Evaluation of Syncope

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The assessment of patients presenting with a history of syncope can be very challenging. The list of causes of syncope is enormous and not easily remembered, yet many of these etiologies can be life-threatening and, thus, cannot be overlooked. An extensive literature base exists regarding the assessment of syncope (but little of it is from randomized trials) and a plethora of tests (many quite costly) can be ordered in an attempt to diagnose the cause of syncope.

Given that this is such a complex, yet important topic, it seems to be one that is amenable to the development of guidelines so that physicians can be assisted in approaching these patients in a consistent, evidence-based, legally-defensible, cost-effective manner.

However, on accessing the National Guideline Clearinghouse (NGC) it was found that only two organizations have developed and endorsed guidelines for the assessment and treatment of syncope - the American College of Physicians (ACP) / American Society of Internal Medicine and the European Society of Cardiology (ESC). Somehow the guidelines developed by the American College of Emergency Physicians that were published in the Annals of Emergency Medicine in June of 2001 were not included in the NGC citations.

As an aside, the NGC (http://www.guideline.gov/body_home_nf.asp?view=home) is an excellent resource developed to assist health care providers and others in finding and comparing authoritative guidelines. The NGC, aside from its apparent omission of the ACEP guidelines, is claimed to be a comprehensive database of evidence-based, clinical practice guidelines and related documents, although clearly it missed inclusion of the ACEP guidelines. It is produced by the Agency for Healthcare Research and Quality (AHRQ) (formerly the Agency for Health Care Policy and Research [AHCPR]), in partnership with the American Medical Association (AMA) and the American Association of Health Plans (AAHP).

Although a small textbook could be written on syncope, it is the purpose of this essay to hone down and focus on the initial assessment of the syncopal patient using these three sets of literature-based guidelines as a reference. The ACP and ESC guidelines can be accessed via the NGC site. The two-part ACP/ASIM guidelines were also published in the June 15th and July 1st issues of the Annals of Internal Medicine (1997), while the ESC guidelines were published in the August, 2001, issue of the European Heart Journal.

Were it not difficult enough to assess syncope alone, a more basic question in patients is whether they were truly syncopal or not. Sometimes a mixed picture can be present (e.g., a patient may have a brief seizure as the results of transient poor cerebral perfusion that precipitated the syncope). Was it just a dizzy spell in which the patients thought they were going to faint? Clearly, it would appear safest to assume the worst and evaluate these patients using the "syncope" algorithm. Specifically excluded from this discussion are patients having syncope in association with ongoing symptoms as may occur with aortic rupture, pulmonary embolism, myocardial infarction, intracranial hemorrhage, cardiac tamponade and the like.

All three sets of guidelines stress the fact that the initial history and physical, if carefully performed and appropriately focused, when combined with an EKG, will
likely be able to identify the cause of syncope in about half of the patients. The ESC guidelines also recommend that orthostatic vital signs be a consistent part of the routine evaluation of these patients as well.

Four Major Categories of Causes of Syncope

To make the diagnosis of the cause of syncope, the basic pathophysiologic mechanisms need to be considered. The following is basically the categorization adopted by the ACP guidelines:

1. Neurally Mediated Syncope

   Syncope associated with inappropriate vasodilatation, bradycardia or both.
   a. Vasovagal syncope is often associated with a sensation of increased warmth and may be accompanied by nausea. It may occur after exposure to an unexpected or unpleasant sight, sound or smell, fear, severe pain, emotional distress and instrumentation. It may also occur in association with prolonged standing or kneeling in a crowded or warm place or on exertion (all three latter scenarios may also be due to autonomic failure)
   b. Situational syncope occurs during or immediately after coughing, micturition, defecation or swallowing. Syncope associated with throat or facial pain, however, may be due to glossopharyngeal or trigeminal neuralgia
   c. Carotid sinus syncope can be associated with neck pressure (shaving, tight collar) or head turning

2. Orthostatic Syncope

   Occurs when there is documented hypotension associated with syncopeal or presyncopeal symptoms. According to ECS guidelines, orthostatic blood pressures are recommended to be taken after five minutes of being supine. Measurements are repeated after one and three minutes of standing and further continued if blood pressure is still falling after three minutes or until symptomatic. A decrease of more than 20mm Hg in the systolic pressure is considered abnormal as is a drop in pressure below 90mm Hg independent of the development of symptoms.

3. Neurologic Syncope

   Neurologic causes of apparent syncope include seizures, TIAs, migraine headaches and subclavian steal syndrome. Confusion after “syncope” that lasts more than five minutes, tongue biting, incontinence, epileptic aura suggest this diagnosis. A significant differential in the blood pressure of the two arms suggests subclavian steal.

4. Cardiac-Related Syncope

   By far, the potentially most dangerous form of syncope falls into this class. Patients with known cardiac disease and syncope have a significant incidence of cardiac-related death. Unfortunately, many patients with syncope may be unknown to have cardiac disease, and, as such, depending on the nature of the history and the age of the patient, a relatively aggressive search for cardiac problems may be necessary. The major categories of cardiac disease associated with syncope are ischemia, valvular and arrhythmic.

   Physicians should be aware that a variety of drugs can be associated with syncope, some as a result of relatively benign causes (orthostatic hypotension) while others have been associated with lethal ventricular arrhythmias. Drugs of particular concern include antianginal agents, diuretics, antihypertensives, antidepressants, antiarrhythmics and drugs associated with QT prolongation. A list of drugs associated with QT prolongation and/or that induce torsades de pointes has been compiled by Raymond L. Woosley, MD, Vice President for Health Sciences at University of Arizona Health Sciences Center and is available at www.qtdrugs.org/.
Clinical Clues to Specific Causes of Syncope

If you don't ask the right question, you won't get the right answer. Specifically, if physicians don't systematically ask questions that will guide them to the cause of syncope, they won't be likely to diagnose the cause. In performing the history, it is particularly important to focus on questions that will help differentiate the cause of a patient's episode of syncope. The following list was adapted from http://www.homestead.com/emguidemaps/files/syncope.htm#clue.

1. Sudden syncope at rest when non-erect suggests a cardiac arrhythmia or atrial myxoma
2. Sudden syncope on exertion suggests aortic stenosis, hypertrophic obstructive cardiomyopathy
3. Preceding "lightheadedness" prodrome with sweating and nausea when erect that has a slow, progressive onset suggests vasovagal syncope (orthostatic hypotension would not likely have the sweating and nausea and is another cause of syncope preceded with lightheadedness)
4. Preceding palpitations suggests a cardiac arrhythmia
5. Preceding or accompanying dyspnea suggests pulmonary embolism (PE), tension pneumothorax, cardiac tamponade and air embolism
6. Preceding chest pain suggests myocardial ischemia, PE, cardiac tamponade, dissecting aneurysm, and mitral valve prolapse
7. Preceding or accompanying back pain suggests dissecting aortic aneurysm or leaking abdominal aortic aneurysm
8. Preceding or accompanying abdominal pain suggests a leaking abdominal aneurysm or ectopic pregnancy
9. Occurring when turning head side to side, shaving or with neck compression suggests carotid sinus syncope
10. Occurring when exercising an upper arm suggests subclavian steal syndrome
11. Occurring during (or immediately after) coughing, laughing, vomiting, swallowing, urination, defecation, combing hair or stretching suggests situational syncope
12. Occurring after prolonged standing suggests vasovagal syncope
13. Occurring after an emotional upset suggests either vasovagal syncope, prolonged QT syndrome or torsades de pointes
14. Recent illicit drug use suggests a cardiac arrhythmia, air or foreign body embolism
15. Syncope associated with a sudden headache suggests a subarachnoid hemorrhage
16. Recent neurologic symptoms suggests a brain stem stroke, vertebrobasilar insufficiency, basilar migraine, carotid or vertebral artery aneurysm or aortic dissection
17. Recent vaginal insufflation suggests an air embolism
18. Recent black stools suggest a GI bleed
19. Recent fluid loss (vomiting, diarrhea, sweating) or poor intake suggest hypovolemia and orthostatic hypotension or Addisonian crisis
20. Postprandial syncope is associated with a recent meal
21. Polypharmacy or sildenafil suggest orthostatic hypotension as a cause of syncope
22. A history of known cardiac ischemia or structural heart disease suggests a cardiac arrhythmia or a drug-induced arrhythmia or cardiac valvular dysfunction
23. A history of a mechanical heart valve can be associated with syncope caused by valve-related thrombosis
24. Cancer, obesity, pregnancy, recent surgery or trauma, prolonged bed rest and prior thromboembolic events suggest the presence of a pulmonary embolism as the cause of syncope.
25. A history of autonomic dysfunction manifested by impotence, anhydrosis, sphincter dysfunction can be associated with orthostatic hypotension-related syncope.

Next month we'll cover key aspects of the physical exam in assessing syncope and the role of diagnostic tests.
The obtaining of a history that effectively addresses the various mechanisms by which syncope can occur, and the diagnostic entities responsible, is likely to be the most rewarding effort in the assessment of the syncopal patient. There are, however, some very specific elements of the physical exam that can be particularly rewarding when assessing syncope and a variety of tests that may add additional useful information.

**The Physical Examination in the Patient with Syncope**

Although the history is likely to have narrowed down the likely cause of syncope in any one patient, a systematic examination, focusing particularly on the cardiovascular and neurologic systems, is essential.

**Orthostatic Vitals**

Orthostatic hypotension has been implicated as the cause of syncope by various studies in 4%-12% of cases. The combination of older age and concomitant use of antihypertensive and/or antidepressant agents and/or vasodilatory antianginal agents clearly predisposes to orthostatic syncope. Autonomic insufficiency and hypovolemia may also be detected on orthostatic testing. Customarily orthostatic hypotension is defined as a drop in systolic blood pressure of 20mm Hg or more when going from the supine to standing position after waiting two minutes.

Unfortunately, orthostatic hypotension can be present in asymptomatic individuals as well, so the test is not particularly sensitive nor specific. In fact, positive orthostatic changes have been documented in up to 40% of asymptomatic patients over the age of 70 and in about a quarter of those younger than 60. Similarly, a goodly number of children who are asymptomatic have been documented to have orthostatic hypotension as well. A differential blood pressure in the arms exceeding 20mm Hg is abnormal and suggests subclavian steal syndrome or aortic dissection.

**Cardiovascular Exam**

An examination of the neck specifically noting the presence or absence of carotid bruits and the presence of neck vein distention is appropriate. Carotid sinus syncope (resulting from reflex-mediated bradycardia and hypotension) can be the result of carotid compression (tight collar, neck pressure) or a hypersensitive baroreceptor response. A loud bruit could suggest subclavian steal syndrome or carotid artery dissection. The presence of neck vein distention may suggest the diagnosis of congestive heart failure (a known cause of arrhythmic sudden death) or pericardial tamponade.

The presence of an unusually slow, fast or irregular cardiac rhythm should be sought as the cause of syncope and, in addition, it is important to determine whether any murmurs are present. Aortic valvular pathology is particularly associated with syncope. Specifically, exertional syncope is characteristic of tight aortic stenosis (cardiac output is unable to keep up with demand in this setting). Typically this occurs in elderly patients. It also seems that there is a predisposition to cardiac arrhythmias in conditions associated with aortic area pathology in the heart, e.g., idiopathic hypertrophic subaortic stenosis (IHSS).

**Miscellaneous Items on The Physical Exam**

As noted in the introduction to this series of essays, we are focusing on transient episodes of syncope without ongoing symptoms. Clearly syncope can be the presenting symptom of a ruptured aortic aneurysm or ectopic pregnancy, a large pulmonary embolism or some other entity in which patients can be expected to have ongoing symptoms after the initial syncopal episode, and as such, they will not be discussed
here. There are, however, a number of findings that should be sought on the physical
exam that will be helpful in evaluating the syncopal patient. The presence
of incontinence or of tongue biting (particularly along the sides) is consistent with
a significant seizure (although it is not uncommon for syncope to be associated with
some convulsive movements as the brain becomes ischemic).

**Easily Accessible Adjunctive Testing in the Setting of Syncope**

**The Electrocardiogram**

Most guidelines advise that EKGs be routinely obtained unless the diagnosis is
otherwise clear cut. Some authors have claimed that about 5% of patients with syncope
will have the diagnosis made via an EKG or rhythm strip. Some of the abnormalities
that are observed on an EKG may have an unlikely relationship to syncope (e.g., stable
left bundle branch block), while others will clearly reflect the etiology (e.g., a short run
of ventricular tachycardia). There is some value in maintaining a patient on a cardiac
monitor during the emergency department evaluation of syncope in the hope of
demonstrating a transient arrhythmia not noted on the EKG. Abnormalities on the EKG
that should be specifically sought are:

- Previous or acute cardiac ischemic changes
- Signs of pericarditis or electrical alternans (cardiac tamponade)
- LVH (hypertension, aortic stenosis, HOCM)
- RVH (PE or pulmonary hypertension)
- Classical/non-specific ECG signs of PE
- WPW or Lown-Ganong-Levine syndrome
- LBBB or bifasicular block (conducting system disease)
- Bradyarrhythmias or tachyarrhythmias
- Long QT interval
- Brugada syndrome (a familial dysrhythmic syndrome characterized by partial
  RBBB with elevated ST segments in leads V1-3 and peculiar downsloping of the
  elevated ST segments with inverted T waves in those leads)
- Arrhythmogenic right ventricular dysplasia (RBBB, QRS complex > 110 msec in
  leads V1-3, inverted T wave or epsilon wave)

**Lab Tests**

All three sets of guidelines make it clear that blood counts and comprehensive
chemistry panels done without concomitant suspicion of abnormalities have a very low
yield in the setting of syncope. The ACP guidelines note that in syncope studies
(including patients with seizures), 2-3% of patients had hypoglycemia, hyponatremia,
hypocalcemia or renal failure. Specifically, the position of the ACP is:

"Routine use of basic laboratory tests is not recommended; these tests should be done
only if they are specifically suggested by the results of the history or physical
examination. Pregnancy testing should be considered in women of child-bearing age,
especially those for whom tilt- table or electrophysiologic testing is being considered."

Here's what the American College of Emergency Physicians guidelines specifically say
concerning the value of lab testing in this setting:

"In an evaluation of syncope, blood tests rarely yield diagnostically useful information
and their routine use is not recommended."

The European Society of Cardiology guidelines support the position of the ACP and
ACEP guidelines stating:

"Basic laboratory tests are only indicated if syncope may be due to loss of circulation
volume, or if a syncope-like disorder with a metabolic cause is suspected."
Other Tests

Although there are a large variety of tests that can be used in the evaluation of syncope, we will only discuss here tests that are generally immediately available. Tests for neurally mediated syncope are the tilt table test and carotid sinus massage. Of the two tests, carotid massage is easily performed and most readily available, while tilt table testing requires a table that will take a patient from 60 degrees to horizontal and, as such, is not routinely available.

The ECS guidelines advise that carotid sinus massage is recommended in patients over the age of 40 with syncope of unknown cause after the initial evaluation. Massage should be avoided in patients with bruits and in those at risk for stroke (prior TIA or stroke within the last three months, except when carotid disease has been ruled out by imaging studies).

EKG and continuous blood pressure monitoring during massage is considered mandatory. Intermittent measurement of blood pressure is specifically not advised because the drop in blood pressure that may occur may be substantial but very transient. Initially the massage should be done on the right carotid artery with massage lasting for five to ten seconds and performed at the anterior margin of the sternocleidomastoid at the level of the cricoid cartilage. A positive response is defined as asystole lasting three seconds or more and/or a drop in the systolic blood pressure or 50mm Hg or more. Because some nonsyncopal patients can also have a positive response, presyncopal symptoms should accompany a positive response for the test to be truly diagnostic.

Some advise performing the procedure with the patient initially in the supine position. If there is no positive response after several minutes, compression of the left carotid can then occur. The ECS suggests that the test also be performed in the upright position if supine testing is negative.

Echocardiography is another commonly available test; however, in the absence of clinical, physical or EKG findings suggestive of cardiac abnormalities, diagnostic yield can be anticipated to be low. Although mitral valve prolapse is likely to be the most commonly found abnormality, its relationship to syncope is probably coincidental in most cases. Examples of cardiac disease in which there can be expected to be echocardiographic abnormalities include: valvular disease (most frequently aortic stenosis), cardiomyopathies, regional wall motion abnormalities, infiltrative heart disease (e.g., amyloid), cardiac tumors, aneurysms and atrial thrombi. It is observed that echocardiography can be useful in stratifying syncopal risk as being cardiac in origin by identifying whether manifestations of heart disease are present and, if so, by determining its severity. Echocardiography is likely to make the definitive diagnosis of the cause of syncope only in the presence of severe aortic stenosis and atrial myxoma.

Indications for Admission for Syncope

The three sets of guidelines all include admission recommendations. ACEP advises admission for any patient with syncope and a history or physical evidence of congestive heart failure, ventricular arrhythmia or valvular heart disease, those with chest pain or findings compatible with an acute coronary syndrome and those with an EKG demonstrating ischemia, prolonged QT interval or bundle branch block. Admission should be considered for those over the age of 60, those with known coronary artery disease or congenital heart disease, a familial history of sudden death and younger patients with exertional syncope without an obvious benign etiology for the syncope.
To this list of patients advised to be admitted, the ACP recommendations add those who are on medications associated with arrhythmias and patients with symptoms suggestive of a TIA or stroke. The ECS extends the admission recommendations to include anyone having syncope associated with exercise and those with syncope associated with severe injury. They also suggest that "occasionally" the following patients may need to be admitted for diagnostic testing – patients without heart disease but with sudden onset of palpitations shortly before syncope, those having syncope while supine, patients with frequent recurrent episodes and those with minimal or mild heart disease when there is high suspicion for cardiac syncope. It would seem that all of these latter patients would warrant routine admission rather than "occasionally." Finally, admission is advised for those patients with cardioinhibitory, neurally-mediated syncope when pacemaker implantation is planned.

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Response

The Evaluation of Syncope: The Neurologist's Perspective

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Emergency department physicians usually have the privilege of obtaining a first-hand history of the syncopal event from the patient and eyewitnesses. Dr. Bukata correctly states that a properly taken history is likely to be the most rewarding effort in the assessment of the patient with syncope. He emphasizes that the history should effectively address the various precipitating mechanisms and diagnostic entities. Many patients are subsequently referred to a neurologist, who only receives second-hand, indirect information, including the emergency department discharge summary. This summary often contains a variety of laboratory and ancillary test results but a very brief history, which leaves many questions unanswered. It may even be misleading when any motor manifestation is labeled as "generalized convulsions".

The most common question, which then confronts the neurologist, is distinguishing between a convulsive syncope and a true seizure. This topic is well covered in several publications, notably those contributed by Dr. Lempert (1,2). The term "convulsive syncope" specifies a common variant of syncope that is accompanied by tonic or myoclonic activity. Convulsions are an integral component of the brain's response to hypoxia; they represent the rule rather than the exception. Whether or not syncope manifests with convulsions depends on the degree of cerebral hypoxia. In contrast to an epileptic seizure, which is a cortical phenomenon, muscle activation during syncope is subcortical and originates from abnormal firing of the reticular formation in the lower