APPROACH TO A PATIENT WITH SYNCOPE

Learning objectives

At the conclusion of this learning activity, participants should be able to;

- Discuss the mechanism of syncope.
- Identify the different etiologies of syncope.
- Discuss the diagnostic modalities.
- Evaluate and manage a patient of syncope.

Introduction

The term "syncope" is derived from the Greek word "to cut short" or "interrupt". It is defined as a transient, self-limiting loss of consciousness, with loss of postural tone, collapse, and spontaneous recovery. Although it can be alarming for the individual, witnesses, family, and physicians, it is most often benign and self-limited. Nevertheless, injuries associated with syncopal attacks occur in 35% of patients and recurrent episodes can be psychologically devastating. In addition, syncope can be a premonitory sign of cardiac arrest, especially in patients with organic heart disease. A transient fall of systemic arterial pressure to levels below cerebrovascular autoregulation requirements is most often to blame. Other mechanisms, such as abrupt hypoxemic events (e.g., aircraft decompression), are rare.

Syncope is characterized by temporary loss of consciousness with spontaneous and complete recovery.

The management of the patient with syncope is the same as that for presyncope, which is the prodromal symptom of fainting. Such patients usually present with symptoms of dizziness.

Epidemiology

- Syncope is very common in the general population.
- About 1% of toddlers may have a form of vasovagal syncope.
- There is a very high prevalence of first faints in patients between 10 and 30 years, with a peak of ~47% in females and 31% in males around the age of 15.
- In the Framingham study the incidence of syncope showed a sharp rise after the age of 70 years, from 5.7 events per 1000 person-years in men aged 60–69, to 11.1 in men aged 70–79.

Pathophysiology

Syncope occurs due to global cerebral hypoperfusion. Brain requires a constant adequate flow of blood flow to provide a constant supply of glucose. As brain tissue cannot store energy a cessation of cerebral blood flow lasting for even only 3-5 seconds can result in syncope. Cerebral perfusion depends on cardiac output, arterial pressure, systemic vascular resistance, intravascular

volume, cerebrovascular resistance and metabolic regulation. A clinically significant defect in any one of these or subclinical defects in several of these systems may cause syncope.

Etiology

Determining the cause of syncope is important for both prognostic and therapeutic reasons (table 1). In general, vasovagal attacks are the most common cause, followed by cardiac etiologies. The cause is unknown in approximately one-third of cases.

Table 1 -- Underlying Causes of Syncope

General cause	Causal subcategory	Indicated disease entities
Reflex mediated	Vasovagal syncope Carotid sinus syndrome Situational (e.g., coughing, sneezing, defecating, micturition, postmicturition) Glossopharyngeal and trigeminal neuralgia	Common faint Carotid sinus hypersensitivity Situational syncope
Cardiac	Mechanical or structural	Valvular disease, particularly aortic or mitral stenosis; acute cardiac syndromes or ischemia; pulmonary embolism or hypertension; acute aortic dissection; hypertrophic cardiomyopathy; pericardial disease or tamponade; atrial myxoma
	Arrhythmia	Sinus node dysfunction; second- or third-degree heart block; ventricular tachycardia; implanted device malfunction (e.g., pacemaker, implantable cardioverter-defibrillator)
Orthostatic	Primary	Multiple system atrophy; pure autonomic failure; Parkinson's disease
	Secondary	Volume depletion; medications; illegal drugs or alcohol; diabetes or amyloid neuropathy
Cerebrovascular	Vertebrobasilar	Transient ischemic attacks
	Vascular steal syndrome	Subclavian steal syndrome
	Seizure	
	Migraine	

Risk stratification

High risk	Intermediate risk
▪ Chest pain compatible with acute coronary syndrome ▪ Signs of severe heart failure ▪ Moderate to severe valvular heart disease ▪ History of ventricular arrhythmia ▪ ECG/cardiac monitor signs of ischemia ▪ Prolonged QTc (>500 ms)	▪ Age > 50 y ▪ Previous history of CAD, MI, HF ▪ Bundle branch block or Q wave without acute changes on ECG ▪ Family history of premature unexplained sudden death (<50 years)

▪ Trifascicular block/ Third-degree AV block ▪ Persistent sinus bradycardia (40 to 60 bpm) ▪ Atrial fibrillation or nonsustained ventricular tachycardia without symptoms ▪ Pacemaker or defibrillator with dysfunction	▪ Symptoms not consistent with reflex-mediated or vasovagal cause ▪ Cardiac devices without evidence of dysfunction ▪ Physician judgment: A cardiac syncope is possible
Low risk ▪ Age < 50 years ▪ No previous history of cardiovascular disease ▪ Normal cardiovascular examination ▪ Symptoms consistent with reflex-mediated or vasovagal syncope ▪ Normal ECG findings	

Presentation

Patients describe their symptoms with words such as "collapse," "fall," or "black-out." Table 2 represents the differentiating features of different types of syncope.

Table 2. Clinical features suggestive of specific causes of real or apparent loss of consciousness

Reflexly mediated syncope ▪ Absence of cardiac disease ▪ Long history of syncope ▪ After sudden unexpected sight, sound, smell, or pain ▪ Prolonged standing or crowded, hot places ▪ Nausea, vomiting associated with syncope ▪ During the meal or in the absorptive period after a meal ▪ With head-rotation, pressure on carotid sinus (as in tumors, shaving, tight collars) ▪ After exertion	**Syncope due to orthostatic hypotension** ▪ After standing up ▪ Temporal relationship with start of medication leading to hypotension or changes of dosage ▪ Prolonged standing especially in crowded, hot place ▪ Presence of autonomic neuropathy or parkinsonism ▪ After exertion
Cardiac syncope ▪ Presence of definite structural heart disease ▪ During exertion or supine ▪ Family history of sudden death ▪ Physical examination and/or echocardiographic evidence of severe structural heart disease ▪ Palpitations at the time of syncope ▪ Abnormal electrocardiogram findings	**Cerebrovascular syncope** ▪ Arm exercise ▪ Differences in blood pressure or pulse in the 2 arms

Diagnosis

The differentiation between syncope and non-syncopal conditions with real or apparent loss of consciousness (LOC) can be achieved in most cases with a detailed clinical history, but sometimes can be extremely difficult.

The following questions should be answered:

- Was LOC complete?
- Was LOC transient with rapid onset and short duration?
- Did the patient recover spontaneously, completely and without sequelae?
- Did the patient lose postural tone?

If the answers to these questions are positive, the episode has a high likelihood of being syncope. If the answer to one or more of these questions is negative, exclude other forms of LOC before proceeding with syncope evaluation.

Table 3 -- Syncope versus seizure

Syncope	Seizure
No aura	Aura
Post-LOC jerks	Pre-LOC jerks
Asynchronous jerks	Synchronous jerks
Tongue bite at tip	Tongue bite lateral
Flaccid	Stiff
Quick recovery	Postictal
No anion gap acidosis	Transient anion gap acidosis

Physical Examination

⇒ The physical examination should focus on vital signs, including measures of orthostatic and bilateral blood pressure and the cardiovascular and neurologic systems.

⇒ The cardiac examination should assess volume status, blood pressure in standing and supine position, valvular heart disease, and rhythm disturbance.

⇒ The neurologic examination should seek signs of focal neurologic deficit. Signs of occult blood loss should be assessed.

Additional Testing

⇒ Cerebrovascular causes of syncope are rare, and head CT, MRI, EEG, and carotid ultrasonography should be ordered only when history or physical examination suggests a neurologic cause or after testing for cardiac or neurally mediated causes of syncope has been completed.

⇒ Carotid sinus massage (CSM) is used as a means of confirming a suspected diagnosis of carotid sinus syncope. CSM should be undertaken for 5-10 seconds with patient in an upright position. CSM is applied for approximately 5 seconds, and the test is considered positive if there is a ventricular pause ≥3-5 seconds and/or a fall of systolic blood pressure ≥50 mm Hg. Relative contraindications to CSM include patients with TIAs or strokes within the past 3 months, or significant carotid artery narrowing.

⇒ Abnormal electrocardiogram (ECG) findings occur in about 90 percent of patients with cardiac-induced syncope and an ECG should be done in all cases with syncope. A 24 ECG (Holter) monitoring is indicated when there is an increased probability of identifying an arrhythmic cause for syncope.

⇒ Echocardiography is usually not useful but may be done in those with a heart murmur.

⇒ Exercise testing can diagnose ischemia and exercise-induced tachyarrhythmias or reproduce exercise-associated and exertional syncope.

⇒ Head-up tilt-table (HUTT) testing is used for evaluation of patients with unexplained syncope. HUTT testing uses changes in position to reproduce the symptoms of the syncopal event by inducing bradycardia or hypotension suggestive of reflex-mediated syncope.

⇒ The Adenosine triphosphatase (ATP) injection test has been advocated as a rapid, safe, and useful means for identifying abrupt bradycardia as a cause of syncope. This test is useful in older fainters in whom other causes have been excluded. However, the true value of the ATP test remains controversial.

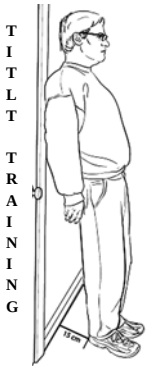
Management

Treatment of the syncope patient may be divided into 2 parts. The first is management of an acute syncopal event and the second part is prevention of syncope recurrences. Treatment of syncope also relies primarily on clinical experience of the physician in dealing with such cases. The management of different types of syncope is discussed below.

- **Neurally mediated reflex syncopal syndromes**
  - Patient must be educated about their disorder and informed that, although reflex faints are almost never life-threatening, they tend to recur and can lead to injury if preventive measures are not taken.
  - Educate susceptible individuals to recognize impending events and take action to prevent the episodes (e.g., sitting or lying down with feet elevated).
  - Patients should also be taught to be alert to warning symptoms such as feeling of being hot or cold, sweaty, clammy, short of breath, or nauseated.
  - Identify and treat any existing psychological and/or psychiatric factors that might exacerbate the symptoms.
  - Carotid sinus syndrome tends to occur in older individuals and predisposes them to falls and injury. In such cases, although avoidance of tight collars, neckties, and abrupt neck movements is prudent advice, early initiation of cardiac pacing is usually recommended.
  - Strategies for reducing syncope recurrences in the long-term comprise: 1) physical techniques; 2) pharmacologic interventions; and 3) cardiac pacing.

    1) Physical Techniques

    - The goal of "tilt-training" ("standing-training") is to enhance neurovascular response to orthostatic stress.
    - Patients are instructed to stand and place only the upper back against a wall (with ankles approximately 15 cm away from the wall) without moving.
    - Initially 3 to 5 min of standing twice daily is recommended. Then, depending on symptom status, the duration can be slowly increased each week. The target is 20 to 30 min twice daily without symptoms. Thereafter, 20 min sessions 3 to 4 times/week are recommended indefinitely.



2) **Pharmacotherapy**

- No much success has been found with drugs except midodrine, which constricts both arterial and venous beds, thus increasing peripheral blood pressure, improving venous return, and diminishing venous pooling. Midodrine has been most extensively studied in patients with orthostatic hypotension
- Volume expansion through use of fluids rich in dietary salts and electrolytes and liberalized salt intake. For volume expansion, fludrocortisone is the most widely used drug.
- Other drugs which may be used are beta-blockers, vasoconstrictors and venoconstrictors (Etilephrine), disopyramide, pure anticholinergics (e.g., scopolamine), and theophylline.

3) **Cardiac Pacing**

- It is considered as an essential part of the treatment of carotid sinus syndrome. However, its role in patients with "refractory" vasovagal syncope is less certain. The proposed mode of benefit is thought to be prevention of severe bradycardia (cardio-inhibitory syncope).

- **Orthostatic syncope and related autonomic disturbances**
  - Orthostatic syncope results from an excessive fall of systemic pressure (hypotension) triggered by postural change (e.g., supine or sitting to upright posture). It is defined as a reduction of systolic blood pressure of ≥20 mm Hg and/or a diastolic fall of ≥10 mm Hg within 3 min of standing, regardless of whether symptoms occur.
  - Patients should be advised regarding maintenance of hydration (volume expanders) and "tilt-training".
  - Patients with autonomic failure should be advised to sleep with the head of the bed somewhat elevated (approximately 20 to 25 cm).
  - Other agents advocated for treatment of orthostatic hypotension in specialized circumstances include erythropoietin, clonidine, octreotide, and desmopressin.

- **Cardiac arrhythmias as primary cause of syncope**
  - Management includes cardiac pacemakers for bradyarrhythmias, ablation for many supraventricular tachycardias and ventricular tachycardias of right or left ventricular outflow tract origin or due to bundle-branch re-entry.

- **Structural cardiovascular disease**
  - Most often, syncope associated with structural heart disease is through either neurally mediated reflex mechanisms (e.g., acute myocardial ischemia) or primary arrhythmias, both of which have been discussed above.
  - For severe valvulopathies surgery may be required.

- **Cerebrovascular syncope**
  - Medical management of migraine includes use of beta-blockers and cranial/basilar artery vasoconstrictors such as sumatriptan.
  - Subclavian steal syndrome requires either surgical or catheter-based intervention.

Conclusions

⇒ The evaluation and treatment of syncope is challenging.
⇒ Syncope is only one of many causes of transient loss of consciousness.
⇒ Symptoms are fleeting, and the patient is usually asymptomatic when seen in the clinic.
⇒ A deliberate approach based on initial risk stratification is more likely to reap the reward of a correct diagnosis.
⇒ A thorough evaluation of the cause of syncope is warranted in all patients
⇒ The goal in every case should be to determine the cause with sufficient confidence to provide a reliable assessment of prognosis and treatment options.

Suggested reading

⇒ Robertson, D, Robertson, M. Causes of chronic orthostatic hypotension. Arch Intern Med 1994; 154:1620.
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⇒ David G. Benditt, John T. Nguyen. Syncope: Therapeutic Approaches. Journal of the American College of Cardiology, Volume 53, Issue 19, 12 May 2009, Pages 1741-1751
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⇒ Weimer LH. Neurological aspects of syncope and orthostatic intolerance. Med Clin North Am - 01-MAR-2009; 93(2): 427-49, ix
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⇒ Miller TH. Evaluation of syncope. Am Fam Physician - 15-OCT-2005; 72(8): 1492-500

TEST QUESTIONS

1. Syncope is characterized by the following features except

A. Transient loss of consciousness
B. Loss of postural tone
C. Collapse
D. Delayed recovery

2. The most commonly accepted pathogenesis of syncope is;

A. Partial cerebral hypoperfusion
B. Global cerebral hypoperfusion
C. Global cerebral hyperperfusion
D. Partial cerebral hyperperfusion

3. **The most common cause of syncope is**
   A. Vasovagal syncope
   B. Carotid sinus syndrome
   C. Valvular heart disease
   D. Vascular steal syndrome

4. **Following are the features which constitutes a high risk group for developing syncope, except;**
   A. Chest pain compatible with acute coronary syndrome
   B. Signs of severe heart failure
   C. Persistent sinus bradycardia (40 to 60 bpm)
   D. Symptoms consistent with reflex-mediated or vasovagal syncope

5. **Features of syncope due to orthostatic hypotension is _____.**
   A. After standing up or exertion
   B. Temporal relationship with start of medication leading to hypotension or changes of dosage
   C. Prolonged standing especially in crowded, hot place
   D. Presence of autonomic neuropathy or Parkinsonism
   E. All the above

6. **All the following features differentiate syncope from seizures, except;**
   A. No aura
   B. Asynchronous jerks
   C. Tongue bite at tip
   D. Stiff muscles

7. **Drug used in the management of neurally mediated reflex syncopal syndrome is;**
   A. Fludrocortisone
   B. Beta-blockers
   C. Vasoconstrictors and venoconstrictors (Etilephrine)
   D. Anticholinergics (e.g., scopolamine)
   E. All the above