EVALUATION AND MANAGEMENT OF SYNCOPE

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DEFINITION OF SYNCOPE

A sudden and transient loss of consciousness associated with loss of postural tone
SYNCOPE

- Accounts for 3% of all emergency room visits
- Accounts for 6% of a hospital’s medical admissions
- Up to 20% of adults have one episode by age 75
ONE YEAR MORTALITY RATE IN PATIENTS WITH SYNCOPE

- 20 to 30% with cardiac syncope
- 5% for non cardiac syncope
- 10% for unexplained syncope
CARDIAC SYNCOPE-MECHANICAL/OBSTRICTIVE

- Aortic stenosis
- Hypertrophic cardiomyopathy
- Pulmonary embolism
- Cardiac tamponade
- Aortic dissection
- Tetrology of Fallot
- Pulmonary hypertension
- Atrial myxoma
CARDIAC SYNCOPE - ARRHYTHMIC

- AV block with bradycardia
- Sinus node dysfunction with bradycardia
- Supraventricular tachycardia
- Ventricular tachycardia
- Long QT syndrome
REFLEX SYNCOPE

- Vasovagal
- Neurocardiogenic or neurally mediated
- Vasovagal (situational)
- Micturiton
- Deglutition
- Defecation
- Post-tussive
- Post-prandial
- Carotid sinus syncope
Failure of sympathetic efferent vasoconstrictor traffic (and hypotension) occurs episodically and, frequently in response to trigger.

In chronic autonomic failure, sympathetic efferent activity is chronically impaired.
DIAGNOSTIC EVALUATION - HISTORY

- Premonitory symptoms
- Supine or during sleep
- During/ after exercise
- Positional changes
- Family history
- Medication
DIAGNOSTIC EVALUATION - PHYSICAL

- Orthostatic vital signs
- Carotid sinus massage
- Cardiac exam
DEFINITION OF ORTHOSTATIC HYPOTENSION

- After 2-5 minutes of quiet standing
- At least a 20 mmHg fall in systolic pressure
- At least a 10 mmHg fall in diastolic pressure

Symptoms of cerebral hypoperfusion
ORTHOSTATIC

- Dysautonomia
- Volume depletion
- Illness, bedrest
- Postural tachycardia syndrome
- Drugs
CAROTID SINUS MASSAGE

- Recommended in patients over age 40 years with syncope of unknown etiology
- EKG monitoring and continued blood pressure measurements during CSM is mandatory
- Duration of CSM is a minimum of 5 and a maximum of 10 sec
- Should be performed with the patient both supine and erect
DEFINITION OF POSITIVE CAROTID SINUS MASSAGE

- Symptoms are reproduced during or immediately after massage, in the presence of asystole longer than 3 sec and/or a fall in blood pressure of 50mmHg or more

- A positive response is diagnostic of the cause of syncope in the absence of any other competing diagnosis
CARDIAC EXAM

- Mitral valve prolapse
- Aortic stenosis
- Left ventricular hypertrophy
- Left ventricular outflow tract obstruction
ELECTROCARDIOGRAM FINDINGS

- AV block
- Right or left bundle branch block
- Prior MI
- Pre-excitation
- Long QT interval
**IMAGING STUDIES**

- Chest x ray – identify pneumonia, CHF, lung mass, effusion or widened mediastinum
- CT of the head-low diagnostic yield in syncope but may be helpful in patients with head trauma or new neurologic deficits
- CT of chest and abdomen-indicated only in select cases (aortic dissection, ruptured AAA, or pulmonary embolism
Ventilation-perfusion scan - suspected PE

Echocardiography - test of choice for suspected mechanical cause of syncope
EKG MONITORING

- Holter monitor-use for patients with daily symptoms and/or when looking for high grade AV block
- Event monitoring-use for patients monthly symptoms
- Implantable loop recorder-use when event recorder or EPS is unrevealing
OTHER STUDIES

- Head up tilt test

- EEG – at discretion of neurologist if seizure is considered likely

- Stress test-cardiac syncope suspected and patient has risk factors for CAD
DIFFERENTIATING CARDIAC SYNCOPE VERSUS

- Syncope surrounding activity - 65% vs. 18%
- Family history of cardiac disease or sudden cardiac death - 41% vs. 25%
- Abnormal physical findings suggesting a cardiac diagnosis - 29% vs. 0%
- Abnormal EKG - 76% vs. 0%

Tretter et al Dec 2013
Triggering of a neural reflex which results in a usually self limited episode of systemic hypotension characterized by both bradycardia (asystole or relative bradycardia) and peripheral vasodilatation.
PATHOPHYSIOLOGICAL MECHANISMS

- Venous pooling in lower limbs reduces ventricular volume and increases sympathetic activity
- Vigorous myocardial contraction stimulates ventricular mechanoreceptors
- Triggering of an inhibitory reflex which causes paradoxical sympathetic withdrawal and vagal over-activity
NEUROCARDIOGENIC SYNCOPE

- Usually young patients who are otherwise healthy
- Prodrome of nausea, warmth, pallor, lightheadedness and/or diaphoresis
- Induced by prolonged standing, venipuncture (experienced or witnessed), heat exposure, painful or noxious stimuli, fear of bodily injury or exertion
- Older patients may not have an identifiable cause
PROVOCATIVE FACTORS

- During or immediately after exercise
- During or immediately after urination, defecation, coughing or swallowing
- During prolonged standing
- During the post-prandial period
- In association with emotional stress, fear or intense pain
DRUGS THAT EXACERBATE NEUROCARDIOGENIC SYNCOPE

- Alcohol
- Diuretics
- Ace inhibitors
- Nitrates
- Tricyclic antidepressants
- Nifedipine
- Prazosin
TILT TEST PROTOCOLS

- Prolonged standing (30-60 min) without medication provocation
- Abbreviated standing (10-15 min) with isoproterenol challenge
- Abbreviated standing (10-15) with nitroglycerin challenge
RESPONSES TO TILT TESTING

- Normal - no hypotension or bradycardia
- Mixed response - vasodepressor and cardioinhibitory (most common)
- Pure vasodepressor (elderly)
- Pure cardioinhibitory (rare)
TREATMENT OF NEUROCARDIOGENIC SYNCOPE

- Avoid predisposing factors-extreme heat, dehydration
- Remove underlying causes-drugs
- Interrupt any part of the cascade of events associated with the development of syncope with drug therapy or pacing
- Behavior modification-high salt, caffeine free diet
PHYSICAL COUNTERPRESSURE

- Leg crossing with simultaneous tensing of leg, abdominal and buttock muscles

- Handgrip – maximum grip on rubber ball or similar object

- Arm tensing – gripping one hand with the other while abducting both
DRUGS TO TREAT NEUROCARDIOGENIC SYNCOPE

- Beta blockers - may have high placebo effect
- Midodrine
- Fludrocortisone
- Serotonin reuptake inhibitors
RATIONALE FOR BETA BLOCKERS

- Catecholamines are often elevated prior to a syncopal episode.
- Beta-blockers can antagonize the neuroautonomic reflex in response to this increase.
- Beta-blockers reduce myocardial contractility and can prevent or reduce the frequency of ventricular contractions that activate mechanoreceptors.
- Beta 2 adrenergic blockers can prevent a reduction in systemic vascular resistance.
MIDODRINE

- Selective, peripherally acting alpha agonist
- Causes arteriolar constriction and decreased venous capacity
- Decreases plasma volume, redistributing plasma into the extra cellular space and thereby decreasing peripheral venous pooling
FLUDROCORTISONE

- Mineral corticoid used to treat orthostatic hypotension
- Causes sodium retention thereby maintaining vascular volume and venous return
- May prevent vigorous contraction caused by under filling of the ventricle that activates mechanoreceptors
Elevated serotonin levels have been shown to reduce central nervous system sympathetic activity and cause hypotension and bradycardia.

Elevated serotonin levels may suppress the baroreceptor reflex.
INDICATIONS FOR PACING IN NEUROCARDIOGENIC SYNCOPE

Carotid sinus hypersensitivity-syncope and greater than 3 seconds of asystole followed by minimal carotid sinus massage
INDICATIONS FOR PACING IN HCSS AND NCS-CLASS IIA

- Recurrent syncope without clear, provocative events and a hypersensitive cardioinhibitory response
- Significantly symptomatic and recurrent NCS associated with bradycardia documented spontaneously or at the time of tilt table testing
Hyperactive cardioinhibitory response to CSM in the absence of symptoms or in the presence of vague symptoms (dizziness)

Recurrent syncope, lightheadedness, or dizziness in the absence of a hyperactive cardioinhibitory response

Situational syncope in which avoidance behavior is effective
Subcutaneously implanted between 3rd and 4th intercostal space

Programmed to detect asystole greater than 3 seconds, bradycardia <35 BPM, tachycardia> 150 BPM
AV BLOCK IS EVIDENT ON RECORDING
POSTURAL TACHYCARDIA SYNDROME (POTS)

- Form of orthostatic intolerance
- Symptoms occur in response to postural change
- Autonomic reflexes are relatively preserved
- Hallmark is exaggerated heart rate increase in response to postural change
POSTURAL TACHYCARDIA SYNDROME

- Most prevalent form of orthostatic intolerance
- 500,000 Americans suffer from this disorder
- Most common syndrome of young people seen in autonomic dysfunction clinics
- Patients present at a young age

POSTURAL TACHYCARDIA SYNDROME

- Women predominate with a female to male ratio of 4-5:1

- May be due to gender differences in muscle sympathetic nerve discharge characteristics and decreased stroke volume

- Etiology is heterogeneous
CLINICAL FEATURES OF POTS

- Dizziness, blurred vision and fatigue upon standing
- Palpitations, tremulousness and anxiety
- Nausea, cramping, early satiety, constipation, diarrhea
- Venous pooling with acrocyanosis and edema when upright
Syncope in 40% of patients

Chronic headaches sometimes exacerbated by postural change

Symptoms may appear abruptly often after a viral illness

Severity of symptoms is variable

Ojha Am J Med 2010;123:245
DIAGNOSIS OF POTS

- Exaggerated increase in heart rate on tilt testing or with standing
- Sustained heart rate increase of 30 BPM or an increase to 120 BPM or greater within the first 10 minutes of tilt
- Usually no orthostatic hypotension
POSSIBLE ETIOLOGIES OF POTS

- Distal denervation - underlying autonomic neuropathy that may be post viral or immune-mediated
- Hypovolemia - patients improve with fluids
- Changes in venous function
- Cardiovascular deconditioning
POSSIBLE ETIOLOGIES OF POTS

- Baroreflex abnormalities
- Increased sympathetic activity
- Genetic abnormalities
- Chronic fatigue syndrome and mitral valve prolapse have overlapping features
DIFFERENTIAL DIAGNOSIS OF POTS

- Autonomic neuropathies
- Central dysautonomias
- Bedrest deconditioning
- Medication side effects
- Dehydration
TREATMENT OF POTS

- Optimal therapy is not established

- Non-pharmacologic measures – avoid exacerbating factors like medications, dehydration and inactivity

- Exercise training

- Fludrocortisone

- Midodrine

- Pyridostigmine
THE HISTORY IS THE MOST IMPORTANT TOOL IN THE DIAGNOSIS OF SYNCOPE

STRUCTURAL HEART DISEASE MUST BE RULED OUT SINCE THE MORTALITY IS HIGHEST IN CARDIAC SYNCOPE

NEUROCARDIOGENIC SYNCOPE IS THE MOST COMMON CAUSE OF SYNCOPE IN OTHERWISE HEALTHY PEOPLE
SUMMARY

- Treatment depends on the frequency and severity of symptoms as well as the patient’s occupation.
- Sometimes patient education and behavior modification are sufficient for treatment.