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HYPOTENSION: A REVIEW

B. Premkumar*, V. Mugilan, P. Venkatesan, K. Vinothkumar, P. Nandhini and A. Anas

Department of Pharmaceutics & Biotechnology, Sree Abirami College of Pharmacy, Eachanari, Coimbatore - 641 021, Tamil Nadu, India. [Affiliated to The Tamil Nadu Dr. M. G. R. Medical University, Chennai]

***Corresponding Author: B. Premkumar**
Department of Pharmaceutics & Biotechnology, Sree Abirami College of Pharmacy, Eachanari, Coimbatore - 641 021, Tamil Nadu, India.

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ABSTRACT
A drop in systemic blood pressure below recognized low levels is referred to as hypotension. Although a standard hypotensive number is not established, pressures below 90/60 are considered hypotensive. Because it is usually asymptomatic, hypotension is an underdiagnosed, generally benign disorder. It only becomes an issue when there is insufficient pumping pressure to supply oxygen-rich blood to vital organs. In addition to reviewing the causes of hypotension, this activity emphasizes the interprofessional team's involvement in managing the condition. In patients with spontaneous intracranial hypotension (SIH), postural headache and low cerebrospinal fluid (CSF) are common symptoms. Typically, a CSF leak is the root cause. Diffuse meningeal enhancement will be seen on brain magnetic resonance imaging (MRI) in most situations. The assessment and treatment of spontaneous intracranial hypotension are described in this exercise, which also emphasizes the importance of the interprofessional team in enhancing patient care.

KEYWORDS: Hypotension, Etiology, Pathophysiology, Symptoms and Treatments.

INTRODUCTION

Reduced systemic blood pressure below established low levels is known as hypotension. Less than 90/60 is considered hypotensive, even if there isn't a universally accepted standard hypotensive number. Because hypotension usually has no symptoms, it is an underdiagnosed generally benign ailment. Only when pumping pressure is insufficient to supply oxygen-rich blood to vital organs does it become an issue. As a result, the patient experiences symptoms that lower their quality of life. The biometric aspects of the blood pressure measurement are used to classify hypotension. It could be absolute if the mean arterial pressure drops to less than 65 mm Hg or the systolic blood pressure drops to less than 90 mm Hg. Hypotensive shock is a potentially fatal condition in acute situations. The definition of blood pressure: Cardiac output times total peripheral vascular resistance equals blood pressure. The average blood pressure during one cardiac cycle is known as the mean arterial pressure. It is computed as follows: 2/3 diastolic pressure plus 1/3 systolic pressure equals the mean arterial pressure. The creation, movement, and absorption of cerebrospinal fluid regulate intracranial pressure. Neurologic symptoms, most often headaches, occur if there is a change in these processes that results in a change in the CSF pressure. In patients with spontaneous intracranial hypotension (SIH), postural headache and low cerebrospinal fluid (CSF) are common symptoms. Typically, a CSF leak is the root cause.[1] Diffuse meningeal enhancement will be seen on brain magnetic resonance imaging (MRI) in most situations. There have been a few documented instances, though, where the patient had normal CSF pressure, no meningeal involvement on MRI, and a non-orthostatic headache. This process has historically been referred to by a variety of names, including liquorrhea, spontaneous or idiopathic low CSF pressure headache.[2]

Figure 1: Blood pressure ranges.

History and Physical

Orthostatic hypotension is diagnosed solely by a thorough history and physical examination. Orthostatic vital signs must be part of the physical examination. Orthostatic vitals screening should be performed on all patients, especially the elderly. It is common for this straightforward diagnostic to be overlooked in a clinical environment.

Leg buckling, headaches, and chest pain are less common presentations for patients than generalized symptoms such light-headedness, dizziness, or syncope. Examining the prescription list and determining any prior incidents or triggering factors is crucial. To rule out a cardiogenic origin of the symptoms, a thorough examination of the heart and nervous system must be conducted. It's crucial to rule out hypovolemia brought on by diuretics, neurogenic reasons, blood loss,

vomiting, and polypharmacy in the elderly. Excluding frequent causes like seizures and neurocardiogenic syncope (vasovagal syncope) is crucial, especially when it comes to syncope.

Orthostatic hypotension is inadvertently discovered during a clinical examination in certain patients who do not exhibit any symptoms. About one-third of patients with orthostatic hypotension in an observational study were found to be asymptomatic.[3][4] It is unknown what the clinical relevance of asymptomatic orthostatic hypotension.

Changing one's position usually causes the symptoms to go away, and lying down or sitting does just that. Patients could experience a brief loss of consciousness or perhaps fall. whether they get out of bed in the morning or throughout the day, whether they shift from sitting to standing, lying to sitting, or even from lying to sitting, they may experience symptoms. It is challenging to determine the diagnostic value of these symptoms, but if they coincide with orthostatic alterations, orthostatic hypotension testing ought to be prompted.

Etiology

Dural abnormalities can cause a congenital or traumatic CSF leak. CSF leaks are typically the result of fistulas or tears, which are the most common form of these problems. Despite the widespread belief that spontaneous intracranial hypotension (SIH) is caused by a barometric reduction in CSF pressure, the true cause of SIH is actually a fall in CSF volume.[5] A multitude of reasons, including the absence of dura surrounding the nerve root sheaths, congenital connective tissue disorders resulting in anatomical abnormalities, osteophyte protrusions, and spinal disc herniation, can cause CSF leaking. The lumbar and thoracic spine regions are the sites of focal weakening in the dura. CSF leaks occur particularly often from these arachnoid diverticula.[6 - 8] Spontaneous intracranial hypotension can also be brought on by trauma, surgery, and excessive CSF shunt drainage.[9] Total peripheral vascular resistance and cardiac output are the two main mechanisms that control blood pressure. Hypotension will thus result from any pathological condition that affects one or more of these factors.[10-14] The heart is a pump system that creates a pressure gradient so that blood can be distributed throughout the body. The cardiac output is the name given to this pumping potential. An equation is used to calculate cardiac output mathematically, various medication types that inhibit calcium channels. Another potential factor that lowers cardiac stroke volume is diuretic medicine. Arrhythmias, valve regurgitation, valvular stenosis, diastolic or systolic heart failure, significant blood volume losses, and cardiac tamponade are examples of disease states. The body's resistance to blood flow through the terminal arterioles of its many organ sites is known as total peripheral vascular resistance. This adds up to: 80 x (Mean Arterial Pressure - Mean Venous Pressure) / Cardiac Output is the systemic vascular resistance (or) Systemic Vascular Resistance can be calculated as (8 X L x n) / (3.14 x vessel radius^4), where n is the blood's viscosity and L is the vessel's length. Functionally, the length of the vessel is unaffected by changes in the body, and viscosity does not change quickly and is typically accepted as the standard value. Thus, the vessel's radius is the only physiological value that may be changed. Blood pressure rises as a result of increased blood flow resistance brought on by a decrease in arteriolar caliber. On the other hand, if terminal arterioles are larger in diameter, blood flow resistance will be reduced, which will lower blood pressure. In order to regulate variations in blood pressure, autonomic neural responses are the main mechanism controlling total peripheral vascular resistance. Dilated arterioles and relaxed smooth muscle tone are the normal states of arteriolar smooth muscle tone. Thus, the lack of or reduction in autonomic distributive shock happens when the heart's attempt to compensate for the loss of complete peripheral resistance fails. Warm skin and extremities, edema, increased mucous secretions, and tachycardia are the typical presentation of this. This is typically connected to septic shock and anaphylactic allergy responses. Failure to reach enough cardiac output while maintaining complete peripheral resistance is known as cardiogenic shock. Typically, these individuals have bradycardia, chilly, dry extremities, and skin. A reduction of total blood volume that prevents blood pressure from being maintained is known as hypovolemic shock. There is no change in total peripheral vascular resistance or cardiac output. This might happen as a result of severe bleeding during trauma or excessive diuretic prescription use that causes urine to lose fluid content. Cortisol deficiency as seen in Addison disease leads to a loss of fluid via urine and a relative cortisol deficiency. Sheehan syndrome is postpartum pituitary necrosis leading to a loss of many pituitary hormones as a result of postpartum shock or hemorrhage.

When the circulatory system is obstructed, constricted, or compressed, blood flow is inefficient or the heart's stroke volume is reduced, leading to obstructive shock. Systemically, this results in a relative decrease in blood pressure. Secondary obstruction can result from constrictive pericarditis, cardiac tamponade, tension pneumothorax, pulmonary embolism, or other restrictive cardiomyopathies. Traditionally, these will show up as peripheral edema, silent heart sounds, pulsus paradoxus, pulmonary crackles, or enlarged jugular veins as symptoms of congestive failure. Conceivable for any combination of the aforementioned diseases to develop it is also on currently and cause hypotensive shock. One instance is Waterhouse-Friderichsen syndrome, which is the inability of the adrenal glands to produce glucocorticoids, sex hormones, and mineralocorticoids as a result of open bleeding into the adrenal glands brought on by a Neisseria bacterium infection. Numerous hypovolemic and distributive shock symptoms follow from this. Heart rate times stroke volume equals cardiac

output. Disease conditions that lower heart rate or stroke volume will also lower the heart's overall cardiac output, which will functionally lower the heart's capacity to produce blood pressure. A number of drugs can also cause hypotension by increasing these biological markers. The drugs known to lower heart rate the most are beta-blockers.

Epidemiology

Orthostatic hypotension is more common as people age. The literature indicates that people 65 years of age or older are most likely to experience orthostatic hypotension, which may be partially explained by decreased baroreceptor sensitivity. In that age group, the prevalence can reach as high as 18.2%. Orthostatic hypotension affects one in five adults over 60 who live in the community and four people in long-term residential homes, according to a systematic review and meta-analysis of 26 research including over 25,000 participants.[15] According to two sizable population-based studies conducted in the United States, orthostatic hypotension affects fewer than 5% of individuals in the 45–49 age range, 15% of those in the 65–69 age range, and over 25% of those in the 85+ age range.[16] Elderly inpatients are more likely to experience orthostatic hypotension.[17][18] Elderly inpatients are more likely to experience orthostatic hypotn. Hospital prevalence may be impacted by clinical environments, testing frequency, and mobilization encouragement.[19][20] Orthostatic hypotension is also more common in patients with cardiovascular conditions such aortic stenosis, pericarditis/myocarditis, or arrhythmias. Younger and middle-aged people may also have it; these patients typically have chronic autonomic dysfunction without volume depletion. The actual cause determines the highly varied epidemiology of hypotension. Elderly patients are generally more likely to experience symptomatic, non-traumatic hypotensive episodes. Furthermore, individuals in better health and greater physical activity will have lower resting asymptomatic blood pressure.

Pathophysiology

Upon rising from a supine position, there is an instantaneous accumulation of blood in the lower extremities, ranging from 300 to 800 millilitres, due to gravity. The Frank-Starling Curve indicates that a decrease in cardiac output follows from a decrease in venous return to the heart. Known as the baroreceptor reflex, the human body typically adjusts by increasing sympathetic tone and decreasing vagal tone. The rise in peripheral vascular resistance caused by this increase in sympathetic outflow limits the drop in blood pressure by increasing cardiac output and venous return. Patients who are devoid of this compensatory mechanism have orthostatic hypotension symptoms. As a result of the equilibrium between the sympathetic and parasympathetic nervous systems, blood pressure is constantly controlled by the autonomic nervous system. By speeding up heartbeat and narrowing blood vessels, the sympathetic nervous system elevates blood pressure. Through the relaxation of arterioles to improve artery diameter and the reduction of heart rate, the parasympathetic nervous system reduces blood pressure.

Symptoms

Condition in which the blood vessels have persistently raised pressure, there are two types of symptoms.

- Common symptoms
- Non-specific symptoms

Common symptoms

- Light-headedness
- Dizziness
- Transient loss of consciousness
- Falls

Non-specific symptoms

- Blurry vision
- Visual field deficits
- Difficulty concentrating
- Cognitive slowing
- Weakness
- Fatigue
- Shortness of breath
- Chest pain
- Backache
- Lower extremity pain
- "Coathanger" headache

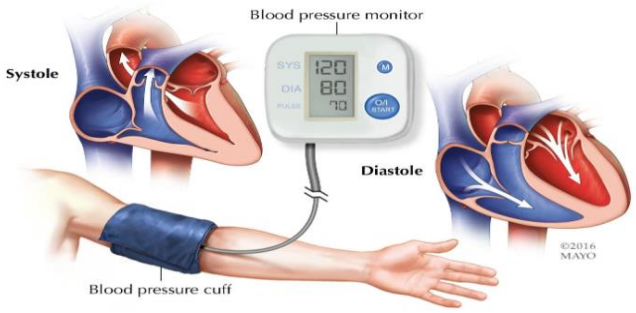

Figure 2: Test for hypotension.

The transportation industry is the primary application for low pressure tests, which are essential for guaranteeing the integrity and safety of packing systems during air transportation. These tests allow the simulation of real-life circumstances that items may encounter while shipping, particularly in the logistics industry. Testing guarantees that products in diverse industries meet standards and requirements and are of high quality and performance.[21]

Evalution

Assessment is based on the possible reason. Basic laboratory tests include ordering cortisol levels, thyroid-stimulating hormone (TSH), free t4, and complete blood count (CBC) with differential. In addition to stabilizing interventions, a STAT echocardiography with inferior vena cava (IVC) variability can be performed on a patient who is in shock. The results of an echocardiography will reveal the presence or absence of pericardial effusion, right ventricular pressures, and left ventricle ejection fraction. The inferior vena cava (IVC) variability test will assist in managing fluid resuscitation if the patient is in distributive shock and the left-ventricular ejection fraction (LVEF) and right-ventricular function are satisfactory. The optimal fluid resuscitation strategy is identified based on variations in pulse pressure. Additionally, pulmonary emboli caused by saddle emboli can also cause frank hypotension.

A comprehensive medication reconciliation is necessary for the initial assessment of patients with orthostatic hypotension. Orthostatic hypotension is frequently brought on by medications, such as diuretics, vasodilators, antidepressants, antipsychotics, and dopaminergic medications. An extensive cardiac evaluation, starting with an ECG and a laboratory assessment to check for evidence of anaemia, dehydration, diabetes, alcohol use disorder, or heart failure, should be performed on patients for whom there is no apparent reason. Not to be overlooked are neurodegenerative illnesses, which are mostly identified by a physical examination and history.

To identify orthostatic hypotension, orthostatic vital signs must be obtained. As you take the patient's pulse and blood pressure, let them lie down. The patient should be instructed to lie supine for five minutes, after which they should stand calmly for two to five minutes while their vital signs are again recorded. If the diastolic blood pressure drops by 10 mmHg or the systolic blood pressure drops by 20 mmHg, orthostatic hypotension might be diagnosed.[22 - 25] It is unknown what the best thresholds are based on these data to diagnose orthostatic hypotension.[26][27] The detection rates are increased when measurements are repeated later.[28] A customized clinical examination should be performed to find characteristics related to the symptoms and likely cause. For example, a patient who experiences palpitations may have a murmur, which could be an indication of an anatomical heart defect. A patient exhibiting rigidity and hypomimia, indicative of Parkinson disease, may also be experiencing tremors and slowness of movement.[29] When collecting readings of blood pressure when laying down or standing, it is crucial to take note of heart rate variations. A spike in heart rate of less than 15 beats per minute may suggest a neurogenic cause for orthostatic hypotension, while a rise in heart rate of more than 15 beats per minute may suggest a non-neurogenic explanation.[30] Heart rate, however, is not a precise predictor of the underlying reason and may be unreliable if the patient is using a beta-blocker, for instance.

Treatment

For low blood pressure, you might not require treatment. Treatment options may include increasing your fluid intake to avoid dehydration, taking blood pressure-raising medications, or modifying medications that lower blood pressure. Your doctor may discuss lifestyle modifications with you, such as altering your diet, eating habits, and sitting and standing posture. If you stand for extended periods of time, your doctor could also advise compression stockings.[31]

Methods of treatment

Non-pharmacological treatments for postural hypotension

- Change position gradually and in phases (from lying to sitting to standing) instead of swiftly.
- Maintain adequate hydration
- Avoid alcohol, warm environments, large meals, and hot showers or baths.
- Sleep with the head of the bed elevated
- Exercise programs
- Cross the legs while standing
- Tense the muscles in the legs and hips after standing
- Lower limb compression
- Abdominal binders

Pharmacological treatment

When nonpharmacologic interventions have failed to alleviate symptoms, pharmacologic treatment ought to be instituted. The research indicates that fludrocortisone and midodrine are still first-line treatments, while pyridostigmine is just one of numerous pharmacologic options available. Midodrine is an alpha-1 agonist, pyridostigmine is an acetylcholinesterase inhibitor, and fludrocortisone is an aldosterone analog. The methods by which each of these drugs increases vascular tone vary. In terms of symptom relief, a recent study found that midodrine was more effective than pyridostigmine.[32 - 34] Only those with autonomic dysfunction-related orthostatic hypotension should take midodrine; otherwise, it is considered off-label for treating other forms of orthostatic hypotension.[35] Another pharmaceutical option for treating orthostatic hypotension is droxidopa, however further research is required to support this medication.

Diagnosis

A cuff is used to take your blood pressure; it is often wrapped around your arm. The fit of the cuff is crucial. Its size might affect blood pressure measurements, making it too big or too small. A machine or a small hand pump is used to inflate the cuff.

Prognosis

Orthostatic hypotension raises mortality and the risk of myocardial infarction, heart failure, stroke, and atrial fibrillation even though it is frequently asymptomatic or has very few symptoms. Because orthostatic hypotension is linked to myocardial infarctions, transient ischemic episodes, electrocardiographic abnormalities, and carotid stenosis, cardiac and cerebrovascular disorders may account for the elevated vascular mortality seen.[36] During diastole, blood passes through the left coronary artery.[37] Accordingly, there may be a decrease in coronary blood flow in individuals with diastolic orthostatic hypotension, which would be detrimental to their chances of survival. Benign hypotension has a very good prognosis. Depending on the cause and severity, the prognosis for symptomatic hypotension varies.

Complication

Orthostatic hypotension results in deteriorating physical function, compromised balance, and decreased capacity for independent daily living tasks.[38] Orthostatic hypotension has been linked to an increased risk of the following, according to large meta-analyses

Untreated hypotension can have serious side effects, including low cardiac output and even death. Untreated hypotension in fulminant or approaching shock might result in multiple organ failure. In order to prevent these consequences, current guidelines for treating patients in shock or on the verge of sepsis emphasize aggressive and sufficient fluid resuscitation.

- Falls
- Heart failure
- Coronary heart disease
- Stroke
- Atrial fibrillation
- All-cause mortality

Enhancing healthcare team outcomes

An interprofessional team comprising an internist, intensivist, endocrinologist, emergency department physician, and nurse practitioner is the most effective in diagnosing and treating hypotension. Treatment is not necessary for outpatients who have asymptomatic hypotension. If symptoms are evident, nevertheless, treating hypotension should concentrate on treating the underlying cause. In order to correct the hypotension, some patients might also require vasopressor assistance in addition to intravenous fluids. It can be necessary to receive blood transfusions if bleeding is the cause. If the mean arterial pressure is less than 65 mm Hg, vasopressors might be recommended. Early medicines and periodic blood cultures are crucial if sepsis is suspected. Intramuscular epinephrine is crucial if anaphylaxis is suspected. Increasing a patient's dose of steroids to treat distributive shock when their constant need for vasopressors.[39 - 41]

Patients counselling

Patients should be informed by their providers about orthostatic hypotension, including its causes and aggravating factors. Prolonged standing, heavy meals, dehydration, alcohol consumption, hot environments, straining, and hot baths or showers might exacerbate symptoms.[42] There isn't much data to justify these policies, according to recent comprehensive reports.[43][44] There is little evidence to support the potential benefits of compression on the lower limbs and abdomen.

CONCLUSION

For patients who had a mean arterial pressure of less than 60 mmHg, a systolic blood pressure of less than 90 mmHg for death, and a mean arterial pressure of less than 55 mmHg for acute renal injury, the relationships between hypotension and patient outcomes were especially consistent in their direction. To completely understand the causal relationship between hypotension during an ICU stay and patient outcomes, interventional studies focusing on certain patient demographics and outcomes are necessary.

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