®	Anti-arrhythmic / abnormal heart rhythm
Quinidine	Cardioquin®	Anti-arrhythmic / abnormal heart rhythm
Quinidine	Quinaglute®	Anti-arrhythmic / abnormal heart rhythm
Sotalol	Betapace®	Anti-arrhythmic / abnormal heart rhythm
Thioridazine	Mellaril®	Anti-psychotic / schizophrenia

Source: www.QTdrugs.org

micturition, defecation, shaving, or standing for a prolonged period at the time increases the likelihood of a benign cause of syncope. A prodrome, consisting of nausea and vomiting, warmth, diaphoresis, and pallor, often precedes neurocardiogenic syncope.

In adolescents a history should be sought for eating disorders, diuretic or laxative abuse, and inhalant abuse. In older patients, a history of Parkinson's disease, multiple sclerosis, and other degenerative conditions should be elicited.

Patient position at the time of syncope is important. Syncope while supine suggests an arrhythmia, while syncope after prolonged standing may reflect a neurocardiogenic cause. Orthostatic syncope follows standing up from a supine or sitting position and is often of benign etiology. A sudden and unexpected onset of syncope without prodromal symptoms implies a more serious cause, such as arrhythmia, while a gradual onset preceded by prodromal symptoms is usually associated with more benign etiologies. The duration of syncope is usually brief, often lasting less than a minute or two. When a syncope-like event persists for more than a few minutes, other conditions, such as seizure, should be considered. It has been estimated that 5% to 15% of patients thought to have syncope have a seizure disorder.⁷ Exertional syncope raises concerns about dysrhythmias and structural heart disease, including outflow obstruction and cardiomyopathy.

A complete list of the patient's medications, especially newly prescribed ones, should be obtained. Particularly important are nitrates, calcium channel and beta blockers, antidysrhythmics, and medications known to prolong the QT interval (Table 2). A family history of sudden death, especially in relatives younger than 45 to 50 years, suggests cardiac syncope, such as the Brugada syndrome. This is a syndrome of sudden death associated with one of several ECG patterns characterized by incomplete right bundle branch block and ST elevations in the anterior precordial leads.

Syncope in patients with a history of congestive heart failure (CHF) has been shown to carry a poor prognosis, even when the event itself was from a benign cause, such as neurally-mediated syncope.¹¹

Physical Examination

Physical examination should begin with a complete set of vital signs, although these may have normalized by the time of evaluation. Hypoxemia suggests possible CHF or PE. Pulse deficits and discrepancies of pulses and blood pressures between extremities suggest aortic dissection or subclavian steal syndrome.

Orthostatic blood pressure measurement consists of pulse and blood pressure after five minutes in a supine position, followed by repeat measurements after standing for three to five minutes. A positive result for orthostatic hypotension is defined as a drop in systolic blood pressure of 20 mmHg, a pulse increase of 20

beats per minute or more, or recurrent syncope. This test is neither sensitive nor specific, but a drop in blood pressure below 90 mmHg associated with symptoms can be diagnostic.

Skin and eye examination might show pallor suggestive of anemia and blood loss. The EP should consider potential sources of hemorrhage, including ruptured AAA, ruptured ectopic pregnancy, ruptured ovarian cyst, and ruptured spleen. Intraoral examination will detect evidence of tongue biting to suggest seizure activity. It may also reveal evidence of dehydration. The neurologic examination in syncope is, by definition, normal. Any residual deficit after a syncope-like event should suggest an acute stroke or structural lesion or a profound toxic or metabolic insult. The lung examination should seek evidence of CHF or focal pulmonary signs suggesting PE. Cardiac examination focuses on gallop rhythms, dysrhythmias, and murmurs. The neck examination identifies transmitted cardiac murmurs and carotid stenoses as well as thyroid enlargement. The detection of a grade III/IV mid-systolic murmur radiating to the neck and loss of S2 splitting is suggestive of critical aortic stenosis. A murmur that gains intensity with Valsalva maneuvers and abolishes with squatting suggests hypertrophic cardiomyopathy. An extra heart sound, either an S3 or S4, may be identified in CHF.

Abdominal examination may reveal a pulsatile mass in ruptured abdominal aortic aneurysm. A rectal examination can identify gross or occult fecal blood.

A thorough head-to-toe examination is essential to detect trauma resulting from a fall. Particular emphasis is placed on the examination of the scalp for lacerations or hematomas, on the face for fractures, on the neck for evidence of trauma, and on the extremities for fractures or dislocations.

Laboratory Examination

The electrocardiogram (ECG) is recommended in the evaluation of most cases of syncope.¹² The American College of Emergency Physicians clinical policy on syncope strongly recommends that an ECG be obtained in the initial evaluation of patients with syncope (Figure 1). It is rapid and inexpensive, and it may identify the etiology of syncope in up to 7% of cases. The ECG may reveal evidence of cardiac ischemia or arrhythmia as the cause of syncope. Myocardial infarction (MI) occurs in up to 3% of syncope patients, and a normal ECG has a negative predictive value for MI as the cause for the syncope of greater than 99%.⁸

ECG evidence of right heart strain may be suggestive of PE. Patients with an ECG that shows sinus rhythm with no new abnormal morphologic changes compared to prior ECGs have been found to be at low risk of adverse events during short-term follow up.¹³ In contrast, the presence of an abnormal ECG (defined as any abnormality of rhythm or conduction, ventricular hypertrophy, or evidence of previous myocardial infarction but excluding nonspecific ST-segment and T-wave changes) has been found to be a predictor for arrhythmia or death within one

Figure 1: ACEPs Clinical Policy on Syncope**A. Critical Questions:****1. What history and physical examination data help to risk-stratify patients with syncope?****Level A recommendations:**

- Use history or physical examination findings consistent with heart failure to help identify patients at higher risk of an adverse outcome.

Level B recommendations:

- Consider older age, structural heart disease, or a history of coronary artery disease as risk factor for adverse outcome.
- Consider younger patients with syncope that is nonexertional, without history or signs of cardiovascular disease, a family history of sudden death, and without co-morbidities to be at low risk of adverse events.

2. What diagnostic testing data help to risk-stratify patients with syncope?**Level A recommendations:**

- Obtain a standard 12-lead ECG in patients with syncope.

Level B recommendations:

- None specified.

Level C recommendations:

- Laboratory testing and advanced investigative testing, such as echocardiography or cranial CT scanning, need not be routinely performed unless guided by specific findings in the history or physical examination.

3. Who should be admitted after an episode of syncope of unclear cause?**Level A recommendations**

- None specified.

Level B recommendations

- Admit patients with syncope and evidence of heart failure or structural heart disease.
- Admit patients with syncope and other factors that lead to stratification as high risk for adverse outcome.

Level C recommendations

- None specified.

B. Factors that lead to stratification as high-risk for adverse outcome:

- Older age and associated co-morbidities*
- Abnormal ECG†
- Hct <30 (if obtained)
- History or presence of heart failure, coronary artery disease, or structural heart disease

*Different studies use different ages as threshold for decision making. Age is likely a continuous variable that reflects the cardiovascular health of the individual rather than an arbitrary value.

†ECG abnormalities, including acute ischemia, dysrhythmias, or significant conduction abnormalities.

From: Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department with Syncope. *Annals of Emergency Medicine*. 2007;49(4):431-7.

From: American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Syncope. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with syncope. *Ann Emerg Med*. 2007;49:431-444.

For a complete discussion of the evidence for these recommendations and for definitions of terms, see the full clinical policy, available online at: <http://www.acep.org/practres.aspx?id=30060/>.

year after the syncopal episode. The one-year mortality of patients with cardiac syncope approaches 30%, and in those with CHF mortality is even higher.¹⁴

Significant ECG findings include: evidence of ACS, severe bradycardia, prolonged intervals (QRS, QTc), ventricular hypertrophy, and preexcitation and other abnormal conduction (e.g., Wolf-Parkinson-White and Brugada syndrome). Wolf-Parkinson-White syndrome is associated with short P-R interval, a delta wave, and wide QRS complexes on ECG. Patients with a QT interval greater than 500 mseconds may have up to a 50% lifetime risk of sudden death. Congenital long QT syndrome may be identified by the presence of notched, broad-based or peaked T waves and UT waves. Brugada syndrome is an autosomal dominant condition affecting the sodium channel and predisposing the patient to lethal ventricular dysrhythmias. This syndrome carries a 10% mortality rate per year in symptomatic patients. The ECG in Brugada syndrome shows a complete or incomplete right bundle branch block pattern and ST segment elevations in leads V₁ and V₂. Brugada syndrome usually presents in patients 30 to 40 years old, and it may be responsible for up to 5% of cardiac arrests treated in the emergency department.^{15,16} (It should be noted that the elevated prevalence of Brugada syndrome is particularly evident in emergency departments that serve a population with a high number of Southeast Asians.)

Hypertrophic cardiomyopathy is associated with high voltage and deep, narrow Q waves in the lateral leads (I, L, V₅, V₆). Low voltage suggests pericardial effusion and abnormal conduction syndromes.

Patients suspected of having abnormal cardiac rhythms should be placed on a cardiac monitor. Monitoring may detect significant bradycardia (heart rate <30 beats/minute), sinus pauses (particularly those >2 seconds), atrial tachycardias, Mobitz II block, complete heart block, ventricular tachycardia, and frequent or multifocal premature ventricular contractions (PVCs).¹⁷

Routine laboratory screening in patients with syncope seldom aids in their evaluation and management, is not cost-effective, and is not supported by clinical evidence.^{18,19}

Hypoglycemia should be suspected in all patients with an altered mental status, and a pregnancy test is advised in all women of childbearing age who have syncope. Critically ill patients, those on diuretic medications, and those suspected of volume loss may benefit from measurement of serum electrolytes. Electrolyte studies are indicated in patients with poor oral intake, excessive vomiting or diarrhea, muscle weakness, alcoholism, altered mental status, or recent electrolyte abnormalities. A hematocrit less than 30 increases the risk of adverse short-term events in patients with syncope, and complete blood count should be considered in the patient with syncope who demonstrates hypotension, tachycardia, pallor, or rectal examination positive for evidence of bleeding.¹³

Carboxyhemoglobin levels may be useful in patients who are involved in house fires or if direct combustion is used for in-

door heating. An electroencephalogram may be useful in ruling out epilepsy.

Head computed tomography (CT) and magnetic resonance imaging (MRI) are generally of low yield and are over-utilized in the evaluation of syncope patients. There is no current evidence that a patient with syncope benefits from routine neuroimaging.²⁰ Given that loss of consciousness requires simultaneous dysfunction of both cerebral hemispheres or of the reticular activating system, it is evident that patients who spontaneously and completely recover without treatment are unlikely to have structural brain abnormalities that would be seen on neuroimaging. Patients without history or examination features that suggest neurologic disease need no further neurological studies. In contrast, patients with a history or physical examination suspicious for new onset seizure, transient ischemic attack, and stroke need further evaluation.

Echocardiography may detect the presence of cardiac valvular anomalies, wall motion abnormalities, elevated pulmonary pressure or right ventricular strain (as is sometimes seen in PE), and pericardial effusions. Echo has been shown to be most useful in patients with a history of cardiac disease or abnormal electrocardiogram findings and when aortic stenosis is suspected clinically. The current literature does not support the routine use of echocardiography as a screening test in patients with an otherwise negative screening evaluation.²¹

In suspected PE, helical CT scan may be indicated. It is noteworthy that patients with PE who present with neurocardiogenic syncope are not at increased risk when compared with other PE patients without syncope.²²

Head CT and lumbar puncture are indicated in syncope associated with a significant headache suggesting possible SAH. Head CT with angiography or MRI and neurologic consultation should be considered in suspected transient ischemic attack or stroke.

Risk Stratification

Several recent studies have attempted to stratify syncope patients with regard to risk for life-threatening events within 30 days. The Boston syncope rule utilized eight categories of signs and symptoms that placed patients at higher risk for adverse outcomes or death at 30 days (Figure 2). These were: 1) signs and symptoms of ACS; 2) signs of conduction disease; 3) worrisome cardiac history; 4) valvular heart disease by history or physical examination; 5) family history of sudden death; 6) persistent abnormal vital signs in the ED; 7) volume depletion, such as persistent dehydration, gastrointestinal bleeding, or hematocrit < 30; and 8) primary central nervous system (CNS) event.²³

The authors found that use of this instrument to screen syncope patients yielded a sensitivity of 97%, specificity of 62%, with a negative predictive value of 99%. In their population, admitting only those patients identified by the decision rule would have led to a 48% reduction in hospital admissions. Quinn et al. published the San Francisco Syncope Rule as a means of predicting patients with serious outcomes at one week. Their data

Figure 2: The Boston Syncope Rule

These criteria can be categorized as follows:

- 1) Signs and symptoms of an acute coronary syndrome (ACS)
- 2) Signs of conduction disease
- 3) Worrisome cardiac history
- 4) Valvular heart disease by history or physical examination
- 5) Family history of sudden death
- 6) Persistent abnormal vital signs in the ED
- 7) Volume depletion such as persistent dehydration, gastrointestinal bleeding, or hematocrit < 30
- 8) Primary CNS (central nervous system) event

Predicts significant risk factors for poor outcome at 30 days.

From: *J Emerg Med.* 2007;October;33(3):233-239. Predicting Adverse Outcomes in Syncope. Shama A. Grossman, MD, MS, Christopher Fischer, MD, Lewis A. Lipsitz, MD, Lawrence Mottley, MD, Kenneth Sands, MD, Scott Thompson, BA, Peter Zimetbaum, MD, and Nathan I. Shapiro, MD, MPH.

Figure 3: The San Francisco Syncope Rule

“CHESS” mnemonic

- C:** history of Congestive heart failure
- H:** Hematocrit <30%
- E:** abnormal ECG
- S:** a patient complaint of Shortness of breath, and
- S:** a triage Systolic blood pressure <90 mm Hg

FROM: Derivation of the San Francisco Syncope Rule to predict patients with short-term serious outcomes. James V Quinn, Ian G Stiell, Daniel A McDermott, Karen L Sellers, Michael A Kohn, George A Wells. *Annals of Emergency Medicine.* February 2004 (Vol. 43, Issue 2, Pages 224-232). San Francisco Syncope Rule as a means of predicting patients with serious outcomes at one week. Their data suggest that age >75 years, an abnormal ECG, hematocrit < 30, a complaint of shortness of breath, and a history of CHF are all significant risk factors for poor outcome at one week.

suggested that age >75 years, an abnormal ECG, hematocrit < 30, a complaint of shortness of breath, and a history of CHF were all significant risk factors. The San Francisco Syncope Rule had a sensitivity of 96% and specificity of 62%.¹³

Other features that place syncope patients at risk for adverse outcomes include: persistently low blood pressure (systolic <90 mmHg), shortness of breath (either with the event or during evaluation), hematocrit <30 (if obtained), older age, associated co-morbidities, and a family history of sudden cardiac death.

Figure 5: The EGSYS Score

- Palpitations preceding syncope - 4 points
- Heart disease and/or abnormal electrocardiogram (sinus bradycardia, second or third degree atrio-ventricular block, bundle branch block, acute or old myocardial infarction, supraventricular or ventricular tachycardia, left or right ventricular hypertrophy, ventricular preexcitation, long QT, Brugada pattern) - 3 points
- Syncope during effort - 3 points
- Syncope while supine - 2 points
- Precipitating or predisposing factors (warm, crowded place, prolonged orthostasis, pain, emotion, fear) - minus 1 point
- A prodrome of nausea or vomiting - minus 1 point

A score of ≥ 3 had 92% sensitivity and 69% specificity for cardiac syncope in the validation cohort. During follow-up at a mean of 20 months, patients with a score ≥ 3 had higher mortality than patients with score < 3 in both the derivation (17 versus 3%) and validation cohorts (21 versus 2%).

Source: Del Rosso A, Ungar A, Maggi R, et al. Clinical predictors of cardiac syncope at initial evaluation in patients referred urgently to general hospital: the EGSYS score. *Heart*. 2008;Jun 2 [Epub ahead of print].

One theme that emerges from a number of recent studies is that patients with an abnormal ECG on presentation or a history of heart disease, particularly structural heart disease (e.g., CHF), are at greater risk for adverse outcomes.

The Evaluation of Guidelines in Syncope Study (EGSYS) is a risk assessment tool that has been prospectively validated (Figure 5).²⁴ This score consists of the six (out of 52) items found to be most predictive of a cardiac cause of syncope: palpitations preceding syncope (4 points), history of heart disease or abnormal electrocardiogram in the ED (3 points), syncope during effort (3 points) or while supine (2 points), precipitating or predisposing factors (-1 point), and nausea or vomiting (-1 point). A score of ≥ 3 had 92 % sensitivity and 69 % specificity for cardiac syncope in the validation study. At a mean follow-up of 20 months, patients with a score ≥ 3 had higher mortality than patients with a score < 3 in both the derivation and validation studies.

One study that assessed syncope decision-making by emergency physicians demonstrated excellent patient risk stratification but that disposition decisions often were not consistent with anticipated risk. These physicians chose to admit nearly 30% of patients whom they felt had a less than 2% chance of a serious adverse outcome.²⁵

An analysis of the American College of Emergency Physicians (ACEP) clinical policy on syncope found that, by applying their recommendations, all patients with cardiac causes of syncope were identified and that the admission rate could safely have

been reduced from 57.5% to 28.5%. These facts must lead to a reassessment of the role of the emergency physician in evaluation and disposition of the patient presenting with syncope.¹²

Management

An algorithmic approach to the syncope patient was suggested by McDermott and Quinn (Figure 4).¹ The first step in this approach to the patient with apparent syncope is to determine whether syncope has actually occurred. Some syncope-like conditions to be considered include seizure, stroke, and head injury. Each of these conditions, though not syncope by definition, requires prompt stabilization, evaluation, and treatment.

The next step is to attempt to determine the cause of the syncope. As outlined above, there are historical, physical examination, and ECG features that suggest specific etiologies of syncope. If the specific cause of the syncope is a serious one (e.g., cardiovascular syncope, ACS, structural cardiac abnormalities, significant hemorrhage, PE, SAH), then admission and specific treatment is required. If a non-serious condition is identified (e.g., neurocardiogenic syncope, orthostatic hypotension, medication-related syncope), then outpatient management is usually appropriate.

If the history, physical examination, and ECG do not suggest a specific etiology of syncope, then the patient is categorized as either high risk or low risk for factors that predict adverse outcome. These high-risk features are: an abnormal ECG (e.g., ACS, dysrhythmias, or significant conduction abnormalities), history of cardiac disease, especially presence of CHF, persistently low blood pressure (systolic < 90 mmHg), shortness of breath with the event or during evaluation, hematocrit < 30 (if obtained), older age, associated co-morbidities, and a family history of sudden cardiac death. Patients with high-risk features should be admitted and evaluated with continuous cardiac monitoring and other tests as indicated. In the absence of high-risk features, asymptomatic patients with unexplained syncope may be discharged safely with outpatient follow up.

Continuous outpatient ambulatory monitoring (i.e., Holter monitoring) is of limited value in patients with rare episodes of syncope and long intervals between episodes.²⁶ Implantable cardiac monitors may be considered in these patients. These devices are placed subcutaneously in the pectoral region under local anesthesia. The monitors function as permanent loop recorders, recording rhythm abnormalities automatically or when activated by the patient. These monitors have reportedly led to a diagnosis in up to 90% of patients. Insertable loop recorders are used, especially for the detection of intermittent arrhythmias.²⁷ Further, one prospective study found that 64% of patients provided with loop recorders experienced an arrhythmia at the time of syncope.²⁷

Summary

Syncope accounts for 3% of ED visits and 1% to 6% of all hospital admissions. It is estimated that more than \$5,000 is spent per inpatient stay for syncope, and that \$2 billion a year is spent

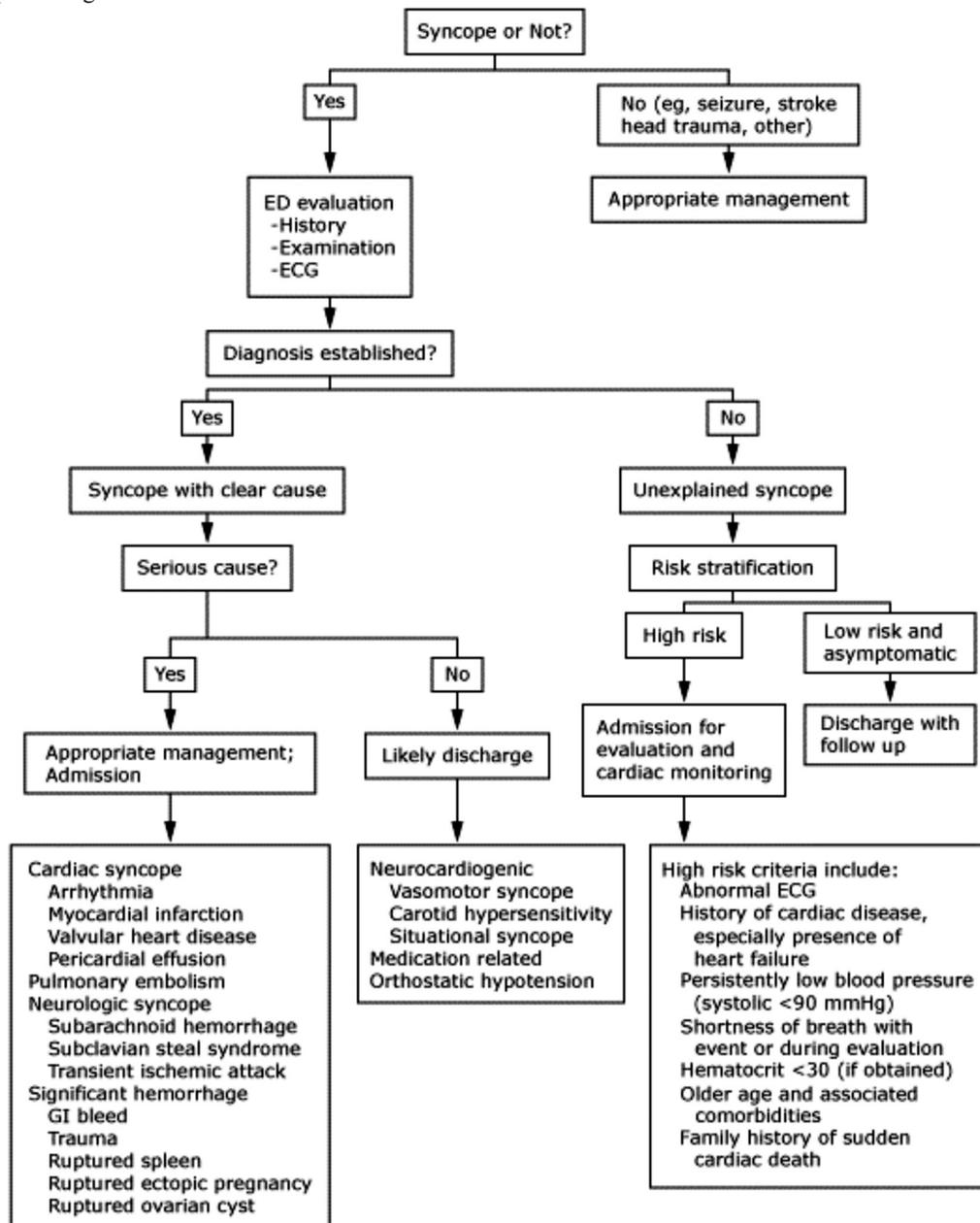
in the United States on hospitalization of patients with syncope.¹² In evaluating these patients, the emergency physicians must decide whether a life-threatening condition is present, and he or she must stabilize the patient and provide appropriate disposition. The EP must next identify those who would benefit from specific treatment or intervention and which of the patients who remain without a diagnosis will require further evaluation. The determination of the appropriate setting for this evaluation (inpatient vs. outpatient) becomes central to the decision-making process. Life threats include cardiac syncope, blood loss, PE, and SAH. Other conditions that resemble syncope, such as seizure, stroke, and head injury, must also be considered and stabilized. Further, less dangerous causes of syncope should be

identified, if possible, including neurocardiogenic, carotid sinus sensitivity, orthostasis, and medication-induced syncope.

High-risk historical and physical examination features should be elicited, and an ECG should be interpreted to differentiate those patients who are safe for discharge from those who require emergent evaluation of potentially life-threatening etiologies and in-hospital management.

Identification of the cause of syncope is possible in fewer than half of the patients during their initial evaluation. It is possible, however, to use an organized and evidence-based approach to the syncope patient that provides appropriate evaluation and stabilization and safe and cost-effective disposition for these patients.

Figure 4: Syncope ED algorithm



From: McDermott D, Quinn J. Approach to the adult patient with syncope in the emergency department. Version 16.3: October 2008. Available at: http://www.uptodate.com/online/about/contact_us.html. Accessed February 12, 2009.

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