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Syncope is defined as a sudden, transient loss of consciousness with an accompanying loss of postural tone.[1,2] As every emergency physician knows, this common symptom is oftentimes the first manifestation of a potentially life-threatening disorder that is difficult to evaluate in a comprehensive fashion within the emergency department (ED) setting.

Not infrequently, however, the ED evaluation of syncope will press into service significant technical and physician resources, requiring substantial financial expenditures, that fail to produce a diagnosis. Moreover, a lack of consensus regarding the optimal approach for assessment of patients with syncope further emphasizes the need for a standardized, evidence-based framework to guide ED physicians in these clinical encounters.

The following review emphasizes the importance of the history, physical examination, and electrocardiogram in order to distinguish between serious and benign causes of syncope. In addition, an expeditious approach for pinpointing the etiology of syncopal episodes is presented. Finally, this issue is intended to help the emergency physician differentiate high-risk patients with potentially lethal conditions that require admission from low-risk patients who may be safely discharged home with outpatient follow-up.

—The Editor

The Clinical Challenge of Syncope: A Cost-Conscious and Outcome-Driven Approach to Patient Evaluation and Disposition

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Epidemiology

Syncope is a common presenting complaint among patients presenting to the ED and accounts for about 1-6% of all ED visits.[3,4] The Framingham study found a 3.3% incidence of syncope in a cohort of patients followed over a 26-year period. Other reports indicate that the lifetime incidence of syncope may be as high as 48%, with a significant percentage of individuals choosing not to seek medical attention.[5,6]

From a practical and statistical perspective, the majority of all patients who have a documented syncopal episode have a benign etiology, they sustain minimal injury, and they have no long-term increase in mortality. Nevertheless, evaluation of syncope is critical, first because it is a common presenting complaint in the ED and second, because patients in whom syncope is the manifestation of a cardiac etiology have a high risk of mortality and sudden death in the absence of appropriate intervention.

In fact, among all patients with syncope, 5-28% present with a cardiac cause, the group with the poorest prognosis. Specifically, syncope of confirmed cardiac origin is associated with a one-year mortality rate of about 18-33%[7-11] as compared to a 6% one-year mortality in patients with syncope of unknown etiology, and 12% one-year mortality in those patients with syncope from other causes.[6,12] The increased mortality is associated with such cardiac precipitants as brad-

yarrhythmias, ventricular rhythm disturbances, and myocardial infarctions, and are the principal reason that the ED evaluation of syncope attempts to identify and triage patients with syncope of cardiac origin.

In addition, syncope occurs across a broad age range with an estimated 15-20% of all children experiencing at least one episode of syncope before the end of adolescence.[13-15] The incidence of syncope in the elderly is approximately 6% per year, with an overall recurrence rate of 30%.[16] Not surprisingly, syncope is a significant cause of morbidity and mortality in the elderly, in which the trauma resulting from falls is more often associated with major injury than it is in other age groups.[17]

Clinical Pathophysiology. Syncope, from the Greek word *synkope* or "pause," usually is caused by events that result in a transient decrease in cerebral blood flow, glucose metabolism, or oxygen supply. Although individual variation exists, a reduction in cerebral blood flow of greater than 35% will produce syncope, and a disruption of cerebral perfusion for 5-10 seconds results in loss of consciousness.[5] Cerebral blood flow is directly related to maintenance of vascular tone and systemic blood pressure that, in turn, is directly related to heart rate (HR), stroke volume (SV), and cardiac output (CO), as well as systemic vascular resistance (SVR). Four general mechanisms best describe the different types of syncope.[6]

- Vasomotor instability and sudden reduction in SVR (vasodepressor syncope);
- Decreased CO caused by mechanical obstruction (aortic stenosis, atrial myxoma);
- Decreased CO caused by hemodynamically significant arrhythmias (VT, VFIB);
- Decreased perfusion due to cerebrovascular disease (SAH, ischemic stroke, basilar artery migraine, etc.).

General Principles. Patients admitted to the ED who have had transient unconsciousness or presyncopal episodes should be treated as presenting with a major symptom with potentially serious sequelae. They should not be kept waiting in the waiting room and should immediately be admitted into the department and onto a bed. A cardiac monitor should be placed, an intravenous line started, and, in the absence of other contraindications, at least low-flow oxygen administered.

A history should then be taken from the patient with the intention of interviewing appropriate witnesses, especially if the patient does not have knowledge of the events surrounding his or her loss of consciousness. A careful history and physical examination will reveal the cause of the syncope or dizziness in up to 70% of cases.

In the history, careful differentiation of the several conditions causing diminished cerebral blood flow must be made. When faintness is related to primary cardiac pathology, there is usually a combination of dermal pallor and cyanosis. On the other hand, when peripheral circulation is at fault, pallor is usually a striking manifestation and is not accompanied by cyanosis or respiratory disturbances. When the primary disturbance is in the cerebral circulation, the face is likely to be florid and the breathing to be slow and stertorous.

During the attack, a heart rate faster than 150 beats per minute indicates an ectopic cardiac rhythm, while a bradycardia of less than 40 beats per minute suggests complete heart block. In a patient experiencing faintness or syncope attended by bradycardia, one must distinguish between a reflex vasovagal attack and cardiogenic or a Stokes-Adams type of bradycardia. Of course, the electrocardiogram (ECG) is decisive and must be taken in any elderly patient who presents with syncope.

Careful delineation of symptoms before the patient's loss of consciousness (premonitory symptoms), during the event, and after the event will allow the practitioner to place the patient in diagnostic categories that will aid in the diagnosis.

With regard to premonitory symptoms, attention should be given to the period of time during which the attack develops. If the attack begins over a period of seconds, carotid sinus syncope, postural hypotension, sudden AV block, ventricular standstill, or fibrillation is likely. When the symptoms develop gradually during a period of several minutes, hyperventilation or hypoglycemia should be considered. The occurrence of syncope during or after exertion would, of course, suggest aortic outflow obstruction.

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Table 1. Differential Diagnosis of Syncope

Cardiovascular Causes	Vasomotor or Neurally Mediated Causes
ARRYTHMIAS	
Tachycardic arrhythmias	Vasodepressor/vasovagal
Supraventricular tachycardia	Subarachnoid hemorrhage
Ventricular tachycardia	Subdural hemorrhage
Ventricular fibrillation	Ischemic stroke
Wolfe-Parkinson-White	Vertebrobasilar Insufficiency
Bradycardic arrhythmias	Transient ischemic attack
Sick sinus syndrome	Basilar artery migraine
Atrial fibrillation with slow conduction	Glasopharyngeal or Trigeminal neuralgia
2nd and 3rd degree heart block	Situation/Reflex syncope
Pacemaker malfunction	Micturation
Carotid sinus sensitivity	Defecation
Long Q-T syndrome	Post-tussive
Structural	Weightlifters
Aortic stenosis	Post-prandial
Mitral stenosis	Swallow
Pulmonic stenosis	Emotional
Hypertrophic cardiomyopathy	Orthostatic disorder
Restrictive cardiomyopathy	Anemia
Dilated cardiomyopathy	Dehydration
Atrial myxoma	TOXIC/METABOLIC
Cardiac tamponade	Drugs
Congenital heart disease	Hypoglycemia
Vascular Disease	Hypoxemia
Aortic dissection	Carbon monoxide poisoning
Myocardial infarction	Other chemicals/
Pulmonary embolus	natural gases
Pulmonary hypertension	PSYCHOGENIC
Air embolism	Anxiety disorder
Subclavian steal	Conversion disorder
Anomalous origin of left coronary artery from the right sinus of Valsalva	Somatization disorder
	Panic disorder
	Breath-holding
	Hyperventilation

The position of the patient at the onset of the attack is important. Epilepsy and syncopal attacks due to hypoglycemia, hyperventilation, or heart block are likely to be independent of posture. Faintness associated with a decline in blood pressure and with tachycardia usually occurs only in the sitting position, whereas faintness associated with orthostatic hypotension is likely to occur shortly after the change from the recumbent to the standing position.

Associated symptoms must also be noted. These include palpitations and numbness and tingling of the hands and face, which are frequent accompaniments of hyperventilation. Genuine convulsions during the attack will also prove diagnostic.

Careful query should be made about the duration of the attack. When the duration is brief (i.e., a few seconds to a few minutes), carotid sinus syncope or one of the syncopal forms of postural hypotension is most likely. A duration of a few minutes but less than an hour suggests hypoglycemia or hyperventilation.

In the physical examination, certain procedures should always be carried out in the patient with syncope. Orthostatic blood pressures with the patient lying, sitting with legs dangling, and standing should be performed, taking careful note of both blood pressure and pulse. A drop in blood pressure without a pulse rise, as indicated before, should indicate a primary autonomic disturbance causing the orthostatic hypotension. As with any patient, note carefully any abnormal vital signs. The thrust of the physical examination should be toward the detection of localizing neurologic signs, with a careful cardiopulmonary examination with specific attention toward suspicious murmurs, abnormal pulse or pulse formation, and bruits.

Etiology

Syncope can result from a variety of causes, ranging from minimal morbidity to severe life-threatening illnesses. These etiologies include: cardiac, vasomotor/neurally mediated, toxic/metabolic, and psychogenic. *(See Table 1.)* In addition, syncope may also be of unknown cause.

It should be stressed that the diagnostic yield of confirmed causes in the work-up of syncope is low and, in fact, the specific etiology of syncope is unknown in up to 50% of patients, regardless of how extensive the evaluation.[2,6,18]

Several causes of syncope listed in Figure 1 have presentations that are suggestive of the diagnosis. Unfortunately, few population-based studies have, in sufficient detail, compared the presentations of various types of syncope to permit clear differentiation among different etiologies, according to symptoms or the clinical features. Nevertheless, Figure 2 identifies characteristics of various causes of syncope that may aid in pinpointing a specific syncopal syndrome or precipitating factor. *(See Table 2.)*

Vasodepressor Syncope. The most common cause of syncope, vasodepressor syncope, results from a transient failure of autonomic cardiovascular control mechanisms. Vasodepressor syncope begins with an increase in sympathetic tone accompanied by an increase in blood pressure, heart rate, and systemic vascular resistance. This is followed by an abrupt and inappropriate reversal as vagal tone increases, causing sudden hypotension with or without bradycardia and negative inotropy. A rapid and pronounced loss of consciousness with an accompanying loss of postural tone results. Normally, the neurovascular system is controlled via reflex arcs of autoregulation involving feedback loops within the carotid sinus, aortic arch, and cardiac mechanoreceptors. *(See Table 3.)* When these reflex arcs fail, vasomotor instability and sudden reduction in SVR result in vasodepressor syncope or the "common faint." The net result is venous pooling in the lower extremities, withdrawal of sympathetic tone, and increased parasympathetic activity. This, along with decreased venous return to the heart, produces a decrease in cerebral perfusion. Because of the increase in parasympathetic- and, especially, vagal-tone, the patient's symptoms often include feelings of weakness, pallor, sweating, nausea, and abdominal cramping, all of which frequently accompany the common fainting spell.[1,6,17,19]

Vasodepressor or vasovagal syncope accounts for approximately 40% of syncopal episodes. Vasodepressor syncope carries a worse prognosis in the elderly than with younger patients. Approximately 16% of elderly patients experiencing vasodepressor syncope have major morbidity or mortality in the intervening six months, whereas less than 1% of patients younger

Table 2. Characteristics of Syncope Diagnoses

DIAGNOSIS	ONSET WITH	QUALITIES	NOTE
Arrhythmias[14,30,48]	Unpredictable triggers, although electrolyte imbalance may increase arrhythmic potential	Often sudden in onset. Often in patients with history of organic heart disease or strong family history of sudden death, arrhythmias. Quick post-event recovery of mentation	Increased risk of VT or AV block in males, the elderly, < 3 episodes syncope, < 6 sec warning. Presence of mechanical pacers often deceptively reassuring
Structural Cardiac Disease[11]	Exertion	Rapidly progressive symptoms to syncopal event. Murmurs characteristic. Positional symptoms common with myxoma	Patients with mechanical valves warrant close evaluation if no admittance
Pulmonary embolus[31,32]	Hypercoaguable state, known DVT	Sudden onset. Dyspnea, tachypnea, chest pain	Incidence of syncope associated with PE = 14%. Dx of syncope missed in 70% of patients who survive the first hour
Carotid Sinus Hypersensitivity[6,11]	Carotid sinus stimulation. Tight collars, sudden neck movement, shaving	Rapid onset. Rapid post-event mental clearing. Relatively common disorder, but uncommonly a cause of syncope.	More common in older age range. Must differentiate with VB insufficiency. More often elicited with R-sided carotid massage.
Aortic Dissection[34,48]	—	Ripping pain. Progressive clinical deterioration	Marfan's, syphilis, Cystic medial necrosis
Vasovagal[33,48]	Upright posture. Often triggered with emotion or pain	Brief prodrome of lightheadedness, visual blurring common but not unique. Rapid resolution of symptoms when patient supine	Potentially high morbidity in the elderly. Patients often otherwise healthy. Symptoms alone do not replace rule out of arrhythmic cause
CVA/TIA[6,7,22]	Unpredictable	Focal neurologic findings. Vertigo, ataxia, paresthesia, diplopia	Patient may have history of TIA, atherosclerosis, or hypercoagulable state. 1.5-7.7% of TIAs have syncope
Subarachnoid Hemorrhage[34]	Unpredictable. Sentinel bleeding possible warning	Severe headache of sudden onset with or w/o focal neurologic signs. Nausea and meningeal signs common	Family history contributive. Systolic hypertension > 200 occurs in up to 35% of patients. Cardiac dysrhythmias and ECG changes not uncommon
Vertebrobasilar Insufficiency[35]	Onset with posture change or movement of neck—especially hyperextension	Accompanying symptoms may include visual blurring, blindness, nausea, dysarthria, dysphagia. Symptoms transient	History and exam suggestive, while MRI aids in more definitive diagnosis
Hypoglycemia[3,7]	Increased insulin administration or inadequate p.o. intake	Symptoms of being jittery, diaphoresis,increasing mental status change often precede any loss of consciousness	Any associated syncopal symptoms not usually transient in nature unless hypoglycemia corrected
Hyperventilation[34]	Emotional upset or pain	Gradual onset. Gradual progression to full mental clearance with control of ventilation	Vertigo common. Association with perioral and extremity numbness, carpo pedal spasms
Basilar Artery Migraine[35]	Unpredictable. Triggers may occur, but visual prodrome often absent	Vertigo common and accompanied by vomiting, dysarthria, and commonly unilateral occipital or suboccipital headache	Usually found in younger women. Family history of migraines common

Table 2. Characteristics of Syncope Diagnoses *(continued)*

DIAGNOSIS	ONSET WITH	QUALITIES	NOTE
Glossopharyngeal or Trigeminal Neuralgia[36]	Swallowing is common trigger w/ glossopharyngeal neuralgia. Onset of syncope sudden and usually associated with paroxysmal pain	Pain a more prominent complaint. Syncope rare.	—
Reflex syncope[34]	Urination, defecation, cough, eating, swallowing, emotional upset, or weight lifting	Morbidity more significant than mortality	Cough—Often COPD and male. Seen in pediatric population. Almost never seen in women. Micturation—Often with male nocturia. Esp with ETOH. Swallow—May occur in patients with esophageal or cardiac conduction disorder
Subclavian Steal[4,11]	Exercise of arm on affected side	Global ischemia and syncope rare. Neurologic defects typically only contralateral to side of effected subclavian	—
Pulmonary Hypertension[34]	May have inciting event such as PE or MI	Functional acute obstruction to pulmonary flow decreasing preload and CO	—
Hypovolemia[43]	Prolonged bleeding, emesis, or diarrhea	Orthostatic hypotension often evident. Historical clues usually foundation of diagnosis	Finding source of hypovolemia key in managing disorder
Drug syncope[13,28,40]	Medication administration or without clear association to particular medicine	More prevalent in the elderly. Multiple medications may contribute. Characteristics of syncope vary widely	Note that the elderly overall tolerate antihtn agents well, and agents such as antiparkinson, antidepressants, and neuroleptics should be suspect as well.
Hypoxemia[35]	Any asphyxiating circumstance	Pulmonary source more common but CO, natural gas, chemical source such as bleach/ammonia mix not rare	Source of hypoxemia must be defined
Myocardial Infarction[15,20]	Unpredictable syncope onset	May be caused by multiple causes, including decreased EF, tamponade, arrhythmia	ECHO and angiogram key to management decision making
Anomalous origin of left coronary artery from the Right Sinus of Valsalva[37,38]	Exercise frequently insisting event	Usually an etiology of the pediatric population	Angiogram key to management decision making
Subdural Hematoma[35]	Unpredictable syncope onset	Preceding findings possibly subtle with forgetfulness, persistent headache	May occur at time of head trauma but most typically occurs at prolonged period afterwards
Air embolus	Large intravascular air bolus from iatrogenic or diving source	Symptoms often consistent with pulmonary embolism due to similar mechanism	Hyperbaric oxygen dive may be essential

Table 3. Causes of Drug Syncope

- Vasodilators
- Diuretics
- Antidepressants
- CNS depressants
- Drugs prolonging QT
- EtOH, marijuana, cocaine
- Adrenergic antagonists (a & B)
- Phenothiazines
- Calcium channel blockers
- Neuropathic drugs
- Central acting hypertensives
- Viagra

than age 30 who present with this type of syncope are at such risk.

This is the common faint experienced by most people. Regardless of the precise mechanism, all of these patients experience hypotension accompanied by an inappropriate slowing of the heart rate. The setting is very important in the diagnosis of vasovagal syncope. Typically, these spells occur after an emotional upset, in crowded warm rooms, or during prolonged standing, as seen in children during school assemblies or in soldiers during parades or inspections. They also occur after injurious shocking events or prolonged bed rest, and during pain and fasting. Mild blood loss, poor physical condition, anemia, fever, and organic heart disease also will predispose a person to vasovagal syncope. Such fainting spells occur in approximately 5% of normal blood donors. Full meals and warm baths, which cause diversion of blood away from the brain to the viscera and extremities, also predispose people to these spells.

Patients are always standing or sitting in the premonitory phase before these spells. Very characteristically, there is a spectrum of premonitory symptoms that lasts for at least a few seconds and usually for a few minutes or longer.

Recovery comes after the patient assumes the recumbent position, although pallor and weakness often persist. Episodes may recur within a half-hour if the patient attempts to stand again. Usually, no specific therapy is needed, although pretreatment with atropine in predisposed individuals can reduce this type of syncope. In the ED, an IV infusion of dextrose and normal saline is usually all that is required, along with monitoring. Adrenergic agents are rarely necessary.

Orthostatic Syncope. This type of syncope affects elderly patients who have a disproportion between blood volume and vascular capacitance or a chronic defect or instability of vasomotor reflexes. The character of the syncopal attack is similar to that of the vasovagal or vasodepressor type of syncope. However, the effect of posture is the cardinal feature here. Sudden rising from the recumbent or sitting position is the circumstance in which it is most likely to happen. Elderly patients are particularly predisposed to this type of syncope. They frequently lack physical conditioning and undergo prolonged illness and recumbency. Their flabby muscles allow more pooling of blood in their legs, and they frequently have venous insufficiency, which increases the capacity for the pooling of blood in the legs. Furthermore, elderly patients are subject to neuropathies, which are more frequent in their age group. The elderly diabetic patient who gets diabetic neuropathy, which may affect the autonomic nervous system, is particularly at risk. Diabetic neuropathy is very rare in juvenile diabetics. In addition, there is a higher incidence of the chronic complications of alcoholism (including peripheral neuropathy and other degenerative neuronal processes) that occur in geriatric patients.

Furthermore, there are more geriatric patients receiving antihypertensive, vasodilator, and antiparkinsonism drugs—all of which may predispose them to orthostatic hypotension.

Loss of vasoconstrictor reflexes in the resistance and capacitance vessels of the lower extremities, as mentioned above, leads to orthostatic hypotension and syncope. Elderly patients are particularly predisposed to specific abnormalities known as primary autonomic insufficiency or dysautonomias. These defects may occur either in peripheral (postganglionic) or central (preganglionic) neurons.

Cardiac Syncope. Although cardiac syncope has several causes, the principal underlying mechanism is decreased cardiac output due to either decreased stroke volume or heart rate. *(See Table 1.)* Causes of decreased stroke volume include mechanical obstructions (aortic stenosis, atrial myxoma), conditions with decreased ejection fraction (myocardial infarction, cardiomyopathies), and conditions with decreased filling time (tachyarrhythmias). Bradyarrhythmias also may lead directly to decreased cardiac output.[6,20]

Approximately 30-35% of syncopal episodes in the elderly patient result from some type of cardiac dysfunction. The morbidity and mortality of cardiac syncope is significantly higher than with other types of syncope, with a mortality rate of 19% in patients admitted to a medical intensive care unit.

Cardiac syncope results from transient reduction in cerebral perfusion from a primary decrease in cardiac output. It is the one specific cause of syncope that may occur while the patient is in the recumbent or supine position. Loss of consciousness in an elderly patient, especially when sitting or supine, must always suggest cardiac syncope until proven otherwise. The cardinal manifestation of cardiac syncope is loss of consciousness, which can occur in any position. The patient may experience a brief premonitory weakness, palpitations, or chest pain, depending on the cause of the cardiac syncope. It is estimated that 4-7 seconds of asystole are required for the patient to lose consciousness in the upright position, and that as long as 20-30 seconds are required if the patient is recumbent so that such brief premonition is not consistent with cardiac syncope. There are multiple types of cardiac syncope, including bradyarrhythmias, tachyarrhythmias, and mechanical obstruction to cardiac output.

Cardiac syncope, resulting from atrioventricular block, is perhaps the most common type of bradyarrhythmic syncope; syncopal episodes associated with this arrhythmia are known as the Morgagni-Adams-Stokes syndrome. When patients develop heart block, there is a sudden interruption of intraventricular conduction, and asystole will exist (the warm-up period) for 10-90 seconds before any ventricular rhythm begins. During this period of asystole, a dizzy spell of syncope may be experienced by the patient. Often, the clinical picture is that of an elderly individual who complains of brief episodes of dizziness and presyncope that occur without warning, often 2-3 times per day. These episodes frequently occur over a period of several weeks. If the diagnosis is not made and a pacemaker not implanted, 50% of these patients will be dead within one year.

Patients who develop complete heart block from coronary artery disease (CAD) usually have prior evidence of conduction disease if, in fact, previous ECGs are available.

There are, of course, other causes of chronic progressive heart block. Calcific encroachment into the atrioventricular (AV) node and His-Purkinje system, usually in association with aortic valve calcification, is also a relatively frequent cause. Iatrogenic heart block may result from cardiac surgery or valve replacement.

Previous ECGs may help the ED physician suspect the occurrence of complete heart block. Variable patterns that may have been present in these patients include right bundle branch block and left anterior hemiblock—perhaps the most frequent pattern in patients subsequently developing complete heart block, right bundle branch alone, left anterior hemiblock alone, or left bundle branch block alone. A chief problem is the prediction of or the likelihood of progression to complete heart block in patients with bundle branch block on a baseline ECG. Prospective follow-up studies suggest that documented or suspected heart block develops in only 4-6% of cases observed for three and one-half years. However, patients with chronic bifascicular and trifascicular conduction abnormalities show a higher incidence of subsequent heart block when they have histories of syncope (17%) than when they do not (2%).

Neurologic Syncope. Neurologic syncope results from cerebrovascular disease associated with decreased global perfusion or focal involvement of the brainstem. Neurologic syncope may also occur in the setting of such systemic metabolic derangements as hypoglycemia, hypoxemia, or secondary to toxins or drugs.

Syncope in the elderly patient is frequently attributed to transient ischemic attacks (TIAs). Loss of consciousness must involve nonperfusion of the reticular activating system, and as such, TIAs in the carotid distribution usually do not involve loss of consciousness. Vertebrobasilar system TIAs, however, may involve loss of consciousness; however, syncope alone is the exception rather than the rule. Other symptoms associated with vertebrobasilar insufficiency (including diplopia, dysarthria, bilateral weakness, bilateral visual loss, and vertigo) usually accompany loss of consciousness. One specific manifestation of vertebrobasilar TIA that both signals its origin and is relatively pathognomonic of vertebrobasilar insufficiency is the so-called "drop" attack or akinetic collapse. In this type of episode, patients usually do not lose consciousness but experience sudden inescapable paralysis of their extremities, especially the legs, falling to the floor helplessly. This usually signifies TIA of the brainstem, and these attacks are characterized by "tunnel vision," speechlessness, or ptosis.

It must be noted that although the possibility of transient cerebral ischemic attacks must always be considered in older patients who complain of spells of dizziness, virtually all symptoms commonly associated with the usual TIAs are focal in nature, whereas patients with a complete loss of consciousness generally fall into a different diagnostic category. Thus, if a careful description of the patient's symptoms indicated that the event was focal in nature, cerebrovascular disease should be the primary consideration. It should be noted, however, that a combination of extracranial occlusive disease and hypotension from another cause, even if only moderate in severity, may lead to syncope in the geriatric patient. Syncope in elderly patients also occurs during relatively brief runs of supraventricular tachycardia. Bilateral tight carotid stenosis or unilateral stenosis with occlusion of the contralateral carotid is particularly likely to predispose such patients to syncope.

Mechanisms protecting against syncope in the general population may be altered in the elderly due to age-related physiologic changes. The elderly may have a decreased baroreceptor sensitivity, as well as a blunted plasma renin and aldosterone response causing adverse alterations in extra cellular fluid balance.[21,22] Thus, any emotional or physiologic stressor applied to the elderly population results in a lower threshold for syncope.

Syncope and Seizures

Seizure disorders must be in the differential diagnosis of transient loss of consciousness in the elderly patient. Seizures are usually marked by an abrupt loss of consciousness. Unlike syncope, seizures usually occur without warning in more than 50% of cases. As with Stokes-Adams attacks, an abrupt loss of consciousness may occur in the supine position, independent of the patient's activity level or posture and without warning. A careful interview of the patient and witnesses may help distinguish the occurrence of the seizure from syncope. Although 50% of seizures occur without warning, the remainder present with a brief, momentary, premonitory syndrome or aura. This aura usually does not include weakness, dizziness, or graying of vision but, rather, does include discrete neurologic symptoms such as an auditory phenomenon, a queasy stomach, complex visual experiences, or unpleasant olfactory sensations. The seizure can occur even during sleep and may be induced by monotonous music or loud noise. It may begin with a cry as air is emitted and forced through the closed glottis. Characteristically, the eyes turn either to one side or upward, although this sign is of little value in localizing the side of the neurologic deficit. There is usually stertorous breathing and cyanosis rather than pallor. Frequently, tachycardia rather than a slow thready pulse is present.

Perhaps the most characteristic and distinguishing feature between seizures and syncope is the postictal phenomenon. Frequently, patients have been injured during their seizure episode—an occurrence most unlikely in syncope where patients have premonitory warnings to allow them to protect themselves. The period of unconsciousness does tend to be longer in epilepsy than in syncope; urinary and fecal incontinence are frequent in epilepsy and rare in syncope. The return of consciousness, as mentioned, is prompt in syncope and slow in epilepsy. Mental confusion, headache, and drowsiness are common sequelae in epilepsy, while these are rare in the postsyncopal period. Physical weakness with clear sensorium usually characterizes the postsyncopal sensorium. Of course, the occurrence of frequent tonic-clonic movements is much more characteristic of a seizure disorder.

The patient with apparent loss of consciousness may present to the ED with myriad complaints, including "falling out," "passing out," or simply "feeling dizzy." The distinction between syncope and near-syncope frequently depends upon a detailed history or eyewitness recollection. Near-syncope is just as important a symptom and, potentially, carries as significant a risk as syncope.

Multiple studies have attempted to pinpoint the etiology of syncope through standardized historical, laboratory, or physical findings.[7,23-26] Unfortunately, there is no simple recipe for determining the cause of syncope in a large percentage of patients. Nevertheless, there are aspects of the history and physical exam that may simplify the diagnosis and help the investigator to focus his/her evaluation. *(See Table 2.)*

The Red Flags of Syncope and the ESP Mnemonic

Bosker and Sequeria have encapsulated all of the dangerous aspects of syncope into an appropriate mnemonic device. They term this the ESP approach to syncope. This approach divides the diagnostic assessment of syncope into three distinct phases, the Early premonitory phase, the Syncopal phase, and the Postsyncopal phase. Each of these phases has a mnemonic to signify the respective red flags or situations in which syncope could be potentially dangerous.

The Premonitory Phase. In the early premonitory phase, the appropriate mnemonic for the dangerous situations is summarized by SCENT:

- Supine posture when syncope occurs;
- Cardiac symptoms occur just before the syncope (chest pain, shortness of breath, palpitations);
- Elderly patients should always be considered to have a serious cause of their syncope;
- No warning to the syncope should always imply cardiac or neurologic disease;
- Trauma associated with the syncope is important because the patient with benign syncope usually can protect himself or herself from the fall.

The Syncopal Phase. Red flags can be organized under the mnemonic TIPS.

- Tongue biting;
- Incontinence of urine but especially of stool;
- Prolonged duration of loss of consciousness;
- Seizure activity.

The Postsyncopal Phase. The postsyncopal red flags are organized under the mnemonic CHAN.

- Confusion;
- Headaches;
- Abnormal vital signs;
- Neurologic dysfunction (especially focal dysfunction).

The goals of the evaluation of syncope in the ED are to 1) stabilize any acute clinical needs of the patient; 2) rule out illnesses of immediate potential danger; 3) thoroughly investigate the possibility of syncope of cardiac origin; and 4) determine the appropriate disposition for each patient whether it be an admission to the hospital, referral for outpatient care, or discharge with reassurance. It is important to remember that it is not always necessary or possible to diagnose the cause of each case.

Symptom Categories. The physician must be able to identify and help the patient identify the significant differences between syncope, faintness or presyncope, lightheadedness, vertigo, and seizure disorders. Syncope primarily comprises a generalized weakness of muscles and an inability to stand and is associated with a transient loss of consciousness. Presyncope or faintness contrasts with syncope in that there is no loss of consciousness, but there is a sense of giddiness and lack of strength with a sensation of impending loss of consciousness.

Patients who have syncope usually experience it in an upright position, either sitting or standing, and have ample warning of the impending faint through a sense of "not feeling good." A sense of giddiness is accompanied by the sensation of swaying of surrounding objects. Patients yawn or gape, see spots before their eyes, and hear ringing in their ears. Nausea may accompany these symptoms, sometimes with vomiting. There is a notable pallor or ashen-gray complexion to the skin. The patient is usually profusely diaphoretic. The deliberate onset frequently allows patients to protect themselves from injury, and a hurtful fall from syncope is rare. Loss of consciousness may occasionally be averted if the patient assumes a recumbent position before he or she passes out. Patients usually remain unconscious for a period of seconds to minutes but may be unconscious for as long as 20-30 minutes. They usually lie motionless, but a few clonic jerks of the limbs and face may occur. Generalized tonic-clonic movements do not occur, although occasional urinary incontinence (but not fecal incontinence) may be noted. Once patients awaken, their color returns. They may experience sensations of being weak and may actually lose consciousness again if arising too quickly. Headache, drowsiness, and mental confusion are unusual after a syncopal episode; if these occur, they usually imply the presence of a convulsion.

It is important for the physician to help patients specify whether the complaint of dizziness represents lightheadedness or vertigo. Lightheadedness usually refers to a sensation of giddiness or faintness, while vertigo refers to a feeling of whirling rotation. Patients with vertigo have no alteration of consciousness or no sensation of an impending faint. The key symptom of vertigo is the sensation of motion, which the patient may perceive as veering, staggering, imbalance, or momentary disequilibrium, although many with vertigo may describe the classic sensation of spinning rotation.

The patient may often describe the sensation of being pulled to one side or to the ground as if drawn by a magnet—the phenomenon of impulsion. The feeling of impulsion is particularly characteristic of vertigo. All but the mildest forms of vertigo are accompanied by diaphoresis, pallor, nausea, and vomiting. As a rule, the patient can walk only with difficulty or not at all if the vertigo is intense. Most patients have previously experienced a sensation of vertigo after normal activities, such as riding a merry-go-round. When queried specifically, they often readily recognize the similarity between their symptoms and those previous experiences. If they cannot be specific in distinguishing their "dizziness," provocative maneuvers may be indicated to attempt to reproduce symptoms they have experienced.

History

A targeted evaluation of syncope requires assembling an accurate and detailed history of the sequence of events involved in the syncopal episode. *(See Table 4.)* When a cause can be found, studies suggest that the physical exam will lead to the diagnosis in 45-85% of cases.[10,11,15,28,29]

When formulating a history in the patient with syncope, it is essential to obtain an accurate account from all available witnesses. No amount of diagnostic testing can compensate for an incomplete or inadequate initial history. Details of the events

Table 4. Differentiation Among Syncope, Stokes-Adams Syndrome, and Seizures

HISTORY	SYNCOPE	STOKES-ADAMS SYNDROME	SEIZURE
Position	Usually upright	Upright/ supine	Upright/ supine
Skin color	Pale	Pallor/ cyanosis	No change
Injury	Rare	Frequent	Frequent
Episode length	Short	Variable	Long
Tonic/ Clonic jerks	Few	Few	Frequent
Tongue Biting	Rare	Rare	Frequent
Incontinence	Rarely urinary	Rarely urinary	Frequent urinary or fecal
Postictal	Promptly lucid	Promptly lucid	Return to consciousness slow; headache; confusion; weakness prolonged

preceding loss of consciousness are essential, as well as the play-by-play of events following the syncopal episode. Circumstances surrounding the episode should be ascertained, including the time, place, and duration of the episode, relationships to posture, fasting, eating, exercise, illness, sleep, medications, and patterns of recurrence. *(See Table 3.)*

Age-Related Factors. Age is a key element in the approach to syncope, inasmuch as younger and older populations each have special considerations that affect diagnostic and triage strategies. For example, it may be particularly difficult to identify the cause of syncope in the elderly (> 60 years old) because syncope in this age group is more likely to be multifactorial.[6,21,22] In particular, with advanced age, it is more likely that complex pharmacologic regimens and coexisting disease contribute to the event. The elderly may also have age-related physiologic changes that alter the syncopal response. These patients are more likely to have a cardiovascular cause of syncope, exhibiting a two-fold higher incidence than younger patients.[21] Moreover, the incidence of sudden death is three times greater in elderly patients who present with syncope.[21,25,39]

Older patients, on average, take approximately three times as many medications as the general population.[21,40] The potential for drug interactions places the older patient at increased risk for drug-induced syncope. *(See Table 3.)* In fact, a recent study identifies neuroleptics, antiparkinsonian medications, and non-tricyclic antidepressants as particularly problematic in patients 65 years and older.[40]

Syncope in the pediatric and adolescent population, in general, is a benign event. Special care should be taken, however, to inquire about syncope during exercise or a strong family history of sudden death, since this may suggest hypertrophic cardiomyopathy, anomalous origin of left coronary artery from the sinus of Valsalva, congenital aortic stenosis, long Q-T syndrome, or catecholamine-sensitive ventricular tachycardia.[14,41]

Adult patients presenting to the ED with syncope will often have cardiac risk factors. Organic heart disease (coronary artery disease, congestive heart failure, valvular heart disease, cardiomyopathy, bundle branch block, bifascicular block, or congenital heart disease) that accompanies syncope is a predictor of arrhythmia or death within one year.[25] These individuals are at a higher risk for arrhythmia and should be examined carefully to exclude potential cardiac causes for the syncopal event. In particular, the presence of three out of four of the following—abnormal ECG, age greater than 45, history of congestive heart failure, or history of ventricular arrhythmia—carries an approximately 10-fold increase in the incidence of serious arrhythmia and or death within one year.[25] Patients who have dilated cardiomyopathy and syncope carry a very high risk for sudden death; 83% of these individuals experience sudden death at 30 months (vs 32% in patients with cardiomyopathy and no syncope).[42] Finally, patients with mechanical pacemakers should undergo careful pacemaker interrogation, since intrinsic pacemaker abnormalities may not be evident on ECG alone.[29] *(See Emergency Medicine Reports, August 3, 1998.)*

Physical Examination

Physical examination of the patient with syncope should be performed with special attention to the cardiovascular and neurological organ systems. Vital signs should be assessed for signs of hypovolemia. Tachycardia or hypotension should be noted. Orthostatic vital sign changes may suggest hypovolemia, but are limited by their lack of sensitivity, particularly in the young patient.[43] The patient's heart rate should be correlated with the pulse to ensure perfusion and to assess its regularity. Differential blood pressures in both arms may indicate subclavian steal or aortic dissection. Although obtaining a temperature is often neglected or delayed in busy EDs, it is particularly important in elderly patients, since hypothermia can suggest sepsis or hypoglycemia; hyperthermia also may indicate infection.

The cardiovascular examination should be particularly thorough. The auscultatory exam may be difficult in a noisy ED, but clues to hypertrophic cardiomyopathy, valvular heart disease, and congenital cardiac malformations should be sought. For example, the murmurs of mitral regurgitation and atrial stenosis become less pronounced with the Valsalva maneuver, while the murmurs of hypertrophic cardiomyopathy or mitral valve prolapse become louder and more prolonged.

Although the nonauscultatory exam has become a lost art, it remains useful, particularly in noisy EDs. The chest should be palpated for signs of a ventricular heave or a laterally displaced PMI, suggesting ventricular enlargement. It should also be felt for irregularities of its rhythm, which are frequently found in disorders of enlarged ventricles. Parasternal palpation may reveal a heave suggesting right ventricular pressure or volume overload. A palpable thrill may accompany severe pulmonic or aortic stenosis. Finally, the neck should be evaluated for the presence and character of jugular venous distension. Cann A-waves suggest AVE block due to atrial contraction

against a closed mitral valve. In addition, the upstroke of the carotic pulse may be delayed due to aortic stenosis.

Carotid massage may be useful for investigating the possibility of carotid sinus hypersensitivity, although it also carries some risk of ventricular fibrillation, prolonged asystole, and ischemic stroke.[10,11,23] It should never be performed in the presence of carotid bruits, a history of ventricular tachycardia, recent stroke, or myocardial infarction. If indicated, the procedure is performed with the patient in a supine position. If negative, then it may be repeated with the patient standing.

Every patient with syncope deserves a thorough neurologic exam, including the pupils and retina, which should be examined for signs of cerebral edema. The abdomen should be examined for signs of a ruptured abdominal aortic aneurysm. Skin examination may reveal evidence of a thrombotic, embolic, or hemorrhagic process.

Modalities: Uses and Abuses

A number of clinical studies have shown that laboratory tests are of low yield in the evaluation of syncope, unless indicated by history or physical exam.[2,3,7,26] In this regard, laboratory tests frequently contribute to the high cost of syncope evaluations, while contributing little to the diagnosis.[1] One possible exception is the acquisition of a B-HCG when evaluating a female patient of reproductive age with syncope, since the history and physical exam may be unreliable for ruling out pregnancy.

Perhaps the greatest controversy in the evaluation of syncope involves the use of costly diagnostic modalities. The primary value of these ancillary tests lies in their ability to assist the emergency physician in making appropriate referrals and disposition decisions, including admission to the hospital. Although some tests are not routinely available in the ED, the emergency physician can often arrange for them on an outpatient basis.

ECG. Many experts advocate obtaining an ECG on all patients presenting to the ED with syncope, primarily because of the risk of sudden death associated with cardiac syncope. Although the diagnostic yield of mandatory ECG is low, ECGs are inexpensive, readily available, and may lead to a life-saving intervention when a significant diagnostic abnormality is found (e.g., complete heart block). A normal ECG is associated with a low likelihood that syncope is caused by an arrhythmia and carries a low risk of sudden death (providing important prognostic information as well).[20,25] Although the ECG in patients with syncope shows some abnormality in up to 50% of patients, significant abnormalities occur in only 2-11% of patients.[20,23,24]

Holter Monitor/Prolonged ECG Monitoring. Approximately 4% of patients with syncopal symptoms have documented arrhythmias on prolonged ECG monitoring.[29] Holter or prolonged ECG monitoring is useful because syncope often has an unpredictable onset with little reproducibility. Moreover, a static ECG may fail to diagnose transient arrhythmias. Because brief arrhythmias commonly occur in asymptomatic individuals, syncope can only be attributed to an arrhythmia when it is associated with symptoms.

How long should patients be monitored? A 24-hour period has been shown to be the optimal length of time for the initial evaluation, since monitoring beyond 24 hours fails to increase the number of clinically significant arrhythmias detected.[44] The decision to admit patients for monitoring should be driven by clinical indicators of serious illness and, especially, a strong suspicion of a cardiac cause for the syncopal episode. Specifically, when a patient reports syncope without a prodrome and has a history of organic heart disease or an abnormal ECG, then 24 hours of inpatient monitoring is indicated. In patients with nonlife threatening recurrent syncope, 24-hour Holter monitoring as an outpatient is a reasonable option. Electrocardiographic loop recording or event monitoring (for periods up to 30 days or longer) may be considered for certain patients with recurrent syncope.

CT Scan, MRI, and EEG. CT scan, MRI, and EEG are indicated in the work-up of syncope only when history or physical exam elicits evidence of neurologic abnormality. Studies investigating the value of EEG have shown that it has negligible use in the absence of history of seizure activity.[23,45] Large-scale studies have not been completed evaluating the role of CT or MRI in the syncope work-up. Still, studies have shown that CT or MRI do not lead to significant findings except when patients have focal neurologic deficits or a history of witnessed seizures.

Echocardiography. Fewer than 5% of all patients with syncope will have echocardiographic manifestations of dilated cardiomyopathy, atrial myxoma, or hypertrophic cardiomyopathy.[4,46] Consequently, the history, physical exam, and ECG are essential for identifying which patients will benefit from echocardiography. In general, however, echocardiography provides no useful additional information in syncope patients in the absence of clinical evidence of heart disease elicited by history, physical examination, or ECG.[46]

Evaluation of Cardiac Ischemia. The prevalence of acute cardiac ischemia among adult syncope patients ranges from 0.5% to 7.0%.[2,8,20] While patients with syncope are often admitted to a telemetry bed to rule out myocardial infarction, patients without chest pain or an ischemic abnormality on initial ECG have a low incidence of acute ischemia as the cause of their syncopal episode.[47] Most experts recommend that further evaluation or cardiac ischemia be reserved for those syncope patients whose history, physical exam, or ECG suggest cardiac ischemia.

Electrophysiologic studies. Electrophysiologic studies (EPS) involve electrical stimulation of cardiac ventricles to uncover disturbances that predispose to brady or tachyarrhythmias. Although EPS has played an important role in the study of ventricular tachycardia, supraventricular tachycardias, and bradyarrhythmias, it is invasive, expensive, and has a low yield when applied to the wrong patient population. EPS is best used in patients with organic heart disease.[24,46,48] Similarly, EPS for the evaluation of the syncope patient should be guided by several clinical predictors that are associated with an increased sensitivity of finding clinically significant arrhythmias. Organic heart disease and nonsustained ventricular tachycardia detected by Holter monitor increases the likelihood of diagnosing ventricular tachyarrhythmias by EPS. In addition, the presence of organic heart disease with nonsustained ventricular tachycardia by Holter monitor increases the sensitivity of EPS to 100% for detecting clinically significant arrhythmias. Sinus bradycardia,[11] heart block, or bundle branch block by ECG are associated with a 79% sensitivity for bradyarrhythmias at EPS.[44] Without these predictors, clinically significant findings detected through EPS are rare.[24]

Management and Disposition

Hemodynamically, patients with syncope require acute stabilization in the ED, with basic attention to airway, breathing, and circulation. Patients with suspected etiologies such as aortic dissection, ruptured aortic aneurysm, dissecting aneurysm, complete heart block, cardiac tamponade, and subarachnoid hemorrhage require rapid diagnosis and immediate intervention. In unstable syncope patients with an undefined diagnosis, consideration of these entities should be entertained and ruled out as rapidly as possible.

In stable patients, the evaluation should begin with a history, physical exam, and ECG, and proceed to other diagnostic modalities as indicated. If a tentative diagnosis can be made, patients should be categorized into three groups: 1) patients with suspected cardiac syncope who carry a high risk of mortality or sudden death; 2) patients with other life- or limb-threatening conditions, such as subarachnoid hemorrhage, requiring admission; 3) patients sustaining significant injury from a syncopal fall (e.g., hip fractures, subdural hematoma); and 4) elderly patients with an unclear diagnosis, but who have risk factors for cardiac or cerebrovascular disease. Patients who do not fall into these categories can generally be discharged from the ED. Some will require outpatient diagnostic testing or consultation, and others will require simple reassurance and discharge home.

Summary

Because syncope is a symptom suggesting potentially lethal underlying disease, it requires a careful and conscientious approach to patient evaluation. History, physical exam, and ECG form the cornerstone of the ED evaluation, which should focus on ruling out syncope of cardiac etiology with its associated risk of sudden death. Multiple diagnostic tools are currently available, although not all are used in the ED. Each has a potential role in the diagnostic evaluation of syncope, but should be used judiciously.

Although substantial research into syncope has been undertaken in the past 15 years, at this time, no suitable standardized approach to evaluation exists. Because the potential causes of syncope are multiple and wide-ranging, the approach to the evaluation of the patient with syncope must still be individualized. Guidelines will continue to evolve as new modalities of evaluation are created and further study clarifies the boundaries of the syncope evaluation.

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Physician CME Questions

41. Patients undergoing evaluation of syncope require all of the following *except*:
    A. history of event.
    B. family history.
    C. CT scan.
    D. ECG.
    E. physical examination.

42. Which of the following syncopal patients is *not* at risk for increased mortality or sudden death?
    A. A 75-year-old with dilated cardiomyopathy
    B. A 10-year-old male with onset of symptoms while running
    C. A previously healthy 14-year-old female with benign family history
    D. A 30-year-old with sudden onset syncope, headache, nausea, and ataxic gait
    E. A 20-year-old man with increased Q-T interval on EKG

43. CT scan or MRI is indicated in which of the following?
    A. All syncope patients older than 60 years
    B. A 25-year-old patient with visual symptoms preceding loss of consciousness
    C. A 35-year-old syncopal patient with family history of seizure disorder
    D. A 40-year-old patient with pronator-drift on neurologic examination

44. Which of the following types of agents has been recently shown to be a particular cause of drug syncope in the elderly?
    A. Selective serotonin reuptake inhibitors
    B. Antihistamines
    C. Benzodiazepines
    D. Thyroid hormone replacement agents
    E. Antiemetics

45. Head-Upright tilt table testing is a suggested modality of evaluation in which of the following patients?
    A. A 35-year-old male with family history of young cardiac deaths
    B. A 25-year-old female with monthly syncope that has a three-minute prodrome of nausea and lightheadedness
    C. A 60-year-old man with distant history of myocardial infarction and two episodes of syncope of unknown cause
    D. A 60-year-old woman with no history of cardiac illness, a normal EKG and physical examination who has had several syncopal episodes within the last five months

46. Carotid sinus massage may be helpful in the examination of which of the following patients?
    A. Syncopal patient with carotid bruits
    B. Syncopal patient with history of cerebrovascular infarction one year previously
    C. Young adult patient with frequent unexplained falls and occasional syncope
    D. Patient with pacemaker and automated defibrillator due to history of ventricular tachycardia.

47. Which of the following is *not* an indication of increased utility in electrophysiologic testing?
    A. Nonsustained ventricular tachycardia
    B. Syncope without prodrome
    C. Bundle branch block
    D. 1° heart block
    E. Sinus bradycardia

48. A history of syncope that accompanies exercise in a pediatric patient should increase suspicion of all of the following *except*:
    A. long Q-T syndrome.
    B. congenital aortic stenosis.
    C. hypertrophic cardiomyopathy.
    D. sick sinus syndrome.
    E. anomalous origin of the left coronary artery from the sinus of Valsalva.

In Future Issues

Community-Acquired Pneumonia