Syncope - A Clinical Point of View

SR Mittal

DEFINITION

Syncope - Sudden loss of consciousness and postural tone caused by transient decreased cerebral blood flow. There is immediate spontaneous recovery \(^1\) to appropriate behavior & orientation without retrograde amnesia.

Presyncope - Symptoms preceding syncope- These may include light headedness, dizziness (not true vertigo), nausea, feeling of warmth, diaphoresis and blurred vision \(^1\). Syncope may occur suddenly without warning or may be preceded by presyncope. Presyncope may recover spontaneously without advancing to loss of consciousness.

EVALUATION OF ETIOLOGY

- Correct evaluation is important because at times syncope may be the only warning sign before sudden cardiac death \(^2\).
- In up to 50% cases etiology may not be evident inspite of detailed evaluation \(^1\).
- In many cases, etiology may be multifactorial in the same episode or may be different in different episodes.
- Patient may have seizure at one time & syncope at other time.

HISTORY

Preceding

1. Physical exertion: Syncope during or immediately after exertion is likely to be cardiac in origin. Common causes include obstructive lesions eg- severe aortic stenosis, hypertrophic cardiomyopathy, Mitral stenosis, malfunction of prosthetic valves, severe pulmonary artery hypertension, TOF, intracardiac tumors. Children with anomalous coronary arteries and adults with critical coronary stenosis may develop syncope after effort. Patients with severe occlusive disease of brachiocephalic vessels eg- Aortic arch syndrome may also develop exertional syncope due to redistribution of cardiac output to exercising muscles resulting in relative cerebral ischemia. Some patients with conversion reaction may pretend fainting after effort.

2. Related to change of posture: Sitting to lying, bending, turning over in bed - intracardiac tumors, Ball valve thrombus.

3. Risk factors or clinical evidence of deep vein thrombosis - Pulmonary embolism.

4. History of critical coronary artery disease, old myocardial infarction, or left ventricular failure - Ventricular tachycardia.

5. History suggestive of acute MI - Ventricular tachyarrhythmia, onset of high degree AV block, transient severe sinus bradycardia.


7. Antiarrhythmic therapy, use of drugs that can prolong QT, drugs or diseases that can cause hypokalemia and/or hypomagnesemia - ventricular tachycardia.

8. History of permanent pacemaker implantation - Pacemaker malfunction.

9. Sudden standing from supine or squatting position - Orthostatic hypotension \(^2,3\). It is defined as a fall of more than 20mmHg in systolic BP on assuming upright posture. Syncope may occur with in seconds or may be delayed for 2-3 minutes. Rate of fall in BP is more important than BP reading in standing position. Rapid fall leads to failure of cerebral auto regulation resulting in syncope even when standing BP appears with in normal range. Such patients may have supine hypertension. Different persons may react
differently in response to a given magnitude of fall in BP. Elderly persons with compromised cerebral autoregulation are more susceptible. Some persons may only feel weakness, palpitation or tremulousness. Symptoms are usually worst immediately on arising in morning, after meal or after exercise. Orthostatic hypotension can be due to
a-Autonomic dysfunction.
b-Loss of intravascular volume.
c-Antihypertensive drugs.

[a] Autonomic dysfunction-
— Primary- Onset of symptoms is gradual & insidious with sensation of positional weakness, light headedness & dizziness.

Primary autonomic failure can have several clinical presentations-
(i)-Pure autonomic failure
(ii)-Multiple system atrophy
(iii)-Postural Orthostatic tachycardia syndrome
(iv)-Acute autonomic failure

(i) Pure autonomic failure: Additional features- Neurocardiogenic bladder (retention, incontinence), constipation, heat intolerance, anhidrosis, erectile dysfunction.

(ii) Multiple system atrophy:
— Striatonigral degeneration- additional muscle tremor similar to Parkinson disease.
— Olivopontocerebellar degeneration- additional cerebellar and/or pyramidal involvement.

(iii) Postural Orthostatic Tachycardia syndrome (4) - Disproportionate tachycardia without fall in blood pressure with in 5 minutes of standing.

(iv) Acute autonomic failure
— Secondary autonomic dysfunction: Diabetes, Amyloidosis, Sarcoidosis, Renal failure, Chronic alcoholism, Vitamin B-12 deficiency, spinal cord lesions & autoimmune disease eg. Guillain Barre Syndrome, SLE, Rheumatoid arthritis, mixed connective tissue disease.

In disautonomic syncope, fall in blood pressure tends to be slow and may produce a drop attack some time after assuming upright posture rather then immediately on standing. There may be little or no prodromal symptoms specially in elderly. There may be no diaphoresis due to autonomic failure. For the same reason, these patients may have relatively fixed heart rate between 50-70beats/min.

10. Shaving, tight collar, turning head to one side — carotid sinus hypersensitivity

11. Situational syncope-
a- Rapid emptying of distended bladder, or rectum
b- Rapid aspiration of pleural effusion, pericardial effusion or ascitis
c- Painful stimulus (needle prick) or unpleasant sight, sound or smell, sight of blood
d- Swallowing syncope
e- Prolonged bout of cough
f- Hot shower
g- Neuralgia- Glossopharyngeal, Trigeminal


13. Upper extremity exercise- Subclavian steal syndrome- Exceedingly uncommon cause of syncope. Major occlusive disease of Subclavian artery proximal to origin of vertebral artery can result in shunting of blood retrogradely from circle of Willis to the distal part of occluded Subclavian artery via ipsilateral vertebral artery.

14. Preceding vertigo, diplopia, dysarthria & ataxia - Vertebrobasilar system insufficiency, basilar artery migrain

15. Post prandial syncope can occur in elderly around one hour after a meal specially following a high carbohydrate diet. This is due to redistribution of cardiac output to splanchnic circulation.

16. Family history of syncope or sudden death- cardiogenic

Observation during an episode ²/ Interogation of a witness

<table>
<thead>
<tr>
<th>Age</th>
<th>Support possibility of</th>
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<tbody>
<tr>
<td>Children</td>
<td>- Cardiogenic</td>
</tr>
<tr>
<td>Elderly</td>
<td>- Orthostatic hypotension</td>
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<tr>
<td></td>
<td>- Postprandrial hypotension</td>
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</tbody>
</table>
- Clinical setting
  - Standing, warm room, emotional upset
  - Old age, during or soon after exercise
- Premonitory symptoms
  - Palpitation, Blurring of vision, Nausea, Warmth, diaphoresis, light headedness
  - Sudden
- Clinical findings
  - Pallor, Diaphoresis
  - Absence of diaphoresis
  - Blue face
  - Frothing at mouth, tongue bite, horizontal deviation of eyes
  - Tonic & clonic movements
- Inconvenience
  - Absence of major arterial pulse
  - Bradycardia
- Tachycardia
  - Hypertension (5)
  - Lack of responsiveness in absence of loss of postural tone
  - Confusion, change in level of consciousness, flushing
  - Throbbing unilateral headache, scintilating scotoma
- Recovery
  - Within seconds
  - Prolonged confusion, disorientation & retrograde amnesia
  - Orientation - normal

cause

**Physical examination**

- BP & Heart rate- Supine, sitting, immediately on standing, 5 minutes after standing >20mmHg fall in SBP and or > 10mmHg fall in DBP- Orthostatic hypotension
- Cardiac auscultation combined with appropriate physical maneuvers- structural heart disease

- Medications
  - Aortic stenosis
  - Carotid sinus hypersensitivity
  - Bradyarrhythmia
  - Neurally mediated hypotension
  - Cardiogenic syncope

- Carotid sinus massage-Pause of >3 seconds-possibility of carotid sinus hypersensitivity.

It is important to remember that some less common but potentially lethal causes such as long QT syndrome, arrhythmogenic RV cardiomyopathy, Brugada syndrome, idiopathic VF, catecholinergetnic polymorphic VT, short QT.
INVESTIGATION OF CHOICE

**Suspected etiology**

1. Anemia
   - Hemoglobin (Hb), Packed cell volume (PBF)
2. Electrolyte imbalance
   - Serum K, Mg
3. Myocardial infarction, Sick sinus syndrome, AV block, Accessory pathway, Short QT, Long QT, Brugada syndrome, Arrhythmogenic RV cardiomyopathy (ARVC)
4. LV dysfunction, Hypertrophic cardiomyopathy, AS, MS, Congenital heart disease, Pulmonary artery hypertension, ARVC
5. Exercise induced ischemia or arrhythmia
   - Stress rest, avoid in severe AS or HOCM
6. Frequent arrhythmias (at least once or more/day)
   - Ambulatory ECG monitoring
   - To correlate symptoms to arrhythmia
7. Carotid sinus hypersensitivity
   - Carotid sinus pressure - gentle pressure below angle of jaw for 5-10 seconds in supine as well as upright position. Monitor ECG & BP. Avoid if carotid bruit, prior TIA, stroke within preceding 3 months. The test is, however, not specific. It is also commonly observed in asymptomatic elderly.
8. Neurocardiogenic syncope & unexplained recurrent syncope
   - Head up tilt table test, 70 inclination for 30-45 minutes
   - BP, Pulse, response
     - Abrupt fall, Bradycardia, Vasovagal
     - Abrupt fall, No change, Vasodepressor
     - Gradual fall, No change, Dysautonomic
     - No change, ↑ by >30/min. or >120/min. in first 5 min., Postural Tachycardia
     - No change, No change, Cerebral hypoxia on EEG
     - No change, No change, Cerebral Vasocostriction on transcranial doppler
     - No change, No change, No change in ECG & transcranial doppler, Psychogenic

Use of provocative agents (isoproterenol infusion, S/L Nitroglycerine) significantly decreases specificity

9. Infrequent arrhythmia
   - Event recorder (30days)
10. Very infrequent arrhythmia
    - Implantable loop recorder
11. Undiagnosed etiology in high risk patients eg: Structural heart disease, suspicious arrhythmias by ECG monitoring, recurrent syncope, high risk jobs, BBB, bifascicular block
    - Electrophysiologic study to assess Sinus and AV node function, Susceptibility to supraventricular & ventricular tachyarrhythmias Limitation
    - Low sensitivity, low positive predictive value
    - Microvolt T wave alternass (6)
    - Prognostic utility under evaluation

Rhythm changes observed during evaluation may not be responsible for different episode of syncope.
syndrome & HCM may be missed on clinical evaluation alone.²

**PREVENTION**

1. **Counseling**
   
   To avoid factors that are likely to precipitate syncope

   **I. Orthostatic intolerance:**
   - Elevation of head end of bed by six inches
   - Move feet & legs prior to rising from bed
   - Rise slowly & stepwise. Supine  Sitting  Standing
   - Tilt training- standing against wall for 40 minutes twice a day

   **II. Neurocardiogenic syncope:**
   - Avoid prolonged standing at one place & working in hot weather
   - Periodically flex & extend feet & legs
   - Maintain salt & water intake specially when going out in hot weather
   - Isometric hand grip or leg squeezing with onset of prodromal symptoms.

   **III. Other causes:**
   - Avoid factors that are likely to precipitate syncope eg- exercise, tight collar, hyperextension of neck, sudden turning of neck, overdistension of urinary bladder.
   - Avoid situation where patient himself or others could be injured by unconsciousness eg.- driving, swimming. This is specially important if syncope is frequent
   - Lie down immediately to avoid unconsciousness/ injury if there are prodromal symptoms
   - Avoid prolonged immobilization to prevent deep vein thrombosis or deconditioning

2. **Non pharmacologic measures**
   - Elastic compression stocking - waist height
   - Minimum of 30mmHg of ankle counterpressure

   **II. Cervical physiotherapy, traction, collar for cervical bone diseases**

3. **Pharmacotherapy**
   - I. Optimise dose of antihypertensives, other drugs causing postural hypotension (eg- tricyclic antidepressants), bradycardia (eg- β blockers), prolongation of QT (eg-antiarrhythmics), hypokalemia, hypomagnesemia.

   **IV. Other drugs:**
   
   **a) Neurocardiogenic syncope**

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<thead>
<tr>
<th>Mechanism of action</th>
<th>Drug</th>
<th>Limitation</th>
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<tbody>
<tr>
<td>i) Reduction of cardiac mechanoreceptor β blockers activation</td>
<td>eg- Metoprolol (7)</td>
<td>Fatigue, cold periphery, bronchospasm</td>
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<td>ii) Increase salt &amp; water retention</td>
<td>Mineralocorticoid</td>
<td>Hypokalemia, hypomagnesemia, oedema</td>
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<td>iii) Peripheral vasoconstriction - α-agonists</td>
<td>eg-Midodrine</td>
<td>Nausea, scalp itching, supine hypertension</td>
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<td>iv) Norepinephrine &amp; Dopamine reuptake inhibitors</td>
<td>eg Clonidine</td>
<td>Dry mouth, Blurred vision</td>
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<tr>
<td>v) Acetylcholinesterase inhibitors</td>
<td>eg Pyridostigmine(8)</td>
<td>Hypertension, Agitation Agitation</td>
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<tr>
<td>vi) Splanchnic mesentric vasoconstriction - ↑ venous return</td>
<td>Octreotide</td>
<td>Nausea, Diarrhoea Gall stone</td>
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<tr>
<td>iv) Serotonin reuptake inhibitors</td>
<td>Escetalompram, Fluoxetine, Venlafaxine, Paroxetine</td>
<td>Tremor, Agitation, sexual problem, Expulsive</td>
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<td>v) Correction of anemia</td>
<td>Erythropoietin</td>
<td>Expensive</td>
</tr>
<tr>
<td>vi) Dilatation of pulmonary vasculature in PPH</td>
<td>Bosentan</td>
<td>efficacy not documented</td>
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Long term trials have failed to confirm beneficial effects seen in smaller trials.⁹

All drugs are not effective in all patients suggesting that our understanding of pathophysiology of syncope is still incomplete.
II. Prophylactic use of Atropine prior to any fluid aspiration, biopsy or invasive procedure.

III. Use of anticoagulants and/or platelet aggregation inhibitors in patients susceptible to thromboembolism—e.g. risk factors for DVT, gross systolic heart failure, MS, atrial fibrillation.

CONCLUSIONS

Syncope is a symptom complex that can range from mild discomfort to clear loss of consciousness. Most of the times patients present with history. Usually the patient is not examined during episode. Multiple factors work in combination even in persons who apparently have single etiology. Labeling of underlying etiology is difficult in nearly 50% patients. Implantable loop recorders may reveal useful information in patients with unidentified etiology. Self-injury is always a risk even when etiology looks benign. Autonomic failure & arrhythmias with structural heart disease have worse prognosis. Dysautonomic syncope is associated with multisystem involvement and may require multidisciplinary approach. In spite of advances in our understanding, management is usually empirical. Permanent pacemaker implantation or implantable cardiovertor defibrillator may help some patients with unidentified etiology & drug failure.

REFERENCES