Prevention and Treatment of Orthostatic Hypotension in the Orthopedic Patient Population

Rachel Swope, PharmD; Aimee Adams, PharmD

Orthostatic hypotension, also known as postural hypotension, occurs in some patients when they stand up or lay down. During episodes of orthostatic hypotension, patients often feel dizzy or lightheaded and can faint and fall. The rates of inpatient hospital falls after orthopedic surgeries is 2.5 falls per 1000 patient-days. Postoperative falls are a major source of morbidity for orthopedic patients, and thus an effort must be made to understand the pathophysiology, risk factors, and prevention techniques of orthostatic hypotension.

**DEFINITION, SYMPTOMS, ANDDIAGNOSIS**

Orthostatic hypotension is defined as a decrease of at least 20 mm Hg in systolic blood pressure or of 10 mm Hg in diastolic blood pressure occurring within 3 minutes of standing. Cerebral hypoperfusion leads to the symptoms that patients experience during an episode of orthostatic hypotension. Symptoms include lightheadedness, weakness, dizziness, difficulty thinking, headaches, feeling faint, and syncope. After the initial episode of cerebral hypoperfusion, a compensatory autonomic response happens. Symptoms from this response include nausea, chest pain, coldness of extremities, palpitations, and syncope.

Diagnosis of orthostatic hypotension can be made by measuring blood pressure while the patient is lying on a tilt table in the supine position at an angle of at least 60°. It can also be diagnosed by monitoring blood pressure in the sitting and standing positions. After 1 minute of standing, orthostatic hypotension will be detected in the majority of patients. Severe orthostatic hypotension can be defined as a further drop in blood pressure after 2 minutes of standing. Orthostatic hypotension developing after 3 minutes of standing usually represents a reflex vasovagal response or a mild and early form of adrenergic dysfunction. A patient’s heart rate should also be recorded in both the sitting and standing positions to help delineate cause.

**CAUSES OF ORTHOSTATIC HYPOTENSION**

Orthostatic hypotension can be caused by drugs or neurogenic or nonneurogenic factors. Common medications that can cause orthostatic hypotension are listed in Table 1. The most common medications are vasodilators, antihypertensive agents, diuretics, antidepressants, and opioids. In the immediate postoperative period, orthopedic patients will often be receiving 1 or more of these medications, putting them at risk for the development of orthostatic hypotension. Hundreds of medications have the potential to cause orthostatic hypotension.

Neurogenic causes of orthostatic hypotension relate to an abnormality in the reflex regulation of circulation by the sympathetic noradrenergic nervous system. Peripheral neuropathies caused by diabetes mellitus, autoimmune diseases, human immunodeficiency virus, antineoplastic agents, or vitamin B12 deficiencies are common causes...
of neurogenic orthostatic hypotension. Central lesions (eg, Parkinson’s disease or spinal cord injuries) can also lead to neurogenic orthostatic hypotension.37 When diagnosing orthostatic hypotension, neurogenic causes will not exhibit reflex tachycardia during the hypertensive phase. Thus, an impairment exists in the cardiovascular impulses to compensate for hypotension.

Nonneurogenic orthostatic hypotension may be caused by hypovolemia, cardiac impairment, or vasodilation.7 Table 2 provides details on nonneurogenic causes of orthostatic hypotension. During the diagnosis of orthostatic hypotension in nonneurogenic cases, a sudden increase is found in the heart rate to compensate for the sudden drop in blood pressure. Patients in the immediate postoperative period after an orthopedic procedure are at risk for orthostatic hypotension from all 3 causes, but specifically from medications and nonneurogenic factors.

### Risk Factors

Modifiable risk factors for orthostatic hypotension include medications, volume depletion, and immobility. All of these factors may play a role in the postoperative period. Orthostatic hypotension is more common in elderly patients who have experienced age-related physiologic cardiovascular changes and a blunting of the sympathetic nervous system, which puts them at an increased risk of orthostatic hypotension. Also, elderly patients are more likely to be taking high-risk medications and have comorbid conditions that further increase their risk.8 Weiss et al9 studied the prevalence of orthostatic hypotension in elderly patients in an acute geriatric ward. Orthostatic tests were obtained 3 times per day after meals for 489 patients (average age, 81.6 years). The study found that 67.9% patients had at least 1 episode of orthostatic hypotension throughout the day, and that diastolic orthostatic hypotension was more common than systolic.9

### Primary Prevention

Prevention of orthostatic hypotension involves treating its causes and avoiding risk factors. Postoperative orthopedic surgery patients are at a high risk for developing orthostatic hypotension due to medications, possible dehydration, and prolonged immobility. Primary prevention of orthostatic hypotension is key.

To prevent orthostatic hypotension, the modifiable risk factors must be considered. First, the practitioner must assess the patient’s current medication regimen and underlying condition should be discontinued or the underlying condition should be treated. Nonpharmacologic interventions should be considered after initial assessment of the medication regimen and disease states. These strategies can be used in any order and should be selected based on patient preference.

### Treatment

The goal of treating orthostatic hypotension is to increase the patient’s standing blood pressure without increasing the supine blood pressure. Achievement of this goal will allow the patient to stand longer, reduce orthostatic symptoms, and improve the patient’s ability to perform activities of daily living. The first step to treating orthostatic hypotension is to identify the underlying cause. If possible, the causative medication should be discontinued or the underlying condition should be treated. Nonpharmacologic interventions should be considered after initial assessment of the medication regimen and disease states. These strategies can be used in any order and should be selected based on patient preference.

### Table 1

<table>
<thead>
<tr>
<th>Medication Class</th>
<th>Medications</th>
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<tbody>
<tr>
<td>Alpha-1 antagonists</td>
<td>Terazosin, prazosin, doxazosin</td>
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<tr>
<td>Antihypertensives</td>
<td>Angiotensin-converting enzyme inhibitors, beta blockers, clonidine</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Hydrochlorothiazide, loop diuretics</td>
</tr>
<tr>
<td>Phosphodiesterase type 5 inhibitors</td>
<td>Sildenafil, vardenafil</td>
</tr>
<tr>
<td>Antidepressants</td>
<td>Tricyclic antidepressants, trazodone, monoamine oxidase inhibitors</td>
</tr>
<tr>
<td>Opioids</td>
<td>Morphine, oxycodone, tramadol</td>
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</table>

### Table 2

<table>
<thead>
<tr>
<th>Cause</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Hypovolemia</td>
<td>Dehydration, blood loss, adrenal insufficiency, diarrhea, or diabetes insipidus</td>
</tr>
<tr>
<td>Cardiac impairment</td>
<td>Bradycardia, aortic stenosis, tachyarrhythmia, myocardial infarction, or pericarditis</td>
</tr>
<tr>
<td>Vasodilation (venous pooling)</td>
<td>Prolonged recumbency or standing, fever, severe varicosities, or postprandial dilation of splanchnic vessels</td>
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</table>
One simple nonpharmacologic intervention for the treatment of orthostatic hypotension is to give a bolus (500 mL) of water orally or of normal saline intravenously if the patient is hospitalized. A fluid bolus will expand plasma volume, which will in turn increase blood pressure. This effect can last up to 2 hours; thus, it can improve symptoms and the patient’s ability to perform activities of daily living. Physical maneuvers to treat orthostatic hypotension are designed to increase venous return by reducing peripheral capacitance and increasing total peripheral resistance. Physical countermeasures should be considered at the first symptoms of orthostatic hypotension. Several maneuvers can be used, and they all involve contracting the muscles below the waist for approximately 30 seconds at a time. Maneuvers include leg elevation, slow marching in place, thigh muscle contraction, leg crossing and contraction, toe raising, and bending at the waist.

Pharmacologic methods to treat orthostatic hypotension should be the last line of treatment after all nonpharmacologic methods. The 2 main medications used for treatment of orthostatic hypotension are fludrocortisone and midodrine. Fludrocortisone, a synthetic mineralocorticoid, increases blood pressure by increasing alpha-receptor sensitivity within the vasculature and by expanding plasma volume. Indications for fludrocortisone include an inadequate response to increased salt intake and contraindication to a high-sodium diet. Fludrocortisone is typically the first agent used, but if patients are unable to tolerate it, then midodrine can be considered. Midodrine improves systolic blood pressure and orthostatic symptoms and increases standing and walking time by causing systemic vasoconstriction via its active metabolite (desglymidodrine), stimulating alpha-1 receptors. Together, midodrine and fludrocortisone have synergistic effects; thus, when used in combination, the doses of both agents can be lowered. However, both medications are complicated by the development of tachphyllaxis, but with a short medication holiday, efficacy can be regained.

Third-line agents that should only be used after fludrocortisone or midodrine have failed include pyridostigmine, nonsteroidal anti-inflammatory drugs, and caffeine. These agents may be used in combination or alone. Pyridostigmine is a cholinesterase inhibitor that improves orthostatic hypotension and total peripheral resistance by improving neurotransmission within the sympathetic baroreflex pathway. Table 3 provides more information on pharmacologic treatment options.

**SECONDARY PREVENTION**

Patient education is one of the most important factors in the control of orthostatic hypotension. Patients should be counseled about the symptoms of hypotension and the conditions that can increase the risk of an episode of orthostatic hypotension (eg, heat, exercise, hot bath, or heavy meal). Education should include information about rising slowly in stages from a supine to a standing position, especially in the morning, when orthostatic tolerance is at its lowest. Patients should also be counseled on countermeasures and other nonpharmacologic mechanisms to treat and prevent orthostatic hypotension.

One option for secondary prevention of orthostatic hypotension is abdominal compression with an elastic binder. This option is ideal for patients who experience adrenergic denervation of vascular beds, causing an increase in vascular capacitance and peripheral venous pooling. Compression of the capacitance beds improves orthostatic symptoms by increasing total peripheral resistance and reducing venous capacitance. In healthy adults, a shift of approximately 500 mL of blood to the lower extremities occurs on standing. Thus, in patients with increased vascular compliance, the shift in fluid will result in a state of hypovolemia, leading to orthostatic hypotension. Smit et al discovered that an elastic abdominal binder increased standing blood pressure.

### Table 3

<table>
<thead>
<tr>
<th>Medication</th>
<th>Class</th>
<th>Initial Dose</th>
<th>Max Dose</th>
<th>Dose Titration</th>
<th>Dose Adjustments</th>
<th>Adverse Effects</th>
<th>Use Caution or Avoid in the Following Populations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fludrocortisone</td>
<td>Mineralocorticoid</td>
<td>0.1 mg by mouth daily</td>
<td>1 mg/d</td>
<td>Increase 0.1 mg/d once per week</td>
<td>None</td>
<td>Undefined frequency: hypokalemia, supine hypertension, edema, adrenal suppression</td>
<td>Uncontrolled hypertension, congestive heart failure, electrolyte disturbances</td>
</tr>
<tr>
<td>Midodrine</td>
<td>Peripheral alpha-1 antagonist</td>
<td>2.5-10 mg by mouth 3 times per day</td>
<td>40 mg/d</td>
<td>Renal: start at 2.5 mg 3 times per day and titrate as tolerated HD: give dose after HD</td>
<td>&gt;10% frequency: supine hypertension, urinary retention, dysuria, pilomotor reactions, pruritus, paresthesia</td>
<td>Uncontrolled hypertension, urinary retention</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: HD, hemodialysis; Max, maximum.

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1. Smit et al.
2. Healio.com
3. Orthopedics
ure approximately 11.6 mm Hg when exerting 15 to 20 mm Hg. Binders should be placed prior to rising from a bed and should be tightened until they exert gentle pressure. Lower-extremity compression stockings may also be used to reduce symptoms of orthostatic hypotension by decreasing blood pooling in the legs. Abdominal compression is thought to be more effective than lower-extremity compression stockings because the venous capacitance of the calves and thighs is smaller than that of the splanchic mesenteric bed, which accounts for 20% to 30% of total blood volume. However, some patients benefit from lower-extremity compression stockings.

Patients should be counseled to sleep with their head at a 10° to 20° angle (ie, elevated 4 inches) to decrease nocturnal hypotension. This can be accomplished by placing blocks under the head of the bed. If patients remain tilted up, the vasomotor tone will increase, which will then gradually reduce orthostatic hypotension. Mild exercise programs may be useful in increasing orthostatic tolerance by increasing plasma volume and decreasing venous pooling. Training in the supine position via stationary bicycle or swimming is recommended over upright exercise due to the increased risk of orthostatic blood pressure drop with standing exercises.

Plasma volume should be maintained adequately to reduce the incidence of orthostatic hypotension. Patients should be advised to drink 5 to 8 glasses of water per day. Also, patients should be counseled about having an adequate sodium intake (10 to 20 grams of salt per day) in their diet to maintain oncotic pressure. However, if the patients have comorbid conditions requiring salt-restricted diets, this may trump the salt therapy for reduction of orthostatic hypotension.

REFERENCES