Evaluation and Management of Syncope

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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/obstructive

- 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)
- Micturition
- Deglutition
- Defecation
- Post-tussive
- Post-prandial
- Carotid sinus syncope
Reflex 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
- 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)
Imaging studies -continued

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 neurocardiogenic syncope

-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
Neurocardiogenic Syncope

• 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
Pathophysiologial Mechanisms

- Venous pooling in lower limbs reduces ventricular volume and increases sympathetic activity
- Vigorous myocardial contraction stimulates ventricular mechanoreceptors
- Triggering of a inhibitory reflex which causes paradoxical sympathetic withdrawal and vagal overactivity
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
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)
After 3 minutes of Head-up Tilt

I

II

BP

mHg

0

100

aVL

100

mHg

0
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 extracellular 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
Selective serotonin reuptake inhibitors

- 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- Class I

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
Class III-not indicated

- 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
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
CASE STUDY
Case study

- 55 year old woman with recurrent syncope and hypertension

- Positive tilt test 2002 initially controlled on beta blocker therapy

- Beta blocker was chosen because she was also hypertensive
Case study

-Patient remained asymptomatic until 2012
-Had syncope while talking on the phone, noted fast heart beat before she fainted, also had some chest pain
Work-up

Lexiscan nuclear –negative for ischemia

Echo- normal EF, stage III-VI diastolic dysfunction, prolapse on anterior leaflet of the mitral valve, mild mitral regurgitation
EP work up

Repeat tilt test – normal on current therapy
EP study – normal conduction intervals, normal sinus node and AV nodal function, no inducible arrhythmias or high grade AV block
Loop recorder implanted

Subcutaneously implanted between 3rd and 4th intercostal space
Programmed to detect asystole greater than 3 seconds, bradycardia <35 BPM, tachycardia> 150 BPM
Implantable loop recorder
Reveal XT size compared to Reveal LinQ
AV Block is evident on recording
Dysautonomia

- Disorder of autonomic nervous system
- Failure of sympathetic or parasympathetic components of the ANS
- Can be local, as in reflex sympathetic dystrophy or generalized as in pure autonomic failure
- Can be acute and reversible as in Guillan-Barre syndrome or chronic and progressive
- Diabetes and alcoholism can include it
- Can occur as a primary condition or in association with degenerative neurological disease such as Parkinson’s disease
Hallmarks of dysautonomia

- Sympathetic failure - Impotence (in men), orthostatic hypotension
- Excessive sympathetic activity - hypertension or rapid heart rate
Treatment of dysautonomia

• No cure

• Secondary forms may improve with treatment of underlying disease

• Primary dysautonomia treatment includes measures to combat orthostatic hypotension including frequent small meals, elevate head of bed, high salt diet, fludrocortisone and midodrine
Guillain Barre Syndrome

- Acute inflammatory Demyelinating Polyneuropathy
- Acute autoimmune Polyneuropathy
- Acute idiopathic polyneuritis
- Idiopathic polyneuritis
Guillain Barre and variants

• Antibodies in the blood affect the autoimmune system
• Risk factors include GI or respiratory viral infection
• Ages 15-35 and 50-75
• Surgery
• Vaccination
Guillain Barre and variants

- Weakness or tingling in legs spreading to upper body
- Restricted muscle use
- Paralysis/Difficulty breathing
- High or low blood pressure
- Abnormal heart rate
Chronic Inflammatory Demyelinating Polyneuropathy

• Rare disorder

• Inflammation of nerve roots and peripheral nerves and destruction of myelin sheaths

• Symptoms of weakness, paralysis and/or impairment of motor function especially of limbs

• Sensory disturbances

• May affect only the autonomic nervous system
Chronic Inflammatory Demyelinating Polyneuropathy

• In contrast to Guillain Barre patients with CIPD cannot identify a preceding viral or infectious illness
Treatment of Guillain Barre and variants

• Plasmapheresis
• Immunoglobulin therapy
• Not benign treatment
• Refer to neurologist for definitive diagnosis
Summary

• 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