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**SUMMARY**

Syncope is a common presenting complaint in trauma patients requiring hospital admission. The primary goal of syncope evaluation is to identify patients at risk for recurrent syncopal events or cardiovascular death. Cardiac syncope carries significantly higher mortality risk compared to non-cardiac causes and requires prompt identification and management. Syncope evaluations in trauma patients frequently result in extensive diagnostic testing that yields limited actionable information. Initial evaluation should include history, physical examination, and EKG findings with patients at increased cardiac risk undergoing further testing.

**RECOMMENDATIONS**

- **Level 1**
  - None
  
- **Level 2**
  - A detailed history and physical examination should be performed to assess the patient’s syncopal event and cardiac risk factors.
  - An EKG should be obtained to assess the patient’s rhythm and atrioventricular (AV) nodal conduction.
  - Orthostatic blood pressure measurements should be performed to assess for orthostatic hypotension.
  - Inpatient telemetry monitoring, echocardiography, and additional testing is indicated when a cardiac etiology is suspected.
  
- **Level 3**
  - Routine cardiac imaging is not indicated unless a cardiac etiology is suspected based upon history and physical examination.
  - In the absence of focal neurological symptoms, carotid imaging, head CT/MRI, and EEG are not indicated.

**INTRODUCTION**

Syncope presents as an abrupt, transient, complete loss of consciousness, associated with inability to maintain postural tone (1-2). Syncope is a common cause of trauma (3). A reduction in cerebral perfusion leads to a transient loss of consciousness, loss of muscular tone, and a fall resulting in potential traumatic injury if the patient is standing. Such injuries are common among the geriatric population. 42% of the population will experience syncope by the age of 70 years (3). Syncope is associated with a two-fold increased risk of death compared to individuals without syncope (3). It accounts for 1-3% of hospital admissions in the United States and 6% of admissions for falls (2,3). Traumatic injuries occur in 30% of syncope cases with major injuries occurring in 14% of patients (3,4). The primary goal of syncope evaluation is to determine which patients are at increased risk for recurrent syncope or complications (1). The differential diagnosis for syncope is broad. It is important to determine the cause and focus management on risk stratification (2). Multiple risk stratification scoring systems have been proposed to assist in the evaluation of syncope but few have been rigorously studied or validated (3).

**LEVEL OF RECOMMENDATION DEFINITIONS**

- **Level 1:** Supported by multiple, prospective randomized clinical trials or strong prospective, non-randomized evidence if randomized testing is inappropriate.
- **Level 2:** Supported by prospective data or a preponderance of strong retrospective evidence.
- **Level 3:** Supported by retrospective data or expert opinion.

DISCLAIMER: These guidelines were prepared by the Department of Surgical Education, Orlando Regional Medical Center. They are intended as a general statement regarding appropriate patient care practices based on the medical literature and clinical expertise at the time of development. They should not be considered protocol or policy nor are intended to replace clinical judgment or dictate care of individual patients.

Syncope can be caused by multiple factors and is age dependent. Reflex syncope is most common (59%), followed by cardiac syncope (10%), and orthostatic hypotension (9%), and idiopathic (11%) (3). Cardiac causes of syncope carry the greatest risk of “sudden cardiac death”.

**Types of Syncope (after Kavi & Gall)**

<b>Pathophysiology</b>	<b>Cause</b>	<b>Signs / Symptoms</b>
Reflex syncope	Vasovagal Situational Carotid sinus syndrome Non- classical forms	Diaphoresis, nausea, pallor, and warmth after upright posture or emotional triggers
Cardiac syncope	With obstructive structural heart disease: <ul style="list-style-type: none"> <li>• Aortic stenosis</li> <li>• Hypertrophic cardiomyopathy</li> <li>• Cardiac masses</li> <li>• Pericardial disease/tamponade</li> <li>• Prosthetic valvular dysfunction</li> <li>• Congenital coronary artery abnormalities</li> </ul> With arrhythmia: <ul style="list-style-type: none"> <li>• Tachyarrhythmias</li> <li>• Bradyarrhythmias</li> </ul> Cardiopulmonary and great vessels: <ul style="list-style-type: none"> <li>• Pulmonary embolism</li> <li>• Aortic dissection</li> <li>• Pulmonary hypertension</li> </ul>	Obstruction of cardiac output limiting blood supply to the brain
Orthostatic hypotension	Medication induced Volume depletion Autonomic failure	Drop in systolic blood pressure of $\geq 20$ mmHg or diastolic blood pressure of $\geq 10$ mmHg on assuming the upright position

Predictors of recurrent syncope in older adults include aortic stenosis, impaired renal function, atrioventricular or left bundle branch block, male sex, COPD, heart failure, atrial fibrillation, advanced age (>70 years), and orthostatic medications (1). The most difficult part of syncope evaluation is differentiating etiology and risk for cardiac causes.

A thorough history and physical examination supplemented by orthostatic blood pressure measurements and an EKG, can diagnose the cause of syncope in up to 50% of patients (3). Determining whether the patient has true syncope or a non-syncopal condition is the first challenge. In obtaining a history, it is important to illicit signs occurring before and after the syncopal event including patient positioning. The presence of a prodrome such as sweating, nausea, vomiting, visual blurring, clamminess, lightheadedness, aura, premonition, and post-ictal confusion prior to the syncopal event are more likely to suggest a neurogenic rather than cardiac cause (1,5,6). A history of frequent / recurrent falls should raise concern for syncope as the cause.

A review of the patient’s medications as well as past medical history should be performed (2,5). A history of myocardial or ventricular dysfunction should raise concern for a cardiac cause of syncope. Heart murmurs could be significant cardiac causes of syncope (i.e., aortic stenosis). Carotid bruits could identify coronary artery disease or raise concern for stroke. Evaluation of neurologic status should be performed paying close attention to abnormalities of cognition, speech, visual fields, motor strength, sensation, tremor or gait disturbances (1-5).

Routine laboratory testing is rarely helpful in the evaluation of syncope. An EKG should be obtained to determine information about rhythm and atrioventricular (AV) nodal conduction. Diagnostic yield is low (2-7%), but this test is inexpensive and easy to perform. Abnormalities identified on EKG in syncopal evaluation could lead to a need for echocardiogram (ECHO) and complete cardiac evaluation (1,2).

Echocardiography is helpful when history (including family history), physical examination and EKG do not provide a specific cause of syncope in a patient with cardiac disease. An echocardiogram can identify valvular disease, structural heart disease or even suggest pulmonary embolism if there is evidence of right heart dysfunction.

Echocardiography can be useful in the young adult athlete when there is concern for hypertrophic cardiomyopathy as a cause of sudden cardiac death.

Patients with a history of cardiac disease should also be evaluated for myocardial ischemia in their syncope evaluation. Patients with a history of cardiac disease and no identified cause of syncope should undergo exercise testing (1-5).

Patients with low-risk features indicative of reflex syncope or orthostatic hypotension can usually be discharged directly from the emergency department with ongoing outpatient management. Patients with high-risk features, recurrent syncopal events, or cardiac causes for syncope usually require hospital admission for additional testing. Further work-up may include ECHO, telemetry, Holter monitor testing, implantable loop recorder, CT head, EEG, carotid duplex studies, or electrophysiology studies, however, each evaluation is patient dependent (5-7).

**Assessment of Syncope Following Trauma**

<b>Risk Stratification</b>	<b>Syncope Categorization</b>	<b>Plan</b>
Low	Reflex syncope	Discharge with outpatient management
	Orthostatic hypotension	
Intermediate	Reflex syncope	Emergency Department observation
	Cardiac syncope	
	Orthostatic hypotension	
High	Reflex syncope	Inpatient evaluation
	Cardiac syncope	
	Orthostatic hypotension	

**LITERATURE REVIEW**

Shirin et al. performed a retrospective study on 134 patients in a single-Level 1 trauma hospital who presented with syncope and fall (8). Demographics, cardiac monitoring, EKG, cardiac enzymes, orthostatic vitals, transthoracic ECHO, carotid duplex, and thyroid stimulating hormone (TSH) levels were recorded. 747 studies were done on 134 patients out of which only 17 studies (2.3%) had positive findings in 9% (12/134) of patients. This translated into 6% of patients requiring a change in treatment based on these findings. The authors concluded that most causes of syncope can be diagnosed with cardiac monitoring and EKG alone and that performing extensive diagnostic studies are not effective.

Bhat et al. conducted a retrospective study on 5420 patients with traumatic falls of which 180 patients had syncope (9). They reported a diagnostic yield of 10.1% for continuous EKG monitoring via telemetry/Holter studies, 0.7% for ECHO, and 0% for carotid duplex ultrasound.

Morrison et al. reviewed 88 patients at a Level 1 trauma center and concluded that a thorough history, physical examination, and admission laboratory values were the most helpful tools in diagnosing syncope 59% of the time with none of the patients with normal admission EKG found to have a cardiac cause of syncope (10).

## REFERENCES

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