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

- May be caused by:
 - Stimulation of mechanoreceptors
 - Elevated catecholamine
 - Dehydration
- Maximum exercise = high cardiac output
 - Due to increased venous return and high heart rate
- $\circ \quad \text{Sudden stop decreases venous return} \\$
- 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
 - Bradycardia
 - Increased parasympathetic output from vagus nerve
 - Vasodilation-due to:
 - Post exercise withdrawal of alpha-agonist activity
 - Increased Beta 2 agonist activity
 - 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

- Postural or exertional symptoms
- Association with neurologic symptoms
- History of cardiac disease
- History of psychiatric illness
- Family history of sudden death
- 2. Evaluate for structural heart disease
 - EKG
 - ECHO
 - Pathological causes of syncope
 - Positive findings may need:
 - Exercise stress testing
 - Cardiac catheterization
 - Coronary angiography
 - Myocardial biopsy
 - Electrophysiology study

- 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
 - o Causes vaso-arterial dilation
- 6. Reduce caffeine intake
- 7. Increase fluid intake

8. Medications:

- o B-blockers, disopyramide, SSRIs, fludrocortisone, midodrine
- No protocol for athletes exist
- Individual response vary

9. Pacemaker

• Currently experimental use only for neurogenic syncope

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