The contents of this clinical practice guideline are to be used as a guide. Healthcare professionals should use sound clinical judgment and individualize patient care. This CPG is not meant to be a replacement for training, experience, CME or studying the latest literature and drug information.

Syncope Evaluation-
See Educational supplement

Abrupt and transient LOC associated with the absence of postural tone, followed by complete and usually rapid spontaneous recovery

Consider CVA/TIA, seizure, hypoglycemia or other non-syncopeal event

History, Physical exam with rectal guiaic testing and orthostatic vital signs, and EKG

High risk for heart disease or short term serious event

No further W/u needed

Diagnostic or low risk with rare syncope

Consider check BNP

1. Carotid sinus massage
2. Echocardiogram
3. ECG monitoring:
   a. Telemetry admit
   b. Holter monitor outpatient
   c. Event monitor outpatient
   d. Implantable loop recorder
4. Orthostatic challenge testing
   a. Active standing
   b. Tilt testing
5. EPS
6. ICD
7. Exercise stress testing
Syncope is the abrupt and transient loss of consciousness associated with absence of postural tone, followed by complete and usually rapid spontaneous recovery. Etiologies include: reflex (neurally-mediated, including vasovagal) 58%, cardiac disease 23%, neuro or psych 1%, and unexplained 18%

1. Initial Evaluation – should answer 3 questions: Is it syncope or something else? Has the etiology been determined? Is there evidence of high risk for cardiovascular event or death?
   A. History
      1. Type of episode – syncope or non-syncopal event with LOC (ie seizure or TIA)
      2. Number of episodes – recurrent or isolated? Isolated and rare tend to be benign
      3. Associated symptoms/PMHx – presence of CP, palpitations or dyspnea suggest etiology
      4. Prodrome – nausea, warmth, pallor, lightheadedness suggest VVS
      5. Position – neuro-cardiogenic often occurs when erect, OH usually occurs with change from supine to erect, and supine syncope suggests arrhythmia
      6. Provocative factors – situational is a neutrally-mediated reflex (ie micturition, defecation, swallowing)
      7. Exertional – an evaluation to r/o life threatening causes is required
   B. Physical
      1. Orthostatic vital signs
      2. Heart rhythm and auscultation for murmurs as well as physiologic maneuvers for murmurs (ie valsalva maneuver)
      3. Neuro exam to r/o CVA (non-syncopal event)
      4. Stool guiaic to r/o GI bleed, anemia, hypovolemia and subsequent syncope
   C. EKG – in all patients with syncope
      1. Arrhythmic cause of syncope suggested by:
         a. Bifascicular block or other IVCD with QRS >0.12 sec
         b. Mobitz I 2nd degree AV block
         c. Sinus brady < 50 bpm or sinus pause > 3 seconds (off meds that cause this)
         d. Pre-excited QRS complexes, suggesting WPW
         e. Long or short QT intervals
         f. RBBB with ST elevation in leads V1-V3 (brugada syndrome)
         g. Q waves suggesting MI
      2. Diagnostic of arrhythmia-related syncope:
         a. Persistent sinus brady < 40 BPM in an awake patient or recurrent sinus pause >3 sec
         b. Mobitz II 2nd or 3rd degree AV block
         c. Alternating LBBB and RBBB
         d. Vtach or rapid PSVT
         e. Pacemaker or ICD malfunction with pause

2. Risk stratification – if the diagnosis is uncertain at this point, further evaluation and management are based on risk. If there are single or rare episodes of syncope with low risk features, no further evaluation is indicated. High risk features necessitating evaluation are:
   A. Evidence of significant heart disease (heart failure or MI)
   B. Clinical or EKG features suggesting arrhythmic syncope
   C. Co-morbidities such as severe anemia or electrolyte disturbances
3. Testing – a variety of cardiologic tests can be used. Neurologic testing is of low yield and is overused, unless specifically suggested by history or physical. 2009 ESC guidelines recommends the following strategy:

A. Carotid sinus massage in patients > 40 years old who have not had a TIA or stroke in the past 3 months and who do not have a bruit.

B. Echocardiogram if known or suspected heart disease – only a finding of severe AS, obstructive tumor or thrombus, cardiac tamponade, aortic dissection or congenital anomaly of the coronary artery is considered diagnostic as a cause for syncope. If none of these are present, further testing is needed to determine the cause of syncope.

C. Telemetry (immediate ECG monitoring) when arrhythmia suspected. ECG monitoring is:
   1. Diagnostic when a correlation between syncope and an arrhythmia is detected
   2. Exclusive of arrhythmic cause when there is a correlation between syncope and lack of rhythm variation
   3. In the absence of above, diagnostic when ventricular pause > 3 seconds, mobitz II or 3rd degree AV block, or rapid prolonged PSVT or Vtach.

4. Options for monitoring are:
   a. In hospital – recommended if known structural heart disease
   b. Holter (24-48 hour) monitoring – recommended if clinical or ECG features suggesting arrhythmia in those with very frequent syncope or pre-syncope.
   c. External event recorder – prospective or retrospective (loop) recorders. The 2009 ESC guidelines suggested that an external loop recorder may be indicated for patients who have clinical or ECG features suggesting arrhythmic syncope AND an intersymptom interval or < or = 4 weeks.
   d. Implantable loop recorder – subcutaneous, battery life of 18-24 months. May be used either in conjunction with or in preference to provocative tests (tilt table testing or EPS). The 2009 ESC syncope guidelines recommend use of ILR:
      1. Recurrent syncope of uncertain etiology, absence of high risk criteria and high likelihood of recurrence during battery life.
      2. High risk patients in whom a comprehensive eval did not find a cause or treatment.
      3. Assess the contribution of bradycardia before pacing in patients with known or suspected reflex syncope with frequent or traumatic syncope.

D. Orthostatic challenge if syncope is related to the standing position. The 2009 ESC guidelines identify 6 orthostatic syndromes, 5 of which cause syncope and are:
   1. Classic OH (orthostatic hypotension) using active standing or tilt testing to dx.
   2. Initial OH – immediate severe BP drop- using active standing to dx
   3. Reflex (vasovagal) – diagnosed with tilt table
   4. Delayed (progressive) OH – diagnose with tilt table
   5. Delayed (progressive) OH plus reflex syndrome – diagnose with tilt table

24 hour ambulatory BP recordings can be helpful if intermittent hypotension is suspected (ie due to meds or post-prandial).

E. EPS – indications per 2009 ESC syncope guidelines:
   1. Recommended in patients with ischemic heart disease when initial eval suggests an arrhythmic cause UNLESS there is an indication for ICD
   2. Should be considered in patients with BBB when noninvasive tests are not diagnostic
F. ICD indications –
   1. Patients with LVEF < 30% and h/o MI (with or without syncope)
   2. Patients with ischemic OR nonischemic heart disease with LVEF < 35% and NYHA class II or III heart failure (and some class IV patients)

G. Exercise testing – the 2009 ESC guidelines recommend exercise testing in patients with syncope during or shortly after exertion. Echo is recommended prior to stress testing.

H. BNP – may be helpful in distinguishing cardiac from noncardiac causes of syncope. The ROSE rule (Risk stratification Of Syncope in the Emergency department) recommends hospital admission if ANY of the following independent predictors of adverse outcome is present (BRACES):
   1. BNP > 300
   2. Bradycardia < 50 BPM
   3. Rectal exam showing fecal occult blood (when GI bleed suspected)
   4. Anemia with Hgb < 9
   5. Chest pain
   6. ECG showing Q wave (not in lead III)
   7. Saturation (oxygen) < 94%

I. Less specific tests such as neuro eval or blood tests are indicated only when there is suspicion of non-syncopal transient LOC. EEG, carotid Doppler, CT or MRI are NOT recommended unless a non-syncopal cause of transient loss of consciousness is suspected.

References:

2. 2006 Scientific Statement from the American Heart Association/ American College of Cardiology (AHA/ACC).