

Syncope

Summary from Rosen's By Ian Bodford

Epidemiology

- Prevalence is 19% in general population. 32% are admitted from ED. Persons older than 65 are 80% of those admissions.
- Risk factors are cerebrovascular disease, cardiac medications, and hypertension. Most causes are benign, especially in young adults and adolescents.

Pathophysiology

- Transient dysfunction of bilateral cerebral hemispheres or the brainstem (RAS), usually from hypoperfusion.
- Other mechanisms include hypoglycemia, toxins, metabolic abnormalities, failure of autoregulation, and 1° neurologic derangements.

Differential Diagnosis

- Huge array of causes. Must distinguish from other causes of apparent sudden LOC (seizures, cataplexy, etc.).
- Three categories of causes
 - Focal hypoperfusion of CNS structures ☒ cerebrovascular disease, hyperventilation, subclavian steal, SAH, basilar artery migraine, and cerebral syncope
 - Systemic hypoperfusion resulting in CNS dysfunction ☒ outflow obstruction, reduced cardiac output, vasomotor, carotid sinus sensitivity, miscellaneous reflexes (sneeze, postmicturition, etc.), orthostatic hypotension, anemia, & drug-induced
 - CNS dysfunction with normal cerebral perfusion ☒ hypoglycemia, hypoxemia, seizure, narcolepsy, psychogenic, and toxic (drugs, CO, etc.)
- Must rule out life-threatening causes first, i.e. dysrhythmias, MI, stroke, etc.

Signs and Symptoms

- Character of syncope ☒ rate of onset, position at onset, duration, and rate of recovery
- Events preceding syncope ☒ exertion, heat exposure, emotional state, micturition, neck stimulation, palpitations, or aura
- Events during episode ☒ tonic-clonic movements or trauma during fall
- Associated symptoms ☒ chest pain, dyspnea, diaphoresis, light-headedness, graying of vision, tongue biting, incontinence
- PMHx ☒ CHF, CAD, CVA, diabetes, HTN, medications causing syncope
- Signs ☒ orthostasis, carotid massage, rectal examination for blood

Work-up

- EKG in all patients unless patient is adolescent with clear vasovagal origin.
- If benign dysrhythmia is suspected but not seen on EKG, can use a Holter monitor as outpatient. If severe dysrhythmia is suspected, consider echo, continuous EKG, or stress testing before discharge.
- CBC, BMP with glucose, UDS, and urine pregnancy test when warranted by history and exam. CXR and BNP if CHF suspected. CT of head only when SAH is suspected. EEG if seizure is suspected. Orthostatic vital signs should be performed but may be unreliable to assess volume status. Carotid sinus massage could potentially elicit presyncope/syncope.

Empiric Management

- By definition, syncope is a transient event and most patients do not have symptoms in the ED. Patients with non-emergent diagnoses can be treated as outpatients.
- San Francisco Syncope Rule ☒ outpatient disposition considered in patients without abnormal EKG findings, shortness of breath, hypotension, anemia, or history of heart failure. Safety and efficacy of this rule have not been established.
- Patients with an abnormal EKG, abnormal vital signs, recurrent syncope, or concerning associated symptoms (chest pain, hypotension, etc.) should be stabilized in the ED and admitted to the ICU or a bed with telemetry.

Disposition

- Unfortunately, the ED evaluation of syncope is often inconclusive. Up to 50% will not have a diagnosis at discharge.
- Patients less than 45 years old without worrisome symptoms, signs, or EKG findings may be treated as outpatients.
- Discharged patients should be warned of the hazards of recurrent syncope occurring during activities such as driving or working at heights. Recurrence may be as high as 50%.