Clinical Evaluation of Syncope

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**SYNCOPE**

Definition: Syncope refers to sudden, transient loss of consciousness and postural tone with spontaneous recovery. It is a fairly common clinical problem, accounting for about 3% of casualty visits. Syncope has the following characteristics. It is a brief episode of loss of consciousness that is:
- Sudden in onset
- Transient
- Followed by spontaneous, and usually a prompt complete recovery

**IMPORTANCE OF DEFINING THE PROBLEM**

Patients or relatives with non-syncopal states often come with the symptom of fainting. Altered states of consciousness, transient ischemic attacks, and elderly people with falls present for evaluation of syncope. “It is important to differentiate states of stupor, altered sensorium as the diagnostic workup for these conditions are different. History of vertiginous feeling (of objects or room spinning around) without loss of consciousness indicates that one is not dealing with true syncope, but a vestibular disorder”. These conditions should not be confused with syncope.

Most causes of syncope are benign but some are associated with adverse fatal outcome. Syncope from cardiovascular causes has the highest risk of death, with a 1-year mortality rate ranging from 18% to 33%. In contrast, reported 1-year mortality for syncope of unknown etiology is 6% and from other, non-cardiovascular causes is 12%-.

**SYNCOPE vs SEIZURES**

Presence of aura, cry and tonic - clonic movements suggests a diagnosis of seizure. “It is important to obtain the history from the person who witnessed the episode of Syncope”. Recovery is usually prompt in syncope. However even brief episodes of seizures are associated with post - ictal states of confusion, sleepiness and/or headache. Clenching of teeth, frothing in the mouth and sphincter incontinence are more often seen in seizures.

**When does a person faint**

It would be useful to think of broad categories of conditions while evaluating syncope:

1. Exertional Syncope is often due to cardiac causes (e.g.: Hypertrophic Cardiomyopathy, Left Ventricular Outflow Obstruction, Arrhythmia, etc).
2. Syncope occurring repeatedly during certain physiological states e.g.: Micturition / cough etc, suggests a reflex mechanism (neurocardiogenic).
3. Neuro Cardiogenic syncope usually occurs after prolonged standing or during states of emotional stress. Orthostatic hypotension occurs after standing.

Recurrence of syncope, particularly among the elderly, is common (approximately 30%). While the actual cause of syncope may be a benign disorder, the consequence of trauma / fall can contribute to significant morbidity and mortality, especially in the elderly.

**Pathophysiology**

Reduction of blood flow to both cerebral hemispheres or the brainstem (reticular activating system), results in acute hypoperfusion. This leads to loss of consciousness and postural tone, with resulting syncope. As syncope is a transient event, it follows that the cause of central nervous system (CNS) dysfunction must also be transient. Persistent causes of significant CNS dysfunction results in altered mental status or even coma (cerebrovascular and metabolic causes).

**CAUSES OF SYNCOPE**

1. **Vascular Causes of Syncope**

   Vascular causes of syncope, particularly reflex-mediated syncope and orthostatic hypotension, are by far the most common causes of syncope, accounting for at least one third of all syncopal episodes.

   In contrast, subclavian steal syndrome is an exceedingly uncommon cause of syncope.

   Reflex-mediated Syncope

   Neurologically mediated hypotension / syncope (Vasovagal Syncope) is a common abnormality of blood pressure regulation characterized by the abrupt onset of hypotension, with or without bradycardia. Triggers associated with the development of neurally mediated syncope either reduce ventricular filling or increase catecholamine secretion. When ventricular preload is reduced by venous pooling, there is a reduction in cardiac output and blood pressure, which is sensed by arterial baroreceptors. The resultant increase catecholamine levels, combined with reduced venous filling, leads to a vigorously contracting but under filled ventricle. Normally the increase in heart rate and contractility helps to maintain the cardiac output. Occasionally a paradoxical response is seen. The heart itself is involved in this reflex by virtue of the presence of mechanoreceptors, or C-fibers, consisting of nonmyelinated fibers found in the atria, ventricles, and the pulmonary artery. Even though cardiac output is normal or low, stimulation of these mechanoreceptors in some people leads to a paradoxical withdrawal of peripheral sympathetic tone and increase in vagal tone.

2. **Cardiac Causes of Syncope**

   When a person stands, approximately 500 ml of blood is displaced to the abdomen and lower extremities, resulting in an abrupt fall in venous return to the heart. This leads to a decrease in cardiac output and stimulation of aortic and carotid baroreceptors that trigger a reflex increase in sympathetic outflow. As a result, heart rate, cardiac contractility, and vascular resistance increase to maintain a stable systemic blood pressure on standing. Orthostatic hypotension, which is a drop in systolic blood pressure of 20-mm or drop in diastolic blood pressure of 10-mm Hg within 3 minutes of standing, can result from a defect in any portion of this blood pressure control system.

   Orthostatic hypotension may be asymptomatic or may be associated with symptoms such as lightheadedness, dizziness, blurred vision, weakness, palpitations, tremulousness, and syncope. Drugs that either cause volume depletion or result in vasodilatation are common causes of orthostatic hypotension. Agents that effect autonomic system like Betablockers and Clonidine are common offending agents.

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Cardiac causes of syncope, particularly tachyarrhythmias and bradyarrhythmias, are common causes, accounting for approximately 20–30% of syncopal episodes. Though less frequent these disorders have an adverse prognosis. Among patients with left ventricular dysfunction, syncope occurs as a result of ventricular tachycardia. Supraventricular arrhythmias can also cause syncope, among patients with structurally normal heart. Bradyarrhythmias due to sick sinus syndrome as well as AV block can cause syncope.

Syncope may be the presenting manifestation of Acute Myocardial infarction in a small percentage of patients. Anatomical causes of syncope result from obstruction to blood flow, such as a massive pulmonary embolus, aortic stenosis, or hypertrophic cardiomyopathy. We feel Pulmonary thromboembolism or Primary PAH presenting as syncope is not uncommon in Indian population. Lack of awareness and investigatory facilities at peripheral centres, leads to under-recognition of this entity.

3. Neurological Causes of Syncope

Neurological causes of syncope include migraines, seizures, Arnold Chiari malformations and transient ischemic attacks. They are surprisingly uncommon causes of syncope, accounting for less than 10 percent of all cases of syncope. The majority of patients in whom a “neurological” cause of syncope is established are found in fact to have had a seizure rather than true syncope.

4. Metabolic/Miscellaneous Causes of Syncope

Metabolic causes of syncope are rare, accounting for less than 5% of syncopal episodes. The most common metabolic causes of syncope are hypoglycemia, and hyperventilation. Syncope in a diabetic is more often due to autonomic insufficiency rather than hypoglycemia.

Diagnostic Approach

The chief consideration in syncope is to identify the life-threatening causes of syncope from the more benign forms. Among the serious causes of syncope the most important are Visceral Bleeding, dysrhythmias and myocardial ischemia. Frequently encountered but equally serious are structural cardiac lesions, such as critical aortic stenosis, Dissection of aorta, pulmonary embolus. Subarachnoid hemorrhage can present as syncope, if untreated can prove to be fatal.

When the patient presents with a recent onset of syncope, it is important to rule out serious underlying conditions.

**Diagnostic Pathways**

Orthostatic Hypotension: BP has to be checked in supine position and 2 to 3 minutes after standing. Three types of response are seen.

1. **Normal Response**: BP drops or rises (especially in hypertensives) but the differences is usually less than 20 mmHg in systolic BP. (e.g.: supine 120/80, pulse 72 /mt, standing 130/80mm, pulse 84 /mt).

2. **Hypovolemic Response**: In this situation as the circulating blood volume is less, there will be a fall in systolic BP while standing. This triggers a sympathetic response and HR increases by > 20 bpm. In severe cases BP will drop by > 20 mm on standing. (e.g.: 130/80, PR 80 /mt, standing 100/80, HR 104 /mt)

3. **Autonomic Insufficiency**: (↓BP but insignificant rise in HR) In this situation when a person stands there is drop in systolic BP. There is inadequate sympathetic response (either due to drugs or autonomic insufficiency) or BP drops on standing, (eg; supine 110/70, pulse 78 /mt, standing 100/70, pulse 82 /mt). There is a failure of autonomic nervous system, and corresponding tachycardia does not occur.

Family history of sudden death (mother, cousin, etc.) is an important pointer to inherited syndromes of sudden cardiac death like long QT syndrome, Hypertrophic cardiomyopathy, Arrhythmogenic RV dysplasia etc. Patient with syncope, headache and diplopia needs an urgent neurological evaluation. Pain in the occipital region, neck may indicate subarachnoid hemorrhage.

Tachypnea / fall in oxygen saturation on pulse oximetry / Syncope in a Bed ridden patient post operative states – should raise the possibility of pulmonary embolism.

Asymmetric pulses and/or back pain should make one suspect dissecting aneurysm of aorta.

Elevated JVP suggests, right heart failure from PPH, myocardial ischemia, tamponade or pulmonary embolus.

Cardiac examination for murmurs of LV outflow obstruction and signs of pulmonary hypertension is important.

Abdominal tenderness or a mass in an elderly person points to visceral bleed (abdominal aortic aneurysm).

Among young women history of amenorrhea ,uterine spotting, adnexal tenderness may indicate a ruptured ectopic pregnancy.

Focal neurological signs indicate an underlying Neurological cause as the cause or consequence of syncope.

**Rapid Assessment/Stabilization**

The patient’s acute symptoms and status of vital signs dictate the need for immediate stabilization. Since syncope is by definition a transient event, most patients will be asymptomatic on presentation. If accompanied by normal or near-normal vital signs, such patients need no immediate stabilization, and a brief history and physical examination are performed. Every patient with the first episode of syncope, should have Orthostatic BP checked. Non cardiac conditions like ruptured ectopic pregnancy, GI Bleeds can present with syncope, if undiagnosed can prove fatal. Tenderness in the skull and deformity, hematoma of scalp warrants investigations. Intracranial bleeds as a consequence of syncope should not be missed.

**INVESTIGATIONS**

Detailed history and physical examination helps us to avoid a lot of unnecessary investigations in patients with syncope. Neuro imaging studies like MRI of Brain, CT Scan, and EEG do not provide useful data in most patients. Age related changes in Carotid Doppler or old infarcts in the Brain are often incidental findings and are not the underlying disease causing syncope.

**Hemoglobin and Hematocrit** are important to rule out occult blood loss. Soon after the bleed, patient may have a faint but hemoglobin may be normal. Acute bleed results in less of plasma and red cells to a similar extent. Hemoglobin levels drop only after dilution of plasma occurs.

Clinical examination may be unremarkable in certain conditions like Long QT Syndrome, Arrhythmogenic RV dysplasia, etc. Electrocardiogram can provide valuable diagnostic information and point to certain serious disorders which can lead to syncope. Signs of old myocardial infarction (pathological Q waves) on the ECG raises the possibility of ventricular arrhythmia as a cause of syncope. Conduction abnormalities (LBBB, ↑ PR interval), prolonged QT interval (QTc > 440 ms) are pointers to arrhythmic cause of syncope. Echocardiogram helps to identify structural heart disease (Left ventricular outflow obstruction, signs of pulmonary hypertension, RV dysplasia, old infarcts, tamponade, thoracic dissection) which may cause syncope.

If patient has an aura, prolonged confusional state after syncope, or any other focal neurological signs a detailed neurological evaluation and Neuro consultation may be warranted.

**Tilt Table Testing**

This is a useful test for diagnosing Neurocardiogenic syncope. A Specially designed table which can be tilted is used. Up to 40 minutes of Passive standing at 70 tilt is maintained. A continuous ECG and BP monitoring is done. In patients with Neurocardiogenic syncope, bradycardia and/or hypotension occurs with reproduction of symptoms. Provocative agents like Isoproterenol or Nitrate are used to unmask this response. However the specificity of this test is less when provocative agents are used.

**Carotid Sinus Massage**
Syncope due to carotid sinus hypersensitivity results from stimulation of carotid sinus baroreceptors, which are located in the internal carotid artery above the bifurcation of the common carotid artery. This condition is diagnosed by applying gentle pressure over the carotid pulsation just below the angle of the jaw, where the carotid bifurcation is located. Pressure should be applied unilaterally for approximately 5 seconds, after first listening for a carotid bruit. It has recently been reported that the sensitivity of diagnosing carotid sinus hypersensitivity can be increased, with no change in specificity, by performing carotid sinus massage during 60- or 70-degree upright tilt. The normal response to carotid sinus massage is a transient decrease in the sinus rate and/or slowing of atrioventricular conduction. Three types of abnormal responses have been described: (1) the cardioinhibitory response, characterized by marked bradycardia (0.3-second pause); (2) the vasodepressor type, characterized by a 50-mm Hg fall in the systolic blood pressure in the absence of bradycardia; and (3) the mixed response. Carotid sinus hypersensitivity is commonly seen in elderly patients. An abnormal response to CSM (i.e., Carotid Sinus Hypersensitivity, CHS) is not diagnostic of Carotid Sinus Syndrome (CSS). Reproduction of symptoms is a crucial diagnostic element. To achieve symptom reproduction, it may be useful to conduct CSM with the patient in the upright posture. If the latter is to be done, the patient should be safely secured to a tilt table in order to prevent injury from a fall.

Holter Monitor

Holter recordings if abnormal during the 24 hrs of recording may be useful. The yield from routine Holter for evaluation of syncope is limited. A normal Holter recording does not rule out serious arrhythmia as the cause of syncope.

Electrophysiological Study

Electrophysiological studies involves placement of intracardiac catheters inside the heart and induction of arrhythmias. In some patients onset of SVT or VT results in marked hypotension associated with syncope. As fainting occurs rapidly they do not feel or complain of palpitations. EF studies will be of help among Patients in whom arrhythmic causes are suspected.

- Presence of structural heart disease. (old MI with LV dysfunction, cardiomyopathy)
- Electrocardiographic Q waves
- Conduction abnormalities on ECG e.g. Complete LBBB.

Insertable Loop Recorder

In some patients with recurrent syncope, all the investigations (including tilt and EP study) may be negative. An Insertable Loop Recorder system offers long-term, continuous, subcutaneous ECG monitoring and event-specific recording. When a patient experiences an episode, the device stores an ECG by an auto-activating feature. This device can monitor continuously for up to 14 months. The probability of capturing an event is high—approximately 65-88%.

The ECG captured during the episode may “reveal” the ECG during the patient’s episode or may allow the clinician to rule in or rule out arrhythmic causes.

The stored ECG data can be retrieved and viewed.

Treatment

Treatment essentially involves treatment of the underlying cause. Arrhythmias need definitive therapy to prevent recurrence.

- Neurocardiogenic syncope is a common disorder and therapy involves some general measures. This involves
  - Ensure adequate hydration; consider TED stockings and salt tablets.
  - Eliminate medications that may induce hypotension.

Acute General Treatment

- Varies with the underlying etiology of syncope (e.g. pacemaker in patients with syncope secondary to complete heart block)

Sympotms are prolonged or recurrent, they are likely to have ventricular arrhythmias as the cause of syncope.
7. In patients with LV dysfunction syncope is often due to scar related VT. may recur despite revascularisation. Syncope and sudden death due to Ventricular arrhythmias continue to occur even after revascularisation. These patients need to be reevaluated for the risk of sudden death, 6-8 weeks after revascularisation.

REFERENCES