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Orthostatic Hypotension

Pathophysiology, Problems, and Prevention

Orthostatic hypotension (OH) has traditionally been associated with falls (R. H. Fortinsky et al., 2008). However, data has been inconsistent, and some believe that more research in the area is needed before a true link can be made (A. Gangavati et al., 2011). OH, a drop in systolic pressure of ≥ 20 points or in diastolic pressure of ≥ 10 points within 3 minutes of standing as compared to the reading while supine may be accompanied by tachycardia, syncope, dizziness, visual changes, and death. Pathophysiological causes of OH include the dependent pooling of blood in organs and the periphery, compromised venous return and stroke volume, and changes in cardiac and baroreceptor function. Hypertension, diet, mobility, and medications may also contribute to the disorder. The treatment goal is to educate the patient, eliminate physiological causes, omit contributing medications, and resort to pharmacologic means as needed (J. B. Lanier et al., 2011). Thorough knowledge about OH can assist home care nurses in assessing for and educating patients about the disorder. This manuscript will discuss the pathophysiology, contributing diagnoses and drug classes, signs and symptoms, treatment, and implications for home care and hospice nurses related to orthostatic hypotension.

Overview

According to the Centers for Disease Control and Prevention (CDC, 2011), one in three adults older than 65 years falls annually and related injuries range from minor to severe including fractures, head injuries, and death. Falls are the leading cause of injury-related deaths, nonfatal injuries, and hospital admissions for trauma among this age group (CDC, 2011; Fortinsky et al., 2008; Gangavati et al., 2011). Traditionally orthostatic hypotension (OH) has been associated with falls (Fortinksy et al.; Romero-Ortuno et al., 2011); however, data have been inconsistent and some believe that more research in the area is needed before a true link can be made (Gangavati et al.). Regardless, it is imperative for home care nurses to include fall risk assessment and management into home care visits (Fortinsky et al.). Thorough knowledge about OH can assist nurses in assessing for and educating patients about the disorder. This article will discuss the pathophysiology, contributing diagnoses and drug classes, signs and symptoms, treatment, and implications for home care and hospice nurses related to OH.

Pathophysiology

Normally when changing position from lying to standing, systolic blood pressure remains unchanged or drops slightly, while diastolic blood pressure rises slightly (Hogan-Quigley et al., 2012). However, sometimes abnormal drops in pressure occur with positional changes. OH, also called postural hypotension or orthostasis, is defined as a drop in systolic pressure of 20 points or more or in diastolic pressure of 10 points or more, within 3 minutes of standing as compared to the reading while supine (Hogan-Quigley et al.; Lanier et al., 2011). OH may also be accompanied by tachycardia (Hogan-Quigley et al.). Studies indicate that the prevalence of OH is between 5% and 30% for healthy community-dwelling older adults, increases with age (Romero-Ortuno et al., 2011), and may be present in up to 50% of long-term-care residents (Harvard Health Publications, 2011).

The pathophysiological cause for positional drops in blood pressure is related to the dependent pooling of blood in the veins of the liver, intestines, organs, legs, and feet. Venous return and stroke volume decrease as a result of the pooled blood, so cardiac output becomes compromised and arterial blood pressure drops (Hogan-Quigley et al., 2012; Mosnaim et al., 2010). Gravity also causes blood to rush downward from the torso to the feet upon standing, further decreasing venous return (Karch, 2006). An estimated 500-1,000 mL of blood pools in the lower extremities and splanchnic circulation upon standing (Lanier et al., 2011). Finally, hypertensive patients may additionally experience transient cerebral ischemia from decreased cerebral perfusion during acute orthostatic drops in pressure (Gangavati et al., 2011), resulting in the neurologic symptoms of OH (Lanier et al.).

Once venous return and stroke volume are compromised during orthostasis, the body attempts to compensate by activating baroreceptors located in the aorta and carotid arteries (Karch, 2006). Baroreceptors are stimulated by pressure exerted upon them by blood in the vessels. When blood pools in lower extremities, upper body vessel pressure falls, baroreceptor stimulation stops, and this information is transmitted to the brain. The medulla then reacts by initiating vasoconstriction, increasing heart rate and cardiac output to return pressure to vessel walls and raise blood pressure. Baroreceptors work constantly to maintain a normal range of blood pressure (Karch). With inadequate response by this autonomic system, OH becomes an issue (Mosnaim et al., 2010).

The compensatory work of the baroreceptors is not immediate so it may take several minutes to restore normal blood pressure after a drop. For this reason many resources define OH as a drop in pressure within 3 minutes of standing (Hogan-Quigley et al., 2012; Lanier et al., 2011; Mosnaim et al., 2010). However, a recent study suggests that orthostatic drops in blood pressure may occur much earlier than 3 minutes, and that earlier blood pressure measurement (such as after 1 minute) may be more accurate in detecting OH and in linking OH to falls (Gangavati et al., 2011).

Many factors can affect blood pressure in patients with OH. As people age, the heart's pumping action loses efficiency and blood vessels stiffen and weaken, causing decreased cardiac output, which contributes to decreased compensation after orthostasis. In addition, baroreceptors become less sensitive to pressure changes and the signals they send to the medulla are not as effective with age. As a result, blood pressure may drop significantly without immediate compensation (Mosnaim et al., 2010). Finally, some patients experience the phenomenon of postprandial

hypotension, whereby systolic blood pressure may drop by 20 points within 75 minutes of a large meal, especially if the meal includes high levels of carbohydrates (Lanier et al., 2011).

There is also evidence to support a connection between hypertension (HTN) and OH (Gangavati et al., 2011; Harvard Health Publications, 2011). Advanced age and HTN both decrease baroreceptor compensatory mechanisms, which then increase the risk of OH. In both hyper- and hypotension blood vessels may be stiffer than normal rendering baroreceptors less responsive to changes (Harvard Health Publications). According to the literature, people with well-controlled HTN tend to have less occurrences of OH than people with uncontrolled HTN (Gangavati et al.). See Tables 1 and 2, respectively, for factors/diagnoses and medication classes that contribute to OH.

Signs and Symptoms

OH can be symptomatic or asymptomatic, mild or severe, and acute or chronic (Poon & Braun, 2005). Symptoms can vary greatly from diminished cognition and disturbed emotional status (Mosnaim et al., 2010), to lightheadedness, dizziness and changes in vision (Harvard Health Publications, 2011), to syncope/fainting (Texas Arrhythmia Institute, 2010), and death (Romero-Ortuno et al., 2011). Other common symptoms of OH include weakness, fatigue, nausea, palpitations, and headache. Syncope, dyspnea, chest pain, and neck and shoulder pain are also possible but occur less frequently (Lanier et al., 2011).

Controversy exists over whether or not OH can be linked to falls. Healthcare professionals tend to automatically associate the two; however, Gangavati and colleagues (2011) state that little data exist linking falls with OH based on the current definition of OH. The definition describes a drop in pressure occurring after standing for 3 minutes. By 3 minutes baroreceptors may have begun the compensatory mechanisms; thus, a normal blood pressure may be recorded despite a possible significant drop in pressure earlier than 3 minutes. Findings from Gangavati and associate's study suggests that if pressure is taken at 1 minute after standing, symptoms may occur, blood pressure may drop, and falls may be the result, thereby possibly linking OH with falls in older adults.

Table 1. Diagnoses Contributing to Orthostatic Hypotension

Diagnosis	Contributing Factors
Diabetes mellitus	Autonomic neuropathy can affect compensatory mechanisms (Romero-Ortuno et al., 2011)
Parkinson's disease	Autonomic failure with involvement of the peripheral autonomic system; dopaminergic drugs for treatment may also contribute (Mosnaim et al., 2010)
Medications	Side effects of many medication classes can cause OH (see Table 2) (Harvard Health Publications, 2011)
Dehydration	Loss of fluid volume contributes to lower blood pressure (TXAI, 2010)
Blood loss	Loss of blood volume contributes to lower blood pressure (Harvard Health Publications)
Varicose veins in lower extremities	Retention of blood in periphery reduces circulating blood volume to the heart; also limits ability to venoconstrict (TXAI)
Bed rest	Decreases cardiac fitness and response of blood vessels (Harvard Health Publications)
Atherosclerosis and vascular stiffness	Decreases vascular response and blood flow to the brain (Gangavati et al; TXAI)
Congestive heart failure	Failure of heart to pump blood (TXAI)
Kidney function alteration	May contribute to dehydration and fluid volume deficit (Harvard Health Publications)

Note: OH = orthostatic hypotension.

Treatment of OH

The goal in treating OH is to improve symptoms without causing supine HTN (Lanier et al., 2011). This includes treating physiological causes, omitting or lowering contributing medications when possible, and using pharmacologic means as a last resort when necessary.

Treatment for acute or chronic OH begins with educating patients about the diagnosis. Following education, nonpharmacologic treatment is the first line of defense. Such interventions include avoiding carbohydrate rich meals, adding sodium-rich foods or sodium tablets to the diet, limiting alcohol intake, and ensuring adequate hydration (Mosnaim et al., 2010). It has been suggested that elevating the bed 10°-20° may decrease the occurrence of OH by slightly decreasing kidney perfusion at night, thus decreasing urine output, and fluid loss. It is recommended that certain medications (especially antihypertensive prescriptions) be taken at bedtime to relieve OH symptoms (Mosnaim et al.; Lanier et al., 2011).

When pharmacologic means are necessary to treat OH, the first line of medications ordered is mineralocorticoids, specifically Fludrocortisone. It works by increasing the absorption of sodium and water and thus raising blood pressure. This drug has several potential adverse effects including headache, supine HTN, hypokalemia, and congestive heart failure. Another drug used for OH is Midodrine, an a-1-adrenergic agonist. Several potential adverse effects exist for this drug as well. Finally, Pyridostigmine, a cholinesterase inhibitor may be used, but also may have adverse effects including loose stool, diaphoresis, and hypersalivation. Given the potential dangers of the medications, nonpharmacologic treatment of OH is preferred whenever possible (Lanier et al., 2011).

Implications for Home Care and Hospice Nurses: Assessment and Patient Education

Care of the client with OH includes assessing blood pressure while lying, sitting, and standing, and evaluating for orthostatic changes along with symptoms. Careful fall risk assessments, review of high-risk medications, and awareness of contributing diagnoses can alert the nurse to potential risks for OH. Uncontrolled HTN may also be a factor to assess for when considering patients at risk.

Table 2. Medication Classes Contributing to Orthostatic Hypotension (OH)

Medication Class	Mechanism of Action Contributing to OH
Diuretics	Increase urine output, therefore decrease fluid and blood volume (Poon & Braun, 2005). Decrease peripheral resistance via vasodilation (Mosnaim et al., 2010). Decrease in sodium reabsorption can result in hyponatremia (Mosnaim et al.).
Alpha blockers	Decrease in sympathetic outflow from central nervous system, especially with first dose (Mosnaim et al.).
Beta blockers	Decrease myocardial contractility, cardiac output, and renin secretion; depress sympathetic nervous system and change baroreceptor sensitivity (Mosnaim et al.).
Calcium channel blockers	Decrease total peripheral resistance resulting in vasodilation (Mosnaim et al.).
Vasodilators	Decrease peripheral resistance (Mosnaim et al.).
Antihistamines	Cause vasodilation (Harvard Health Publications, 2011).
Tricyclic antidepressants	Inhibit sodium, calcium and potassium channels resulting in arrhythmias Cause vasodilation and reduce pressure (Mosnaim et al.).
Nitrates	Result in transient episodes of dizziness associated with OH; exacerbated by alcohol (Mosnaim et al.).
Acetyl cholinesterase inhibitors (Donepezil)	Inhibits sympathetic nervous system (Mosnaim et al.).
Dopaminergics	Cause vasodilation (Mosnaim et al.).

Box 1. Case Study

Mrs. B, an 83-year-old White female, was referred to home care services by her physician after her husband called 911 following a collapse in her yard. Mrs. B had been working outside on a hot day and was sitting on her front steps slightly short of breath (SOB). On standing to enter the house, she became extremely dizzy and diaphoretic resulting in a fall and brief loss of consciousness. She began to vomit and was incoherent, causing Mr. B to call 911.

When emergency medical technicians arrived, vital signs were: blood pressure. 80/palpable; pulse, 112, thready; respirations, 22; temp, afebrile. She was oriented to person/place, but disoriented to time and event. She was extremely diaphoretic, complaining of nausea and dizziness, and unable to lift her head or sit up. She was transported to the emergency department and monitored for several hours while intravenous fluids were administered. Mrs. B recalled feeling SOB at home and taking a sublingual nitroglycerin (NTG) tablet to relieve the SOB. With no relief after 15 minutes, she took a second NTG tablet and sat down on her steps to rest. She had no recollection of what happened upon standing. The patient denied chest pain/pressure, and cardiac enzymes and electrocardiograms were normal. With no other injuries, Mrs. B was discharged to home after fluid replacement.

Upon admission to home care services the next day, the patient reported a past medical history of a Coronary Artery Bypass Graft 15 years earlier, with occasional shortness of breath currently, and no other significant history. She denied having angina, and was adamant that her NTG was prescribed solely for SOB, thus contact was made with the provider to clarify indication for usage of the NTG. She had no respiratory distress upon examination. The patient was assessed for dehydration related to the hot weather, outdoor work, and insufficient fluid intake. Blood pressure was assessed lying, sitting, and standing. Medication teaching regarding the proper use and side effects of NTG for chest pain, (and not SOB) was necessary along with education about orthostatic hypotension to avoid future preventable adverse events.

Patient education focused on ways to decrease OH is a valuable tool and could potentially save lives. Simple instructions include teaching patients to rise slowly from lying to standing and allowing feet to dangle first while wiggling/flexing toes and feet, which allows blood time to push upward from the periphery (Harvard Health Publications, 2011). Encouraging an exercise program may improve cardiac health, thus helping to avoid OH. Regimens may also include isometric exercises of the extremities, toe raises, and thigh contractions (Lanier et al., 2011). Such exercise routines may require the input of a physical therapist. Educating patients about dehydration and ways to avoid it is critical. Encouraging small frequent meals to avoid postprandial OH related to large carbohydrate-rich meals may also be helpful (Mosnaim et al., 2010). Some resources suggest the use of compression stockings to avoid peripheral blood pooling (Mosnaim et al.); however, they may only be helpful if waist high (Harvard Health Publications, 2011), making them somewhat uncomfortable and difficult to apply. Teaching patients to keep a diary of symptoms so healthcare professionals can track patterns may also be helpful (Lanier et al.). Reviewing medications with patients and teaching them about potential side effects and how to prevent them may be critical in decreasing OH and falls. Finally, patients who have access to telemonitoring systems from their home care agencies can benefit from education focused on the need to check blood pressure sitting, and then 1 and 3 minutes after standing. Information gathered in this way may be useful for titrating medications during physician visits (See Box 1.)

Conclusions

Many factors contribute to OH in older adults. Symptoms can range from discomfort to injury and death, while falls may also be associated with OH. Changes in the autonomic nervous system, the heart and vessels, comorbidities, and the use of certain medications may all contribute to OH as people age. Nonpharmacologic methods for

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treating OH are preferred, and nursing interventions can best focus on patient education. Home care nurses can easily assess for orthostatic changes and can intervene by educating the patients, and alerting the provider about reported and/or observed symptoms. ⌂

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