Postural (Orthostatic) Hypotension

By: Alex Rabinovich  November 19, 2003

Definition

• Drop in blood pressure when changing position from supine to standing
• Symptoms are due to cerebral hypoperfusion

Epidemiology

• Multiple studies have found up to 20% of people > 65 y.o. have PH
• Most people with PH are asymptomatic
• Many patients also have supine hypertension, and are treated with BP meds that can exacerbate PH

Physiology

• Standing up causes pooling of 500-1000 ml in the lower extremities and splanchnic circulation

Normal Response

• Rapid decrease in venous return to the heart
• Reduced ventricular filling (Stroke Volume) results in decreased Cardiac Output and MAP
  o  $CO = \text{HR} \times \text{Stroke Volume}$
  o  $MAP = CO \times \text{Total Peripheral Resistance}$
  o  $MAP = \text{Diastolic} + (\text{Systolic} – \text{Diastolic})/3$
• Autonomic reflex from the Baroreceptor Reflex causes increase sympathetic and decreased parasympathetic outflow from the nervous system
  o  Stretching the baroreceptors increases firing, causes inhibition of sympathetic drive from the medulla
  o  The opposite is true with hypotension (i.e. baroreceptors are NOT stretched) thus decrease firing
• Increased sympathetic drive causes increase total peripheral resistance, venous return and hence increases CO and MAP to compensate for volume shift
• This response causes a small fall in Systolic BP (5-10 mmHg) and small increase in HR (10-15 bpm)

Abnormal Response

1. Autonomic Dysfunction
   o  Autonomic reflex is impaired, and standing causes pooling without compensated vasoconstriction
   o  Autonomic Failure can be identified when there is no increase in HR when BP falls
2. Volume Depletion (or volume shift away from arterial circulation)
   o  Excessive pooling, or excessive loss of fluid from the arterial circulation, without compensation will cause significant decrease in CO, MAP and Cerebral Perfusion

Signs and Symptoms

• Blurry vision
• Lightheadedness
• Dizziness
• Diminished hearing
• Nausea
• Weakness
• Falls on standing
• Syncope

Diagnosis

1. Review medications
2. Review coexisting medical disorders
3. Determine the relationship of orthostatic hypotension to meals, exercise, straining or Valsalva maneuvers, and standing up after getting out of bed in the morning
4. Record supine and standing blood pressure and pulse after two-five minutes
5. Perform a complete neurologic examination (MME, CN, Motor, Reflex, Balance, Coordination)

PH is diagnosed when, within 2-5 minutes of standing, you get:
• A 20 mmHg fall in systolic pressure OR a 10 mmHg fall in diastolic pressure
• Clinical signs and symptoms of cerebral hypoperfusion can also be used for the diagnosis
## Differential Diagnosis

<table>
<thead>
<tr>
<th>Neurogenic</th>
<th>Non-Neurogenic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary automatic system failure</strong></td>
<td><strong>Cardiac pump failure</strong></td>
</tr>
<tr>
<td>o Multisystem atrophy (Shy-Drager syndrome)</td>
<td>o Myocardial infarction</td>
</tr>
<tr>
<td>o Pure autonomic failure (Parkinson's Disease)</td>
<td>o Myocarditis</td>
</tr>
<tr>
<td>o Subacute dysautonomia</td>
<td>o Constrictive pericarditis</td>
</tr>
<tr>
<td><strong>Secondary autonomic system failure</strong></td>
<td><strong>Constrictive pericarditis</strong></td>
</tr>
<tr>
<td>o Brain and brainstem</td>
<td>o Aortic stenosis</td>
</tr>
<tr>
<td>o Tumor</td>
<td>o Tachyarrhythmias</td>
</tr>
<tr>
<td>o Stroke</td>
<td>o Bradyarrhythmias</td>
</tr>
<tr>
<td>o Multiple sclerosis</td>
<td><strong>Reduced intravascular volume</strong></td>
</tr>
<tr>
<td>o Spinal cord</td>
<td>o Straining on heavy lifting, urination, defecation</td>
</tr>
<tr>
<td>o Transverse myelitis</td>
<td>o Dehydration</td>
</tr>
<tr>
<td>o Syringomyelia</td>
<td>o Hemorrhage</td>
</tr>
<tr>
<td>o Tumor</td>
<td>o Burns</td>
</tr>
<tr>
<td>o Tabes dorsalis</td>
<td>o Salt-losing nephropathy</td>
</tr>
<tr>
<td>o Peripheral nervous system</td>
<td>o Adrenal insufficiency</td>
</tr>
<tr>
<td>o Diabetes mellitus</td>
<td>o Diabetes insipidus</td>
</tr>
<tr>
<td>o Guillain-Barre syndrome</td>
<td><strong>Venous pooling</strong></td>
</tr>
<tr>
<td>o Alcoholic polyneuropathy</td>
<td>o Alcohol</td>
</tr>
<tr>
<td>o HIV</td>
<td>o Postprandial dilation of splanchnic vessel beds</td>
</tr>
<tr>
<td>o Amyloidosis</td>
<td>o Vigorous exercise</td>
</tr>
<tr>
<td>o Porphyria</td>
<td>o Heat: hot environment, hot showers/baths, fever</td>
</tr>
</tbody>
</table>

**NOTE:** Neurogenic causes of PH are NOT accompanied with HR increase. Because the autonomic reflex is compromised, therefore no response.

### Treatment

#### Non-Pharmacologic treatments
- Avoidance of prolonged standing
- Slow, careful changes in position, especially on arising in the morning
- Avoidance of alcohol
- Avoidance of hot environments and hot showers or baths
- Avoidance of rigorous exercise
- Sleeping with head-up tilt
- Scheduling of activities in the afternoon
- Increased salt and fluid intake
  - Volume loading
- Increase caffeine intake
  - Vasopressor effects
- Compressive stockings
  - Decrease pooling potential, increase venous return

#### Pharmacologic treatments
- Removal of medications that exacerbate hypotension
  - Antihypertensive (ACE-I, CCB)
  - Diuretics (Thiazides)
  - Vasodilators: Nitrates, Hydralazine
  - Alpha- and beta-blocking agents
  - CNS Depressants: Barbiturates, Opiates, Benzo’s
  - Tricyclic antidepressants
  - Phenothiazines
- Fludrocortisone (Florinef)
  - Enhances sensitivity of blood vessels to catecholamines.
  - Increases BP by increasing TPR
  - Mineralocorticoid affect, increases blood volume
  - Observe patients for pedal edema, CHF or worsening of supine hypertension. May require K+ supplements
- Sympathetic antagonists such as Midodrine, Phentylephrine, Methylphenidate, Dextroamphetamine Sulphate
  - Vasopressor effects (alpha-1 adrenoreceptor agonists)
  - Vasoconstriction
- Erythropoietin
**Differential Diagnosis of Orthostatic Hypotension**

**Autonomic dysfunction**
- Baroreceptor failure (common in the elderly)
- Bradbury-Egleston syndrome (pure autonomic failure)
- Shy-Drager syndrome (multiple system atrophy)
- Autonomic failure with Parkinson’s disease
- Riley-Day syndrome (familial dysautonomia)
- Dopamine-β-hydroxylase deficiency
- Acute pandysautonomia
- Diabetes mellitus
- Amyloidosis
- Porphyria
- Paraneoplastic autonomic neuropathy
- Acute inflammatory neuropathy
- Hereditary sensory

**Hypovolemia**
- Hemorrhage or plasma loss
- Overdiuresis
- Overdiatysis

**Endocrine disorders**
- Primary adrenal insufficiency
- Hyperaldosteronism
- Pheochromocytoma
- Renovascular hypertension

**Vasodilator excess**
- Mastocytosis (histamine, prostaglandin D2)
- Hyperbradycinism
- Carotid syndrome (bradyklin)
- Hypermagnesemia

**Paroxysmal autonomic syncope**
- Glossopharyngeal syncope
- Micturition syncope
- Carotid sinus syndrome
- Swallow syncope
- Cough syncope
- Bezold-Jarisch reflex activation

**Miscellaneous**
- Drugs and toxins
- Alcohol
- Stroke-Adams attacks
- Mitral valve prolapse syndrome
- Hypothesis, weakness, bed rest

**Vascular insufficiency**
- Varicose veins
- Absent venous valves
- Arteriovenous malformations

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**Figure 1.** Location and innervation of arterial baroreceptors.

**Figure 3.** Baroreceptor feedback loop depicting a sudden decrease in arterial pressure (e.g., hemorrhage).