

Syncope

Morning Report

March 2006

Introduction

- The abrupt loss of consciousness associated with the absence of postural tone; followed by a rapid and complete recovery
- Loss of cerebral oxygenation and perfusion is usual mechanism
- 3% of ED visits and 6% of hospital admissions in adults
- Among children, only 0.125% of ED visits
- 47% of college students report having fainted
- 15% of children suffer from syncope before the end of adolescence

Etiology

- Cardiac
- Noncardiac
- Neurocardiac (vasovagal)
- 75% of children who faint have neurocardiogenic syncope due to neurally mediated hypotension and bradycardia
- Cardiac syncope is more rare but can be life-threatening and deserves careful attention

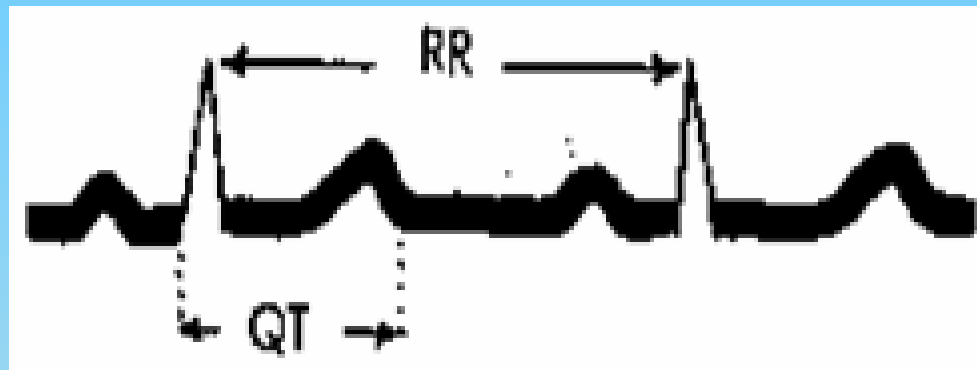
Cardiac Syncope

- Arrhythmogenic or structural heart conditions must always be considered in the differential
- Arrhythmias:
 - congenital (CHD, prolonged QT)
 - acquired (ARF, myocarditis)
 - following surgical correction of CHD
 - electrolyte abnormalities
 - medications (digitalis, TCAs, theophylline, antiarrhythmics, and illicit drugs)

Prolonged QT

- Predisposed to episodic ventricular arrhythmias, torsade de pointe, syncope, and generalized seizures
- Can be secondary to acquired heart disease (myocarditis, MVP, electrolytes, drugs) but more often is congenital
- Jervell and Lange-Nielsen syndrome has associated sensorineural deafness and prolonged QT
- Romano-Ward syndrome is AD form of prolonged QT and is not associated with deafness
- Multiple genes have been identified for the syndrome and the genotype influences the clinical course

QT INTERVAL



The QTc is calculated as:

$$QT_c = QT \text{ (sec)} / \sqrt{RR \text{ (sec)}}$$

The R-R interval should extend from the R wave in the QRS complex in which you are measuring QT to the preceding R wave. Normal values for QTc :

0.440 sec is 97th percentile for infants 3 to 4 days old[6]

≤0.45 sec in infants <6 months old

≤0.44 sec in children

≤0.44 sec in adults

Other Arrhythmias

- WPW: shortened PR interval and delta wave on proximal part of QRS complex; predisposes to re-entrant SVT via abnormal pathway
- Congenital complete AV block: infants with maternal SLE
- Complete heart block: RA, dermatomyositis, Sjogren, complex CHD, abnormal embryonic conduction system, myocardial tumors, myocarditis, sick sinus syndrome (following surgical repair of transposition with Mustard)
- Arrhythmogenic right ventricular dysplasia

Syncope: Structural Heart Disease

- Familial hypertrophic cardiomyopathy/ Idiopathic hypertrophic subaortic stenosis- may present with exercise-induced syncope, chest pain, palpitations
- Factors associated with increased risk of sudden death include: FH of sudden death, clinical symptoms, young age, presence of ventricular arrhythmia, and a thickened intraventricular septum
- ECG shows LVH, ST-T wave changes, deep and wide Q waves in left precordial leads
- Echo can judge severity and progression of cardiomyopathy

Other Structural Heart Diseases

- Severe pulmonary or aortic stenosis
- Anomalous origin of the left coronary artery
- Pulmonary hypertension
- Coronary aneurysms complicating Kawasaki disease
- Myxomas in interatrial septum (obstruction through mitral valve)
- All may induce syncope with exercise

Noncardiac Syncope

- Seizures can usually be distinguished by history and physical exam and are not true syncope – can result from the syncopal episode
- Hyperventilation can produce cerebral vasoconstriction
- Atonic seizures (drop attacks) – sudden loss of antigravity muscle tone; can be manifestation of Lennox-Gastaut syndrome
- Vertebrobasilary vascular spasm in migraines can cause syncope; HA persists after episode
- Orthostatic hypotension: pregnancy, bedrest, drugs
- Situational syncope: cough, micturition, defecation
- Hypoglycemia and electrolyte abnormalities
- Cataplexy: LOC in response to emotional reactions, associated with narcolepsy

Breath-Holding Spells

- Very common
- Occurs between ages 1-5
- Rare prior to 6 months of age, peaks at age 2 years
- 80% cyanotic form (classic)
- 20% pallid form

Cyanotic "Classic" Breath-holding

- Prodromal period of crying followed by forced expiration and apnea
- Syncope likely due to involuntary activation of Valsalva
- Increasing thoracic pressure, interferes with venous return to the heart, decreased CO, unconsciousness, loss of muscle tone
- Can be associated with generalized clonic jerks, opisthotonos, and bradycardia
- EEG normal
- Reassure and explain mechanism to parents

Pallid Breath-holding Spells

- Initiated by innocuous stimulus, such as frustration at play, scolding, painful experience or a sudden startle
- Initial quieting, breath-holding in the end-expiratory phase, followed by pallor, brief LOC, loss of muscle tone, and a fall to the ground
- Often parent arrives to a still child, with a fixed stare and perioral cyanosis
- Tonic rigidity and clonic jerking may occur
- Abnormal vagal responses
- EEG normal, ocular compression test with bradycardia and a 3 s period of asystole can be elicited in more than 50%

Psychogenic Syncope

- Suspect when episode is prolonged, no change in vital signs or appearance, does not raise concern in patient, or patient's recall or responsiveness during the event suggests that consciousness has been maintained
- Episodes often occur in front of audience
- Occurs primarily in adolescents
- Patient often falls gently, without injury
- Patient calmly relates specific details of the episodes

Neurocardiogenic (Vasovagal) Syncope

- Most common form in children
- Often associated with orthostatic intolerance
- Mechanism is reflex mediated and originates from a decreased systemic venous return that leads to decreased left ventricular end diastolic volume
- Increased mechanical contractility results in stimulation of cardiac vagal fibers and a paradoxical response of marked bradycardia, vasodilation and hypotension

Neurocardiogenic Syncope

- 3 clinically recognized forms:
 - Cardioinhibitory (primary bradycardia with subsequent hypotension)
 - Vasodepressor (hypotension but no bradycardia)
 - Mixed (hypotension and bradycardia)
- Nausea, vomiting, sweating, lightheadedness typically precedes the episodes
- Treat with fluid therapy as primary therapy
- Other therapies: increase salt intake or volume expansion with salt tablets or fludrocortisone, and psuedoephedrine

Evaluation

- ECG should be part of evaluation of all patients who present with syncope
- Cardiology consult indications:
 - Pathologic murmur
 - Chest pain preceding syncope
 - Arrhythmia or prolonged QT is suspected
 - Q waves on ECG or +FH sudden death/cardiomyopathy
 - recurrent syncope is unresponsive to medical management
- Holter monitoring, Echo, exercise stress testing as determined by history, physical and ECG findings (all indicated with exercise-induced syncope)

Tilt Table Testing

- In 1996 American College of Cardiology published guidelines for tilt testing
- Indicated when cause of syncope is not clear
- Reserved for patients who have recurrent syncope or for high-risk patients after a single syncope event
- Adolescents with history consistent with neurocardiogenic syncope do not need tilt testing

Echocardiography

- What Is the Yield of Screening Echocardiography in Pediatric Syncope? *Pediatrics* 2000
- An abnormal history, physical exam or ECG allowed the identification of a cardiac cause of syncope with sensitivity of 96%
- In the absence of a h/o exercise-induced syncope, +FH or abnormal PE, the echo does not contribute to the evaluation of pediatric syncope
- Paucity of data regarding its value