

*Clinical Policy: Critical Issues
in the Evaluation and Management
of Patients Presenting With
Syncope*

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Clinical Policy: Critical Issues in the Evaluation and Management of Patients Presenting With Syncope

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INTRODUCTION

Syncope is a sudden, transient loss of consciousness associated with an inability to maintain postural tone and is distinct from seizure, coma, vertigo, hypoglycemia, and other states of altered consciousness.¹ In syncope, patients return spontaneously to their baseline status with no therapeutic intervention and do not experience prolonged confusion. Syncope is a common medical problem that accounts for 6% of hospital admissions and up to 3% of emergency department visits.² The ED evaluation of patients presenting with syncope is problematic for 2 reasons. Patients are often asymptomatic when they arrive in the ED, and the differential diagnosis ranges from benign etiologies to life-threatening processes. Concerns that well-appearing patients are at risk of significant arrhythmia and sudden death has fueled extensive broad-based evaluations and frequent hospital admissions.

Studies of the 1980s demonstrated the low yield of nondirected diagnostic testing.^{1,3,4} The available medical literature looks at morbidity and mortality for patients with syncope at 6 to 12 months after the event. It remains unclear whether the current practice of admitting patients with syncope for 24 to 48 hours positively affects patient outcome. These facts, coupled with a growing awareness of rising health care expenditures, have led to a reassessment of the role of the emergency physician in evaluation of the patient presenting with syncope.

Syncope is a symptom with a broad differential diagnosis that occurs when the brain is temporarily deprived of blood flow. A 35% reduction in cerebral blood flow or complete disruption of cerebral perfusion for 5 to 10 seconds results in syncope.⁵ Any physiologic process that

transiently reduces cerebral perfusion can be the precipitant. All of these processes can be classified as 1 of 3 basic mechanisms. Vasomotor instability is associated with decreased vascular resistance, venous return, or both. Reduced cardiac output may result from pump failure, mechanical obstruction, or arrhythmias. Neurologically reduced cerebral perfusion results from cerebrovascular disease or vasospasm. The most lethal of these mechanisms is cardiac-related.

The responsibilities for the emergency physician in evaluating patients with syncope include identifying patients with life-threatening processes (eg, pulmonary embolism, subarachnoid hemorrhage) and identifying patients with non-life-threatening processes that will benefit from intervention (eg, medication-induced orthostatic hypotension). Often, however, ED evaluation of a patient presenting with syncope demonstrates neither of these processes. The emergency physician must then determine which patients require further evaluation and in what setting that evaluation should occur. This document does not attempt to outline the proper evaluation of patients presenting with syncope in whom specific diagnoses are considered but rather focuses on assisting the emergency physician in meeting these 2 responsibilities. Two critical questions are addressed: "What data help to risk-stratify patients with syncope?" and "Who should be admitted after a syncopal event?"

Recommendations offered in this policy are not intended to represent the only diagnostic and management options that the emergency physician should consider. The American College of Emergency Physicians (ACEP) clearly recognizes the importance of the individual clinician's judgment. Rather they define for the clinician those strategies for which medical literature exists to provide strong support for their utility in answering the critical questions addressed in this policy.

Methodology

This clinical policy was created after careful review and critical analysis of the peer-reviewed literature. A MEDLINE search for English-language articles published between 1985 and March 1998 was performed using the key word, syncope, with a yield of 547 articles. Abstracts and articles were reviewed by subcommittee members, and 101 pertinent articles were selected. These were evaluated, and 29 articles addressing the questions considered in this document were chosen. Subcommittee members also supplied references from bibliographies of initially selected articles or from their own knowledge base. All publications were stratified by at least 2 of the subcom-

mittee members into 1 of 3 categories of strength of evidence. Some articles were downgraded 1 or more levels based on a standardized formula that considers the size of test population, methodology, validity of conclusions, and potential sources of bias.

The reasons for developing clinical policies in emergency medicine and the approaches used in their development have been enumerated.⁶ This policy is a product of the ACEP clinical policy development process, including expert review, and is based on the existing literature; where literature was not available, consensus of emergency physicians was used. Expert review comments were received from emergency physicians, members of ACEP's Pediatric Emergency Medicine Committee and Pediatric Section, physicians from other specialties, such as cardiologists, and specialty societies including members of the American Academy of Family Physicians, American Academy of Neurology, American Academy of Pediatrics, and the American College of Cardiology. Their responses were used to further refine and enhance this policy. Clinical policies are scheduled for revision every 3 years; however, interim reviews are conducted when technology or the practice environment changes significantly.

During the review process, all articles were given a baseline "strength of evidence" by the subcommittee members according to the following criteria:

Strength of evidence Class I—Interventional studies including clinical trials, observational studies including prospective cohort studies, aggregate studies including meta-analyses of randomized clinical trials only.

Strength of evidence Class II—Observational studies including retrospective cohort studies, case-controlled studies, aggregate studies including other meta-analyses.

Strength of evidence Class III—Descriptive cross-sectional studies, observational reports including case series, case reports; consensual studies including published panel consensus by acknowledged groups of experts.

Strength of evidence Class I and II articles were then rated on elements the committee believed were most important in creating a quality work. Class I and II articles with significant flaws or design bias were downgraded from 1 to 3 levels based on a set formula. Strength of Evidence Class III articles were downgraded 1 level if they demonstrated significant flaws or bias. Articles downgraded below a Class III strength of evidence were given an "X" rating and were not used in formulating this policy.

Clinical findings and strength of recommendations regarding patient management were then made according to the following criteria:

Level A recommendations. Generally accepted principles for patient management that reflect a high degree of clinical certainty (ie, based on “strength of evidence class I” or overwhelming evidence from “strength of evidence class II” studies that directly address all the issues).

Level B recommendations. Recommendations for patient management that may identify a particular strategy or range of management strategies that reflect moderate clinical certainty (ie, based on “strength of evidence class II” studies that directly address the issue, decision analysis that directly addresses the issue, or strong consensus of “strength of evidence class III” studies).

Level C recommendations. Other strategies for patient management based on preliminary, inconclusive, or conflicting evidence, or, in the absence of any published literature, based on panel consensus.

Scope of Application

This guideline is intended for physicians working in hospital-based EDs.

CRITICAL QUESTIONS

I. What data help to risk-stratify patients with syncope?

Historical Data.

The demographic variables of age, sex, and race are potential risk factors for cardiovascular disease. Multivariate analysis in a prospective cohort study of ED patients with syncope found that age older than 45 years, male sex, and nonwhite race were predictors of clinically important arrhythmias within 1 year of presentation.⁷ Cardiovascular diagnoses and age older than 60 years increase the risk of sudden death. In a prospective cohort study, patients older than 60 years and those with a cardiovascular diagnosis regardless of age had an increase in sudden death within 2 years. The elderly were also more likely to have severe trauma from falls related to syncope.⁸ Other studies correlate short-term mortality after a syncopal episode with advanced ages.⁹⁻¹²

A detailed account of the syncopal episode can be helpful in establishing a diagnosis. An absent or brief prodrome (<5 seconds) is typical of cardiac syncope, whereas vasovagal reactions are characterized by longer prodromes.^{4,9} Obvious precipitating events or stress with a consistent history may be sufficient to diagnose vasovagal syncope.^{2,9} Postexertional syncope raises special concerns about structural heart lesions producing fixed cardiac output.¹³

Syncope that occurs while the patient is seated or reclining is likely to have a cardiac or neurologic etiology,⁹ whereas syncope that occurs within 2 minutes of standing suggests orthostatic hypotension.¹⁴ Associated respiratory or neurologic symptoms or chest pain may lead to a diagnosis of the underlying medical condition.

An account of the actual syncopal episode is ideally obtained from witnesses because the patient generally does not have accurate recall. This includes estimation of duration of loss of consciousness and evidence of seizure activity. Mild, brief tonic-clonic activity may commonly accompany syncope of any etiology.^{4,15} Witnesses also may report falls or other trauma during the episode. Post-syncopal history, also best obtained from witnesses, includes duration of confusion or lethargy after the episode and evidence of focal neurologic deficits. After a syncopal episode, patients may appear disoriented or confused, but for no more than 20 to 30 seconds, which is shorter than the postictal period typical of generalized seizures.^{4,15}

The most predictive factor for adverse outcome in the patient’s past medical history is known cardiac disease, especially a history of ventricular arrhythmia or congestive heart failure.^{1,8,10,11} Patients with poor left ventricular function and cardiac syncope are at greater risk of sudden death.^{16,17}

Medications and drug interactions are commonly associated with syncope. Some drugs prolong the QT interval and are associated with life-threatening arrhythmias. Other common offenders include vasoactive drugs such as antihypertensive agents and vasodilators used for angina. In 1 study, antihypertensive agents, other cardiovascular drugs, diuretics, and central nervous system agents were most frequently cited as a cause of syncope. Drug-related syncope was especially common in elderly patients taking multiple medications.¹⁸

The family history may be significant in young people with syncope. A family history of sudden death at a young age may indicate the presence of prolonged QT interval or hypertrophic cardiomyopathy.¹³

Patient management recommendations: historical data.

Level A recommendations. None specified.

Level B recommendations. (1) Patients older than 60 years with a history of cardiovascular disease should be considered to be at high risk of adverse outcome. (2) Patients younger than 45 years without cardiovascular disease or other risk factors should be considered at low risk of adverse outcome.

Level C recommendations. Patients with suspected reflex-mediated or vasovagal syncope should be considered at low risk of adverse outcome.

Physical Examination Data.

Vital signs. Tachycardia and hypotension may represent ongoing hemodynamic instability or volume depletion, but they are very nonspecific. Orthostatic hypotension is usually defined as a decline in systolic blood pressure on standing of 20 mm Hg or greater. This finding may identify some patients with syncope related to volume depletion, autonomic insufficiency, or medications. Recurrence of symptoms such as light-headedness or even syncope on standing is more significant than any numeric change in blood pressure. Orthostatic hypotension is common in patients with syncope of unknown etiology, as well as in patients with other documented diagnoses such as cardiac disease, and is detected in most patients within 2 minutes after standing. This finding is also present in up to 40% of asymptomatic patients older than age 70 and 23% of those younger than age 60.¹⁴

Cardiopulmonary. Physical examination findings of congestive heart failure are indicators of high risk of sudden death or early mortality after syncope.^{16,17} Murmurs indicative of valvular heart disease or obstruction to flow may prompt further evaluation as either an inpatient or outpatient depending on the presence of other risk factors.

Head (tongue). Tongue biting, particularly if it is lateral, has a high specificity for true tonic-clonic seizures. Because of low sensitivity, absence of bites has no diagnostic significance. Tongue lesions caused by falls from syncope are most likely to be anterior lacerations.¹⁹

Patient management recommendations: physical examination data.

Level A recommendations. None specified.

Level B recommendations. Patients with physical examination findings of congestive heart failure should be considered at higher risk of adverse outcome.

Level C recommendations. Patients with physical examination findings consistent with cardiac outflow obstruction should be considered at higher risk of adverse outcome.

Diagnostic Testing Data.

The cause of syncope is not established in 38% to 47% of patients despite extensive evaluation.^{1,3,4} In patients for whom a diagnosis is established, history and physical examination identified the cause in up to 85%. Despite the fact that laboratory blood studies are rarely helpful, it is common for them to be ordered.^{1,3,4}

ECG. When history and physical examination do not reveal an etiology for syncope or when cardiovascular disease is suspected, a standard 12-lead ECG is generally warranted. Although the yield of the ECG is low (5%),¹⁻⁴

the test is noninvasive and relatively inexpensive. Results are obtained quickly and obvious serious abnormalities such as evidence of myocardial infarction or life-threatening dysrhythmias can be acted on immediately. A patient with a normal ECG has a low likelihood of dysrhythmias as a cause of syncope.²⁰ In 1 study, the presence of an abnormal ECG (defined as any abnormality of rhythm or conduction, ventricular hypertrophy, or evidence of prior myocardial infarction but excluding nonspecific ST-segment and T-wave changes) was a multivariate predictor for arrhythmia or death within 1 year after the syncopal episode.⁷ The ECG allows assessment of the QT interval and consideration of idiopathic prolonged QT interval as a cause of the syncope.²¹

Cardiac monitoring. Continuous cardiac monitoring during the ED visit occasionally detects an arrhythmia not evident on a single 12-lead tracing. A strong suspicion of arrhythmias may prompt inpatient or ambulatory monitoring. For most patients, monitoring longer than 24 hours is not likely to increase the yield of significant arrhythmias. One study found 4 factors that identified patients likely to have an abnormality found on prolonged monitoring up to 72 hours: age older than 65 years, male sex, history of heart disease, and nonsinus rhythm on initial ECG. However, none of the arrhythmias detected in the second and third 24-hour periods were symptomatic.²²

Laboratory blood testing. In an evaluation of syncope, blood tests rarely yield any diagnostically useful information and their routine use is not recommended.^{1,3,4} Hemoglobin/hematocrit determination may confirm suspected acute blood loss, but examination of stool for blood may be more accurate as hemoglobin and hematocrit may be normal early in acute blood loss. Although syncope related to pregnancy is generally innocuous, a pregnancy test may be helpful in women of childbearing potential. The presence of pregnancy should not dissuade the physician from evaluation for other risk factors.²³

Patient management recommendations: diagnostic testing data.

Level A recommendations. None specified.

Level B recommendations. Obtain a standard 12-lead ECG in patients with syncope when history and physical examination do not reveal a diagnosis.

Level C recommendations. In patients without a clear etiology of syncope after history and physical examination: Initiate cardiac monitoring.

II. Who should be admitted after a syncopal event?

Closely linked to the assessment of risk in patients with syncope is the determination of the appropriate set-

ting in which any further evaluation should occur. Although current practice reveals a liberal policy toward hospital admission, no study proves that hospital admission improves outcome for patients with syncope of undetermined etiology. The primary reason for admitting patients with syncope is that the physician's risk assessment has indicated that a patient may be at risk of significant arrhythmia or sudden death. Short-term outcome, however, within the 24 to 48 hours of a typical hospital admission is not discussed in the medical literature. Endpoints for patients followed after a syncopal episode are typically reported at intervals of 6 months to 1 year.²⁴ Therefore, the emergency physician must make the admission decision in the shadow of this limitation of available literature.

One ED-based study attempted to identify factors that would define low- and high-risk groups with syncope to assist in the admission decision.⁷ Four multivariate predictors of adverse outcome were identified: a history of ventricular arrhythmias, an abnormal ECG in the ED, age older than 45 years, and a history of congestive heart failure. Using data from this study, it has been extrapolated that the 72-hour cardiac mortality for patients with no risk factors was 0%. The 72-hour risk of development of arrhythmia was 0.7% (99% confidence intervals 0% to 5.0%).²⁵ Risks for 1-year all-cause mortality or significant arrhythmia increased as the number of risk factors rose, reaching 57.6% to 80.4% in patients with 3 or 4 risk factors.⁷

Numerous studies demonstrate that 1-year mortality for patients with a cardiac cause of syncope is significantly higher (18% to 33%) than for patients with noncardiac syncope or syncope of undetermined etiology (3% to 4%).^{1-4,24,26} It was also shown that a cardiac cause of syncope was an independent predictor of mortality even when adjustments were made for differences in baseline comorbid conditions.²⁴ Although patients with a past history of angina, myocardial infarction, ventricular arrhythmia, or congestive heart failure may have noncardiac-related syncope, they are significantly more likely to have an identified cardiac cause.²⁴

Syncope in children, adolescents, and young adults is generally a benign event. However, syncope that occurs during exercise may identify some patients with a potentially fatal condition.^{13,27} Sudden death in young patients has been associated with hypertrophic cardiomyopathy, congenital heart disease, anomalous origin of the left coronary artery, myocarditis, idiopathic prolonged QT interval, and cystic medial necrosis.¹³

Patient management recommendations: admission after a syncopal event.

Level A recommendations. None specified.

Level B recommendations. Admit patients with syncope and any of the following:

1. A history of congestive heart failure or ventricular arrhythmias
2. Associated chest pain or other symptoms compatible with acute coronary syndrome
3. Evidence of significant congestive heart failure or valvular heart disease on physical examination
4. ECG findings of ischemia, arrhythmia, prolonged QT interval, or bundle branch block

Level C recommendations. Consider admission for patients with syncope and any of the following:

1. Age older than 60 years
2. History of coronary artery disease or congenital heart disease
3. Family history of unexpected sudden death
4. Exertional syncope in younger patients without an obvious benign etiology for the syncope

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