Exercise Induced Collapse: Neurocardiogenic Syncope

Background
1. Vasovagal syncope - most common
2. Must rule out congenital heart defects

Pathophysiology
1. Mechanism - sudden reflex vasodilation/bradycardia
   o May be caused by:
     ▪ Stimulation of mechanoreceptors
     ▪ Elevated catecholamine
     ▪ Dehydration
   o Maximum exercise = high cardiac output
     ▪ Due to increased venous return and high heart rate
   o Sudden stop decreases venous return
   o Heart continues forceful contraction with low end diastolic volume
     ▪ Stimulate vasovagal reflex in medulla to increase vagal tone
     ▪ Increased vagal tone leads to bradycardia/vasodilation
       ▪ Hypotension/syncope
2. Other possible mechanisms
   o Bradycardia
     ▪ Increased parasympathetic output from vagus nerve
   o Vasodilation - due to:
     ▪ Post exercise withdrawal of alpha-agonist activity
     ▪ Increased Beta 2 agonist activity
   o Increased catecholamines/dehydration during prolonged exercise can stimulate ventricular contractions
     ▪ Catecholamine directly stimulates ventricles
     ▪ Dehydration increases heart rate/contractility

Diagnostics
1. Detailed history of syncopal event
   o Postural or exertional symptoms
   o Association with neurologic symptoms
   o History of cardiac disease
   o History of psychiatric illness
   o Family history of sudden death
2. Evaluate for structural heart disease
   o EKG
   o ECHO
     ▪ Pathological causes of syncope
     ▪ Positive findings may need:
       ▪ Exercise stress testing
       ▪ Cardiac catheterization
       ▪ Coronary angiography
       ▪ Myocardial biopsy
       ▪ Electrophysiology study
1. Ambulatory heart monitoring
   - Necessary if EKG/ECHO negative but continued syncopal episodes
     - Possible arrhythmia
   - Consider cardiology consult
   - Holter-24 hour monitoring
   - Event monitor-allows up to two week monitoring

3. Neurally mediated syncope
   - Carotid-sinus syncope
   - Psychiatric illnesses (panic, anxiety disorders, major depression)
   - Carotid sinus massage
     - Rubbing carotid sinus stimulates baroreceptors and glossopharyngeal nerve
     - Can cause decreased heart rate
     - May slow rapid heart rate
       - Atrial flutter or atrial tachycardia
     - Can provide diagnostic information
   - May be useful in older patients
     - Carotid-sinus syncope common in elderly
   - Avoid in patients with carotid bruit/known cerebrovascular disease
     - May dislodge thrombus/plaque
   - Diagnostic testing
     - Head up tilt table test
     - Increased sensitivity with provocation with isoproterenol or nitroglycerin

**Therapeutics**
1. Activity modification
2. Avoid sports that may cause syncopal episodes
3. Proper post exercise cool-down periods
4. Increase salt and water intake
5. Wear compression stockings
6. Reestablish cerebral perfusion
   - Place athlete in Trendelenburg position
   - Medications:
     - Beta-blockers, disopyramide, SSRIs, fludrocortisone, midodrine

**Prevention**
1. Proper hydration
2. Cool down periods
3. Avoid provocative environments
4. Be aware of warning signs
   - Lie down, cross legs, tighten leg muscles may help avoid fainting
5. Avoid alcohol
   - Causes vaso-arterial dilation
6. Reduce caffeine intake
7. Increase fluid intake
8. Medications:
   - B-blockers, disopyramide, SSRIs, fludrocortisone, midodrine
   - No protocol for athletes exist
   - Individual response vary

9. Pacemaker
   - Currently experimental use only for neurogenic syncope

References
3. Camargo, C, Simmons, FE. Anaphylaxis: Rapid recognition and treatment. UpToDate Online 16.3 Topic last update 10/10/08

Author: Tony Chang, MD, University of Nevada FPRP
Editor: Carol Scott, MD, University of Nevada Reno FPRP