Special Issue: Vascular Disease Risk Factors

Hypertension management in patients with vascular disease: An update

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Hypertension (HTN) is a major risk factor for cardiovascular disease. About 80 million U.S. adults (33%) have HTN. Of these individuals, approximately 77% use antihypertensive medication, however, only 54% have controlled HTN. Studies have demonstrated that patients whose blood pressures are controlled achieve a minimum of 50% reduction in cardiovascular events compared to similar patients with poorly controlled blood pressure. This article will define HTN and its consequences. Diagnostic evaluation and evidence-based treatment guidelines for HTN to include lifestyle modifications and pharmacotherapy will be discussed. Finally, this article will examine why the treatment of HTN can prevent the development and reduce the progression of atherosclerosis in vascular disease. (J Vasc Nurs 2016;34:87-92)

Hypertension (HTN) is a major risk factor for cardiovascular disease. HTN is one of several modifiable risk factors for cardiovascular disease to include tobacco use, dyslipidemia, diabetes mellitus, level of physical activity, and weight. Nonmodifiable cardiovascular risk factors include age, gender, and family history of premature cardiovascular disease.[1]

About 80 million U.S. adults (33%) have HTN. Of these individuals, approximately 77% use antihypertensive medication, however, only 54 % have controlled HTN. Approximately 77% of people who have a first stroke have blood pressure (BP) higher than 140/90 mm Hg.[2]

This article will define HTN, list the types of hypertension and their consequences. It will define the diagnostic evaluation and the treatment recommendations from the latest evidence-based guidelines. Lifestyle modifications and pharmacotherapy for HTN will be outlined. Finally, this article will examine why the treatment of HTN is essential in vascular patients.

DEFINITION OF HTN

As early as 1906, the insurance industry suggested a correlation between HTN and longevity by measuring systolic BP (SBP).[3] However, not until the 1940s did medical research (regarding malignant HTN) recognize the harmful cardiovascular effects of HTN.[4]

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BP is defined as the force of blood pushing against the arterial walls as it relates to blood viscosity (thickness) and resistance of blood vessel. SBP is the highest arterial pressure when the heart contracts and empties. Diastolic BP (DBP) is the lowest arterial pressure when the heart relaxes to fill with blood. HTN or high BP is defined as excessive pressure applied to the blood vessel walls.[5]

HTN is a disease of vascular regulation from the alteration of the mechanisms that control arterial pressure within the normal range. The predominant mechanisms of BP control are the central nervous system, the renin-angiotensin-aldosterone system, extracellular fluid volume and the arterial wall stenosis and compliance.[1]

A number of factors can increase BP, including obesity, insulin resistance, high alcohol intake, high salt intake, aging, sedentary lifestyle, stress, low potassium intake, and low calcium intake.[6]

TYPES OF HTN

HTN can be classified as either primary or secondary HTN. Ninety-five percent of all patients with HTN have primary or essential HTN which occurs when the diastolic or systolic pressure exceeds 140/90 mm Hg. An individual is considered hypertensive when the average of two or more properly measured, seated BP readings taken at rest on each of two or more office visits, exceeds the upper limits of normal.[1,7] The actual cause of essential HTN is unknown. However, the following are possible causes of HTN: hyperactivity of sympathetic vasoconstricting nerves, the presence of vasoactive substance released from the arterial endothelial cells causing smooth muscle vasoconstriction, increased cardiac output leading to arteriole constriction, excessive sodium intake, insulin resistance, and hyperinsulinemia.[5]

Secondary HTN occurs in approximately 5% of patients with HTN because of other pathology such as: renal pathology (chronic kidney disease, congenital anomalies, pyelonephritis, acute and chronic glomerulonephritis and hydronephrosis), reduced blood flow to kidney by renal artery stenosis which

MODIFICATION	RECOMMENDATION	AVG. SBP REDUCTION
Weight Reduction	Maintain normal body weight (Body Mass Index 18/.5-24.9 kg/m^2)	5-20 mmHg/10kg
DASH Eating Plan	Adopt a diet rich in fruits, vegetables, lowfat dairy products with reduced content of saturated and total fat	8-14 mmHg
Dietary Sodium Reduction	Reduce dietary sodium intake to less than 100 mmol/day (2.4 g of sodium)	2-8 mmHg
Aerobic Physical Activity	Regular aerobic physical activity at least 30 minutes/day, most days of the week	4-9 mmHg
Moderation of Alcohol Consumption	Men: limit to ≤2 drinks/day Women and lighter weight persons: limit to ≤ 1 drink/day	2-4 mmHg

Figure 1. *Lifestyle modification recommendations (JNC-7[1]).*

causes release of renin which leads to increased BP, coarctation of aorta (stenosis of aorta) causing greater blood flow to upper extremities than to the kidneys and lower extremities causing release of renin, endocrine disturbances such as: pheochromocytoma (a rarely found tumor of the adrenal gland that causes release of epinephrine and norepinephrine and a rise in BP), adrenal cortex tumors leading to an increase in aldosterone secretion (hyperaldosteronism) and an elevated BP, Cushing's syndrome leads to an increase in adrenocortical steroids (causing sodium and fluid retention), hyperthyroidism causes increased cardiac output, obstructive sleep apnea causes nocturnal HTN which leads to sustained daytime HTN.[5,8]

Herbs and medications (either prescription on over-the-counter), can also contribute to HTN. Prescription medications such as estrogens and steroids cause fluid retention, appetite suppressants cause tachycardia and vasoconstriction, sympathomimetics cause vasoconstriction and tachycardia, and antidepressants can prevent the breakdown of epinephrine which can lead to HTN. Nonprescription drugs and substances that can cause HTN include cocaine and decongestants which both cause vasoconstriction and tachycardia, nonsteroidal anti-inflammatory drugs which cause fluid retention and can lead to renal insufficiency, herbal agents (St. John's Wort, ginseng, ephedra) all have an unclear etiology of HTN, nicotine causes vasoconstriction, and food substrates (sodium chloride, ethanol, licorice, and glucose) all cause fluid retention.[5]

CONSEQUENCES OF HTN

HTN can cause intimal wall injury in the arteries leading to atherosclerosis in which smooth muscle cell proliferation, lipid infiltration, and calcium accumulation occur in the vascular epithelium. Prolonged HTN damages small blood vessels in the brain, eyes, heart, and kidneys. Chronic overstretching of the artery wall can lead to arterial dilation, arterial wall tearing or injury.[9]

The ideal objective would be prevention of HTN altogether. However, once diagnosed, the goal in patients with HTN is to prevent target organ damage of the heart (left ventricular hypertrophy [LVH], angina, myocardial infarction, and heart failure), brain (stroke, transient ischemic attack, and dementia), kidneys (chronic kidney disease), eyes (retinopathy and blindness), or vasculature (aneurysm and peripheral arterial disease).[1]

BP MEASUREMENT

BP measurement should be performed with a properly fitting and calibrated instrument. The patient should be seated quietly for at least 5–10 minutes in a chair with their feet on the floor with their arms supported. At least two BP measurements two minutes apart should be recorded and averaged.[1]

Blood pressures in both arms should be measured to evaluate for subclavian stenosis which can lower the BP measurement in the affected arm. Because subclavian stenosis is a predictor for cardiovascular disease, bilateral arm BPs should be routinely and accurately performed, at least at the initial visit. Monitoring BPs in an arm with undiagnosed subclavian artery stenosis, could lead to lack of secondary prevention, ineffective HTN management and failure to uncover asymptomatic cardiovascular disease.[7]

DIAGNOSTIC EVALUATION OF HTN

The following diagnostic studies are helpful to determine the causes and consequences of the damaging effects of HTN. An electrocardiogram (EKG) should be done to determine the effects of HTN on the heart (LVH and ischemia) or underlying heart disease. If the EKG identifies LVH, an echocardiogram should be considered. A chest x-ray may help identify cardiomegaly or aortic dilation by the presence of a widened mediastinum. Proteinuria, elevated serum blood urea nitrogen, and creatinine levels indicate kidney disease as a cause or effect of HTN. Serum potassium is lower in primary hyperaldosteronism and is elevated in Cushing's syndrome, both causes of secondary HTN. A renal scan can detect renal vascular disease and renal artery duplex can identify renal artery stenosis. A 24-hour urine test for catecholamines should be performed, which when elevated, could be an indication of pheochromocytoma. A sleep study should be performed to detect obstructive sleep apnea.[5]

DEVELOPMENT OF HTN GUIDELINES

There have been a number of federal initiatives to encourage application of these scientific advances to the evaluation, treatment, and prevention of HTN. A 1965 report of the President's Commission on Heart Disease, Cancer, and Stroke recommended