Syncope
By Remus Popa

A 66 years old male is brought to the ED from a restaurant where he fainted while dining out with his family. He complained of nausea and stood up to go to the restroom but immediately fell back and was helped by his son to the chair. He was unconscious for approx. one minute, and then recovered completely. Denies any palpitations before the event. He has a past medical history of HTN, Hyperlipidemia and Depression. 3 years ago was briefly admitted to a hospital for chest pain and told that he had no heart problem. Medications are Lisinopril, Zocor and Prozac. In ED he states he feels at baseline, no symptoms to report, BP 127/63,HR 78,RR 14/min. Physical exam was unremarkable. One liter of IV fluids was administered and he feels even better. Labs were unremarkable including H/H and Troponin.

1. What is the best next step?
2. Admit for observation and additional testing on telemetry
3. Discharge home, no other tests indicated
4. Discharge home with referrals for outpatient Echo and Holter monitor
5. ECG in ED
6. Discharge home with referral for outpatient tilt table testing

- **Definition:** Transient loss of consciousness (T-LOC) due to transient global cerebral hypoperfusion, with inability to maintain postural tone. Rapid onset, short duration and spontaneous complete recovery are main characteristics.
- **Etiology:** Multifactorial etiology that makes diagnostic work up difficult. Three main mechanisms are explaining syncope: neural-mediated (vasovagal), orthostatic hypotension and cardiac. All these result in transient global cerebral hypoperfusion.
- **Presentation:** History should be obtained from patient, witnesses, and medical records.
  Some forms of syncope have prodromal symptoms (lightheadedness, nausea, sweating, weakness, visual disturbance). Typical syncope is brief (20 seconds to one minute. When longer, differentiate from other causes of LOC.)

- **Diagnosis**
  1. **Main steps for an accurate diagnosis are:**
     - differentiation of syncope from other causes of T-LOC (epilepsy, metabolic disorders like hypoglycemia, hypoxia, hypercapnia, intoxication)
     - etiologic diagnosis, or at least of type of syncope
     - risk stratification if etiology unclear
  2. **History features that can help differentiate types of syncope**
     a. **Neural-mediated syncope**
        i. Absence of heart disease, long history of recurrent syncope.
        ii. After sudden and unexpected unpleasant sight, sound, smell or pain. After prolonged standing, hot environment.
        iii. Associated nausea or vomiting

iv. During a meal or immediately postprandial. With head rotation or pressure on carotid sinus suggesting carotid sinus hypersensitivity.
v. After exertion

b. **Orthostatic hypotension**
    i. Shortly after standing up, standing after exertion, presence of diseases or medications that can cause autonomic dysfunction (Parkinson's, amyloidosis, DM, etc.)

c. **Cardiac**
    i. Known or highly suspected structural heart disease, family history of unexplained sudden death. Syncope during exercise or in supine position. Sudden onset of palpitations immediately followed by syncope. Abnormal ECG.

3. Physical examination: including orthostatic vital signs immediately after presentation as well as a thorough cardiac for structural heart disease (murmurs) and neurologic exam for vertebro-basilar insufficiency (limb weakness, ataxia, occulomotor palsies, diplopia and not blurred vision, visual field cut, dizziness, dysarthria), focal weakness.

4.Diagnostic testing: Multiple tests can be used in diagnosis of syncope and it is crucial to guide the work up based on information from history and physical exam.

Carotid sinus massage

Should be avoided in patients with previous TIA, stroke within the past 3 months (some also consider a carotid bruit a relative contraindication)
Carotid sinus hypersensitivity CSH-ventricular pause >3 sec and/or fall SBP>50mmHg
CSH associated with spontaneous syncope defines CSS (and benefits from pacemaker placement).

Orthostatic challenge-tilt testing

Enables reproduction of neural-mediated reflex
Clinical situation corresponding to tilt testing is reflex syncope triggered by prolonged standing. Questionable sensitivity, specificity, diagnostic yield. Can be used to confirm a neural-mediated mechanism and help with identification of prodromal symptoms that enable safety measures

ECG

Helps with diagnosis in a limited number of patients but is important for risk stratification when etiology is not clear

Telemetry monitoring

Inpatient cardiac monitoring has low yield if there are no history or exam suspicions of heart disease.

Holter monitoring-ideal for episodes that occur every day.

Implantable loop recorders-records at patient's command and all episodes of brady or tachy. Helpful for recurrent syncope, uncertain origin, absence of high-risk criteria, high likelihood of recurrence within battery lifetime and for high-risk patients with negative comprehensive evaluation.

EPS-low yield in patients without underlying heart disease, should be done as a result of abnormalities on other arrhythmia testing

Echocardiogram
Helpful if the history, physical examination and ECG suggest structural heart disease. Should not be done routinely in patients with no clinical suspicion of heart disease.

Tests for ischemia
Stress testing may play a role in patients with unexplained syncope especially if the episode was exercise related. Otherwise Troponins, stress test and cardiac catheterization have a low yield in the diagnosis of syncope without clinical or ECG suspicion of ischemia

Neurologic tests
EEG-not recommended when syncope is the most likely cause of LOC. Very low diagnostic yield.
CT, MRI –only when neurological condition is suspected based on clinical history and examination
Carotid Doppler ultrasound, MRA-no value in diagnostic work up of syncope unless clinical history and neurologic examination raise suspicion of severe cerebrovascular disease

5. Risk stratification-important especially when etiology is unclear.
Multiple risk stratification tools are available. EGSYS is a risk score derived from the European guideline and performed better than other risk scores in predicting a cardiac etiology for syncope and mortality (followed for 2 years after the index event). Patients with any of the following: 1.Abnormal ECG 2.Structural heart disease (history or signs of CHF, ischemic heart disease, valvular heart disease, ventricular arrhythmias)
3. Hemoglobin< 9 g/dl 4.Syncope during activity, exercise 5.Palpitations before syncope are considered high risk and might warrant additional work up.

Relevant ECG abnormalities	
Sinus bradycardia <40 bpm or repetitive sinoatrial blocks or sinus pauses >3 s	
Mobitz 2 or advanced (2:1, 3:1, etc) second-degree atrioventricular (AV) block or third degree AV block	Ventricular tachyarrhythmias

Alternating left and right bundle branch block (significant conduction system disease)	Q wave changes consistent with chronic ischemic heart disease, ST/T changes consistent with acute ischemia
Pacemaker malfunction with cardiac pauses	Abnormal conduction intervals (QRS>O.1 ms, QTc>450ms)
Rapid paroxysmal supraventricular tachycardia	Delta waves

Treatment

Depends on etiology or at least mechanism of syncope.

Vasovagal: No therapy has been proven to be effective in recurrent vasovagal syncope. Reassurance and education are important. Increased awareness and early recognition of prodromal signs can improve safety.

Orthostatic hypotension: Arising slowly, review medications, maintain volume status, raising head of bed 10-20 degrees to decrease nocturnal diuresis, custom-fitted compression stockings, increasing salt and water intake (target dose 6-10g/day sodium). Patient education can improve safety even if OH is not reduced.

Cardiac-treatment depends on etiology, from pacemakers to AICD, ablation, treatment if ischemic heart disease etc.

Case discussion

This patient had a vasovagal syncope (preceded by nausea, after a meal) or orthostatic (event preceded by standing). He has no history or signs of structural heart disease. The mechanism for his syncopal episode is unlikely to be cardiac, but is not entirely clarified.

In this case risk stratification is useful for further management decisions. The element missing for meaningful risk stratification is an ECG and therefore option 4 is the best answer

1. This patient does not need admission especially if ECG is normal.
2. This is the best next step once ECG is done and normal.
3. Without any suspicion for heart disease, echo and Holter will be low yield and are not indicated.
4. Since vaso-vagal syncope is the most likely diagnosis, a tilt test will not add much.

References for Further Reading

1.European Society of Cardiology Guidelines for the diagnosis and management of syncope (version 2009)
European Heart Journal (2009) 30, 2631–2671
2.Standardized Approaches to the Investigation of Syncope: Canadian Cardiovascular Society Position Paper
Canadian Journal of Cardiology 27 (2011) 246–253
3.AHA/ACCF Scientific Statement on the Evaluation of Syncope
Journal of the American College of Cardiology Vol. 47, No. 2, 2006 January 17,

2006:473–84
4.New Concepts in the Assessment of Syncope
Journal of the American College of Cardiology Vol. 59, No. 18, 2012
5.Syncope: Therapeutic Approaches
Journal of the American College of Cardiology 2009;53;1741-1751
6. Del Rosso A, Ungar A, Maggi R, et al. Clinical predictors of cardiac syncope at initial evaluation in patients referred urgently to general hospital: the EGSYS score. *Heart.* 2008;Jun 2.