VITEBSK STATE MEDICAL UNIVERSITY

CHAIR OF THE FACULTY THERAPY

**FACULTY THERAPY**

(Methodics work out for foreign students, studying the course of faculty therapy)

Part 1

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**HYPERTENSION**

 Hypertension is one of the most common diseases afflicting humans throughout the world. Because of the associated morbidity and mortality and the cost to society, hypertension is an important public health challenge. Over the past several decades, extensive research, widespread patient education, and a concerted effort on the part of health care professionals have led to decreased mortality and morbidity rates from the multiple organ damage arising from years of untreated hypertension. Hypertension is the most important modifiable risk factor for coronary heart disease (the leading cause of death in North America), stroke (the third leading cause), congestive heart failure, end-stage renal disease, and peripheral vascular disease. Therefore, health care professionals must not only identify and treat patients with hypertension but also promote a healthy lifestyle and preventive strategies to decrease the prevalence of hypertension in the general population.

**Frequency**

Forty-three million people in the US are estimated to have hypertension, defined by a systolic blood pressure of 140 mm Hg or greater and/or diastolic blood pressure of 90 mm Hg or greater or defined as those taking antihypertensive medications. The age-adjusted prevalence of hypertension varies from 18-32%, according to data from the National Health Examination Surveys. According to the National Center for Health Statistic Surveys, the awareness for hypertension increased from 53% in 1960-1962 to 89% in 1988-1991. The percentage of patients engaged in hypertension treatment increased from 35% to 79% during this period.

The National High Blood Pressure Education Program (NHBPEP) has reported estimates of hypertension prevalence in United States. The hypertension survey was conducted from 1989-1994, and actual blood pressure and self-reported information was used. Hypertension was defined as systolic blood pressure equal to or more than 140 mm Hg, diastolic blood pressure equal or more than 90 mm Hg, or taking medication for hypertension. The data estimated 43.3 million adults with hypertension in November 1991.

In the Framingham Heart Study, the age-adjusted risk of congestive heart failure was 2.3 times higher in men and 3 times higher in women when highest blood pressure was compared to the lowest. Multiple Risk Factor Intervention Trial (MRFIT) data showed that the relative risk for coronary heart disease mortality varied from 2.3-6.9 times higher for persons with mild-to-severe hypertension compared to persons with normal blood pressure.

The relative risk for stroke ranged from 3.6-19.2. The population-attributable risk percentage for coronary artery disease varied from 2.3-25.6%, whereas the population-attributable risk for stroke ranged from 6.8-40%.

Race: Blacks have a higher prevalence and incidence of hypertension than whites. The prevalence of hypertension was increased by 50% in African Americans. In Mexican Americans, the prevalence and incidence of hypertension is similar to or lower than in whites. The National Health and Nutrition Examination Survey (NHANES) III reported an age-adjusted prevalence of hypertension at 20.6% in Mexican Americans and 23.3% in non-Hispanic whites.

Sex: The age-adjusted prevalence of hypertension was 34%, 25.4%, and 23.2% for men and 31%, 21%, and 21.6% for women among African Americans, whites, and Mexican Americans, respectively. In the NHANES III study, the prevalence of hypertension was 12% for white men and 5% for white women aged 18-49 years. However, the age-related blood pressure rise for women exceeds that of men. The prevalence of hypertension was reported at 50% for white men and 55% for white women aged 70 years or older.

Age: A progressive rise in blood pressure with increasing age is observed. The third NHANES survey reported that the prevalence of hypertension grows significantly with increasing age in all sex and race groups. The age-specific prevalence was 3.3% in white men (aged 18-29 y); this increased to 13.2% in the group aged 30-39 years. The prevalence further increased to 22% in the group aged 40-49 years, to 37.5% in the group aged 50-59 years, and to 51% in the group aged 60-74 years. In another study, the incidence of hypertension appeared to increase approximately 5% for each 10-year interval of age. Age-related hypertension appears to be predominantly systolic rather than diastolic. The systolic blood pressure rises into the eighth or ninth decade, while the diastolic blood pressure remains constant or declines after age 40 years (Cornoni-Huntley, 1989).

**Historical perspectives**

Blood pressure was measured for the first time by Stephen Hales in 1773. Hales also described the importance of blood volume in blood pressure regulation. The contribution of peripheral arterioles in maintaining blood pressure, described as "tone," was first described by Lower in 1669 and subsequently by Sénac in 1783. The role of vasomotor nerves in the regulation of blood pressure was observed by such eminent investigators as Claude Bernard, Charles E. Edouard, Charles Brown-Séquard, and Augustus Waller. William Dayliss advanced this concept in a monograph published in 1923. Cannon and Rosenblueth developed the concept of humoral control of blood pressure and investigated pharmacologic effects of epinephrine. Three contributors who advanced the knowledge of humoral mechanisms of blood pressure control are T.R. Elliott, Sir Henry Dale, and Otto Loew.

Richard Bright, a physician who practiced in the first half of the 19th century, observed the changes of hypertension on the cardiovascular system in patients with chronic renal disease. George Johnson in 1868 postulated that the cause of left ventricular hypertrophy (LVH) in Bright disease was the presence of muscular hypertrophy in the smaller arteries throughout the body. Further clinical pathologic studies by Sir William Gull and H.G. Sutton (1872) led to further description of the cardiovascular changes of hypertension. Frederick Mahomed was one of the first physicians to systematically incorporate blood pressure measurement as a part of a clinical evaluation.

The recognition of primary, or essential, hypertension is credited to the work of Huchard, Vonbasch, and Albutt. Observations of Janeway and Walhard led to the recognition of target organ damage, which branded hypertension as the "silent killer." The concepts of renin, angiotensin, and aldosterone were advanced by several investigators in the late 19th and early 20th centuries. The names of Irwine, Page, van Slyke, Goldblatt, Laragh, and Tuttle prominently appear throughout the hypertension literature, and their work enhances our understanding of the biochemical basis of essential hypertension. Cushman and Ondetti developed an orally acting converting enzyme inhibitor from snake venom peptides and are credited with the successful synthesis of the modern antihypertensive captopril.

**Definition**

Defining abnormally high blood pressure is extremely difficult and arbitrary. Furthermore, the relationship between systemic arterial pressure and morbidity appears to be quantitative rather than qualitative. A level for high blood pressure must be agreed upon in clinical practice for screening patients with hypertension and for instituting diagnostic evaluation and initiating therapy. Because the risk to an individual patient may correlate with the severity of hypertension, a classification system is essential for making decisions about aggressiveness of treatment or therapeutic interventions.

Based on recommendations of the Seventh Report of the Joint National Committee of Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VII), the classification of blood pressure (expressed in mm Hg) for adults aged 18 years or older is as follows\*:

**Norma** - Systolic lower than 120, diastolic lower than 80

**Prehypertension** - Systolic 120-139, diastolic 80-99

**Stage 1** - Systolic 140-159 or diastolic 90-99

**Stage 2** - Systolic equal to or more than 160 or diastolic equal to or more than 100

*\*Based on the average of 2 or more readings taken at each of 2 or more visits after initial screening*

Normal blood pressure with respect to cardiovascular risk is less than 120/80 mm Hg. However, unusually low readings should be evaluated for clinical significance.

Prehypertension, a new category designated in the JNC VII report, emphasizes that patients with prehypertension are at risk for progression to hypertension and that lifestyle modifications are important preventive strategies.

Staging: BP and hypertension itself have been divided into the following stages:

Stages of Blood Pressure Elevation and Hypertension

Category Systolic BP Diastolic BP

 mm Hg mm Hg

Optimal <120 <80

Normal <130 <85

High-normal 130-139 85-90

Stage I 140-159 90-99

Stage II 160-179 100-109

Stage III >180 >110

According to the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7), the normal and high-normal stages are now (as of 2003) grouped together as the prehypertension stage. Stage I is the same, and stage III and IV are grouped under stage II.

Hypertension may be either essential or secondary. Essential hypertension is diagnosed in the absence of an identifiable secondary cause. Approximately 95% of American adults have essential hypertension, while secondary hypertension accounts for fewer than 5% of the cases.

**Causes**

* Primary or essential hypertension (90-95%)
* Secondary hypertension: A small percentage of patients (2-10%) have a secondary cause.

The following is a list of secondary causes of hypertension:

* Renal (2.5-6%)

Renal parenchymal disease

Polycystic kidney disease

Urinary tract obstruction

Renin-producing tumor

Liddle syndrome

Renovascular hypertension (0.2-4%)

* Vascular

Coarctation of aorta

Vasculitis

Collagen vascular disease

* Endocrine (1-2%)

 Oral contraceptives

Adrenal

Primary aldosteronism

Cushing syndrome

Pheochromocytoma

Congenital adrenal hyperplasia

Hyperthyroidism and hypothyroidism

Hypercalcemia

Hyperparathyroidism

Acromegaly

* Neurogenic

Brain tumor

Bulbar poliomyelitis

Intracranial hypertension

* Pregnancy-induced hypertension
* Drugs and toxins

Alcohol

Cocaine

Cyclosporin

Erythropoietin

Adrenergic medications

**Pathophysiology**

 Arterial blood pressure is a product of cardiac output and systemic vascular resistance. Therefore, determinants of blood pressure include factors that affect both cardiac output and arteriolar vascular physiology. There is potential relevance of blood viscosity, vascular wall sheer conditions (rate and stress), and blood flow velocity (mean and pulsatile components) on vascular and endothelial function regulating blood pressure in humans. Furthermore, changes in vascular wall thickness affect the amplification of peripheral vascular resistance in hypertensive patients and result in reflection of waves back to the aorta, increasing systolic blood pressure.

**Regulation of blood pressure**

Regulation of normal blood pressure is a complex process. Although a function of cardiac output and peripheral vascular resistance, both of these variables are influenced by multiple factors.

The factors affecting cardiac output include

sodium intake,

renal function,

mineralocorticoids;

the inotropic effects occur via extracellular fluid volume augmentation

an increase in heart rate and contractility.

Peripheral vascular resistance is dependent upon the sympathetic nervous system, humoral factors, and local autoregulation.

The sympathetic nervous system produces its effects via the vasoconstrictor alpha effect or the vasodilator beta effect.

The humoral actions on peripheral resistance are also mediated by other mediators such as vasoconstrictors (angiotensin and catecholamines) or vasodilators (prostaglandins and kinins).

Autoregulation of blood pressure occurs by way of intravascular volume contraction and expansion, as well as by transfer of transcapillary fluid. Interactions between cardiac output and peripheral resistance are autoregulated to maintain a set blood pressure in an individual. For example, constriction of the arterioles elevates arterial pressure by increasing total peripheral resistance, whereas venular constriction leads to redistribution of the peripheral intravascular volume to the central circulation, thereby increasing preload and cardiac output.

**Pathogenesis of hypertension**

The pathogenesis of essential hypertension is multifactorial and highly complex.

 Multiple factors modulate the blood pressure for adequate tissue perfusion and include

 humoral mediators,

vascular reactivity,

circulating blood volume,

vascular caliber, blood viscosity,

cardiac output,

blood vessel elasticity,

neural stimulation.

A possible pathogenesis of essential hypertension has been proposed in which multiple factors, including genetic predisposition, excess dietary salt intake, and adrenergic tone, may interact to produce hypertension. Although genetics appears to contribute to essential hypertension, the exact mechanism has not been established.

The natural history of essential hypertension evolves from occasional to established hypotension. After a long invariable asymptomatic period, persistent hypertension develops into complicated hypertension, in which target organ damage to the aorta and small arteries, heart, kidneys, retina, and central nervous system is evident. The progression begins with prehypertension in persons aged 10-30 years (by increased cardiac output) to early hypertension in persons aged 20-40 years (in which increased peripheral resistance is prominent) to established hypertension in persons aged 30-50 years, and, finally, to complicated hypertension in persons aged 40-60 years.

The early stage of hypertension has been described as high-output hypertension. High-output hypertension results from decreased peripheral vascular resistance and concomitant cardiac stimulation by adrenergic hyperactivity and altered calcium homeostasis. In contrast, the chronic phase of essential hypertension characteristically has normal or reduced cardiac output and elevated systemic vascular resistance.

The vasoreactivity of the vascular bed, an important phenomenon mediating changes of hypertension, is influenced by the activity of vasoactive factors, reactivity of the smooth muscle cells, and structural changes in the vessel wall and vessel caliber, expressed by a lumen-to-wall ratio. Patients who develop hypertension are known to develop a systemic hypertensive response secondary to vasoconstrictive stimuli. Alterations in structural and physical properties of resistance arteries, as well as changes in endothelial function, are probably responsible for this abnormal behavior of vasculature. Furthermore, vascular remodeling occurs over the years as hypertension evolves, thereby maintaining increased vascular resistance irrespective of the initial hemodynamic pattern.

**Genetic factors**

Hypertension is likely to be related to multiple genes. Hypertension develops secondary to multiple environmental factors, as well as to several genes, whose inheritance appears to be complex. Very rare secondary causes are related to single genes.

**Role of the vascular endothelium**

The vascular endothelium is presently considered a vital organ, where synthesis of various vasodilating and constricting mediators occurs. The interaction of autocrine and paracrine factors takes place in the vascular endothelium, leading to growth and remodeling of the vessel wall and to the hemodynamic regulation of blood pressure.

Numerous hormonal, humeral vasoactive, and growth and regulating peptides are produced in the vascular endothelium.

These mediators include angiotensin II, bradykinin, endothelin, nitric oxide, and several other growth factors.

 Endothelin is a potent vasoconstrictor and growth factor that likely plays a major role in the pathogenesis of hypertension.

Angiotensin II is a potent vasoconstrictor synthesized from angiotensin I with the help of an angiotensin-converting enzyme (ACE).

Another vasoactive substance manufactured in the endothelium is nitric oxide. Nitric oxide is an extremely potent vasodilator that influences local autoregulation and other vital organ functions.

Additionally, several growth factors are manufactured in the vascular endothelium; each of these plays an important role in atherogenesis and target organ damage. These factors include platelet-derived growth factor, fibroblast growth factor, insulin growth factor, and many others.

**Pathophysiology of target organ damage**

* Hypertension and the cardiovascular system

Cardiac involvement in hypertension manifests as

 LVH,

left atrial enlargement,

aortic root dilatation,

atrial and ventricular arrhythmias,

systolic and diastolic heart failure,

 ischemic heart disease.

LVH is associated with an increased risk of premature death and morbidity. A higher frequency of cardiac atrial and ventricular dysrhythmias and sudden cardiac death may exist. Possibly, increased coronary arteriolar resistance leads to reduced blood flow to the hypertrophied myocardium, resulting in angina despite clean coronary arteries. Hypertension, along with reduced oxygen supply and other risk factors, accelerates the process of atherogenesis, thereby further reducing oxygen delivery to the myocardium.

Hypertension remains the most common cause of congestive heart failure. Antihypertensive therapy has been demonstrated to significantly reduce the risk of death from stroke and coronary heart disease. Two published meta-analyses have shown 14% and 26% reductions in cardiovascular mortality rates.

* Left ventricular hypertrophy

The myocardium undergoes structural changes in response to increased afterload. Cardiac myocytes respond by hypertrophy, allowing the heart to pump more strongly against the elevated pressure. However, the contractile function of the left ventricle remains normal until later stages. Eventually, LVH lessens the chamber lumen, limiting diastolic filling and stroke volume. The left ventricular diastolic function is markedly compromised in long-standing hypertension.

The mechanisms of diastolic dysfunction have been elucidated only recently. An aberration in the passive relaxation of the left ventricle during diastole appears to exist. Over time, fibrosis may occur, further contributing to the poor compliance of the ventricle.

As the left ventricle does not relax during early diastole, left ventricular end-diastolic pressure increases, further increasing left atrial pressure in late diastole. The exact determinants of left ventricular diastolic dysfunction have not been well studied; possibly, the abnormality is governed by abnormal calcium kinetics.

* The central nervous system

Long-standing hypertension may manifest as hemorrhagic and atheroembolic stroke or encephalopathy. Both the high systolic and diastolic pressures are harmful; a diastolic pressure of more than 100 mm Hg and a systolic pressure of more than 160 mm Hg have led to a significant incidence of strokes. Other cerebrovascular manifestations of complicated hypertension include hypertensive hemorrhage, hypertensive encephalopathy, lacunar-type infarctions, and dementia.

* Renal disease

Despite widespread treatment of hypertension in the United States, the incidence of end-stage renal disease continues to rise. The explanation for this rise may be concomitant diabetes mellitus, the progressive nature of hypertensive renal disease despite therapy, or a failure to reduce blood pressure to a protective level. A reduction in renal blood flow in conjunction with elevated afferent glomerular arteriolar resistance increases glomerular hydrostatic pressure secondary to efferent glomerular arteriolar constriction. The result is glomerular hyperfiltration, followed by development of glomerulosclerosis and further impairment of renal function.

Two studies (Shulman, 1989; Pettinger, 1973) have demonstrated that a reduction in blood pressure may result in improved renal function.

 Therefore, earlier detection of hypertensive nephrosclerosis using means to detect microalbuminuria and aggressive therapeutic interventions, particularly with ACE inhibitor drugs, may prevent progression to end-stage renal disease.

Nephrosclerosis is one of the possible complications of long-standing hypertension. The risk of hypertension-induced end-stage renal disease is higher in black patients, even when the blood pressure is under good control. Furthermore, patients with diabetic nephropathy who are hypertensive are also at high risk for developing end-stage renal disease.

The renin-angiotensin system activity influences the progression of renal disease. Angiotensin II acts at both the afferent and the efferent arterioles, but more so on the efferent arteriole, which leads to an increase of the intraglomerular pressure. The excess glomerular pressure leads to microalbuminuria. Reducing intraglomerular pressure using an ACE inhibitor has been shown to be beneficial in patients with diabetic nephropathy, even in those who are not hypertensive. The beneficial effect of ACE inhibitors on the progression of renal insufficiency in patients who are nondiabetic is less clear.

* Hypertension in renal disease

Hypertension is commonly observed in patients with kidney disease. Volume expansion is the main cause of hypertension in patients with glomerular disease (nephrotic and nephritic syndrome). Hypertension in patients with vascular disease is the result of the activation of the renin-angiotensin system, which is often secondary to ischemia. Most patients with chronic renal failure are hypertensive (80-90%). The combination of volume expansion and the activation of the renin-angiotensin system is believed to be the main factor behind hypertension in patients with chronic renal failure.

**CLINIC**

**History**

Following the documentation of hypertension, which is confirmed after an elevated blood pressure, properly measured, has been documented on at least 3 separate occasions (based on the average of 2 or more readings taken at each of 2 or more visits after initial screening), a detailed history should extract the following information:

* Extent of target organ damage
* Assessment of patients' cardiovascular risk status
* Exclusion of secondary causes of hypertension
* Patients may have undiagnosed hypertension for years without having had their blood pressure checked. Therefore, a careful history of end organ damage should be obtained.
* A history of cardiovascular risk factors includes

hypercholesterolemia,

diabetes mellitus,

 tobacco use (including chewing tobacco).

* Obtain a history of over-the-counter medication use, current and previous unsuccessful antihypertensive medication trials, and ethanol intake.
* The historical and physical findings that suggest the possibility of secondary hypertension are a history of known renal disease, abdominal masses, anemia, and urochrome pigmentation.
* A history of sweating, labile hypertension, and palpitations suggests the diagnosis of pheochromocytoma.
* A history of cold or heat tolerance, sweating, lack of energy, and bradycardia or tachycardia may indicate hypothyroidism or hyperthyroidism.
* A history of weakness suggests hyperaldosteronism. Abdominal bruit suggests the possibility of renal artery stenosis. Absence of femoral pulses suggests coarctation of aorta.
* Kidney stones raise the possibility of hyperparathyroidism. The presence of papilledema and other neurologic signs raises the possibility of increased intracranial pressure. A history of drug ingestion, including oral contraceptives, licorice, and sympathomimetics, should be obtained.

**Physical examination**

 An accurate measurement of blood pressure is the key to diagnosis. Several determinations should be made over a period of several weeks.

At any given visit, an average of 3 blood pressure readings taken 2 minutes apart using a mercury manometer is preferable. Blood pressure should be measured in both the supine and sitting positions, auscultating with the bell of the stethoscope. On the first visit, blood pressure should be checked in both arms and in one leg to avoid missing the diagnosis of coarctation of aorta or subclavian artery stenosis.

As the improper cuff size may influence blood pressure measurement, a wider cuff is preferable, particularly if the patient's arm circumference exceeds 30 cm.

The patient should rest quietly for at least 5 minutes before the measurement.

Although somewhat controversial, the common practice is to document phase V (a disappearance of all sounds) of Korotkoff sounds as the diastolic pressure.

A funduscopic evaluation of the eyes should be performed to detect any evidence of hypertensive retinopathy. These are flame-shaped hemorrhages and cotton wool exudates.

Palpation of all peripheral pulses should be performed.

Look for renal artery bruit over the upper abdomen; the presence of a unilateral bruit with both a systolic and diastolic component suggests renal artery stenosis.

A careful cardiac examination is performed to evaluate signs of LVH. These include displacement of apex, a sustained and enlarged apical impulse, and the presence of an S4. Occasionally, a tambour S2 is heard with aortic root dilatation.

**Lab Studies**

Unless a secondary cause for hypertension is suspected, only the following routine laboratory studies should be performed:

CBC count,

serum electrolytes,

serum creatinine,

serum glucose,

 uric acid,

 urinalysis

Lipid profile (total cholesterol, low-density lipoprotein [LDL] and high-density lipoprotein [HDL], and triglycerides)

Additional tests described below are indicated when specific clinical situations warrant further investigation.

* Microalbuminuria is an early indication of hypertensive nephrosclerosis and is also a marker for a higher risk of cardiovascular morbidity and mortality. Present recommendations suggest that individuals with type I diabetes should be screened for microalbuminuria. Usefulness of this screening in hypertensive patients without diabetes has not been established.
* Plasma renin activity (PRA) is performed to detect evidence of primary hyperaldosteronism. Low renin values confirm the diagnosis of primary hyperaldosteronism; however, hypokalemia may be associated with a form of hypertension, but it is not often present.
* Determination of sensitive thyroid-stimulating hormone (TSH) level excludes hypothyroidism or hyperthyroidism as a cause of hypertension.

**Imaging Studies**

Ambulatory blood pressure monitoring: Indications for ambulatory blood pressure monitoring include labile blood pressure, a discrepancy between blood pressure measurement inside and outside the physician's office, and poor blood pressure control. Ambulatory monitoring also identifies patients who have the distinct syndrome called white coat hypertension.

Routine testing includes electrocardiograms.

Echocardiography: The limited echocardiography study, rather than the complete examination, may detect LVH more frequently than electrocardiography. The main indication for limited echocardiography is evaluation for end organ damage in a patient with borderline high blood pressure. Therefore, the presence of LVH despite normal or borderline high blood pressure measurements requires antihypertensive therapy.

Imaging studies for renovascular stenosis: If the history suggests renal artery stenosis and a corrective procedure is considered, further radiologic investigations are performed.

**TREATMENT**

Medical Care:

Consider lifestyle modifications.

 As the cardiovascular disease risk factors are assessed in individuals with hypertension, pay attention to the lifestyles that favorably affect blood pressure level and reduce overall cardiovascular disease risk. A relatively small reduction in blood pressure may affect the incidence of cardiovascular disease on a population basis. A decrease in blood pressure of 2 mm Hg reduces the risk of stroke by 15% and the risk of coronary artery disease by 6% in a given population.

JNC VII recommendations to lower blood pressure and decrease cardiovascular disease risk include the following:

* Lose weight if overweight.

Limit alcohol intake to no more than 1 oz (30 mL) of ethanol (ie, 24 oz [720 mL] of beer, 10 oz [300 mL] of wine, 2 oz [60 mL] of 100-proof whiskey) per day or 0.5 (15 mL) ethanol per day for women and people of lighter weight.

* Increase aerobic activity (30-45 min most days of the week).
* Reduce sodium intake to no more than 100 mmol/d (2.4 g sodium or 6 g sodium chloride).
* Maintain adequate intake of dietary potassium (approximately 90 mmol/d).
* Maintain adequate intake of dietary calcium and magnesium for general health.
* Stop smoking and reduce intake of dietary saturated fat and cholesterol for overall cardiovascular health.

The medical care of patients with hypertensive heart disease falls under 2 categories—treatment of the elevated BP and prevention and treatment of hypertensive heart disease. Various treatment strategies include dietary modifications, regular aerobic exercise, weight loss, and pharmacotherapy directed toward hypertension, heart failure secondary to diastolic and systolic LV dysfunction, coronary artery disease, and arrhythmias.

* Dietary modifications

Studies have shown that diet and a healthy lifestyle alone or in combination with medical treatment can not only lower the BP and decrease the symptoms of heart failure, but also reverse LVH. Specific diet recommendations include a diet low in sodium, high in potassium (in patients with normal renal function), rich in fresh fruits and vegetables, and low in cholesterol, and low alcohol consumption.

A low-sodium diet, alone or in combination with pharmacotherapy, has been shown by numerous studies to reduce BP in patients with hypertension, with a more prominent response in a subset of patients with hypertension—mainly African Americans—with low renin levels. Restriction of sodium in these patients does not lead to compensatory stimulation of the renin-angiotensin system and thus has a potent antihypertensive effect. Restriction of daily sodium intake to 50-100 mmol, equivalent to 3-6 g of salt per day, is recommended.

"A diet rich in fresh fruits and vegetables (DASH diet) has been shown to significantly lower the BP in patients with hypertension despite a constant sodium content." This diet should be advised in patients with hypertension.

A low-cholesterol diet is part of secondary prophylaxis in patients with coronary artery disease. It is also a part of the primary prophylaxis of coronary artery disease in patients at high risk for this disease.

* Heavy alcohol consumption has been associated not only with high BP but also with an increase in LV mass. Moderation in alcohol consumption is advised; no more than 1-2 drinks per day is recommended.
* Regular aerobic exercise

Regular dynamic isotonic exercise, such as walking, running, swimming, and cycling, has been shown not only to decrease BP but also to improve cardiovascular well-being. Regular isotonic exercise has additional favorable cardiovascular effects, including improved endothelial function, peripheral vasodilatation, reduced resting heart rate, improved heart rate variability, and reduced plasma levels of catecholamines.

Regular 30-minute sessions of aerobic exercise 3-4 times a week should be advised. Isometric and strenuous exercise should be avoided.

* Weight reduction

Obesity has been linked to hypertension and LVH in various epidemiological studies, with as many as 50% of obese patients having some degree of hypertension and as many as 60-70% of patients with hypertension being obese. Abdominal adiposity, clinically measured as waist-to-hip ratio and more accurately assessed by abdominal CT scan, is a more sensitive risk factor for hypertension. Studies have shown that weight reduction is one of the most effective ways to reduce BP.

Gradual weight reduction (1 kg/wk) should be advised. Pharmacological interventions to reduce weight should be used with great caution because diet pills, especially those available over the counter, frequently contain sympathomimetics. These not only can raise BP but also can worsen angina or symptoms of heart failure and exacerbate tendencies for cardiac arrhythmias.

**Pharmacotherapy**

Treatment of hypertension and hypertensive heart disease can involve the following classes of antihypertensive medications: thiazide diuretics, beta-blockers and combined alpha- and beta-blockers, calcium channel blockers, ACE inhibitors, angiotensin receptor blockers (ARBs), and direct vasodilators such as hydralazine.

Thiazide diuretics and beta-blockers are the drugs of first choice in patients with uncomplicated hypertension as outlined by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI). Calcium channel blockers specifically are effective in systolic hypertension in elderly patients. ACE inhibitors are the first choice in patients with diabetes and/or LV dysfunction. ARBs are a reasonable alternative, especially for patients with adverse effects with ACE inhibitors. Peripheral alpha-channel blockers should not be used in patients with hypertension in view of recent findings of their adverse effect on cardiovascular morbidity and mortality rates. Central alpha-antagonists have no evidence-based support and have excess adverse effects.

Intravenous drugs used in patients with hypertensive emergency include the following: nitroprusside, labetalol, hydralazine, enalapril, and beta-blockers (avoided in acutely decompensated heart failure).

Treatment of LVH

LVH, a marker of increased risk of cardiovascular morbidity and mortality, should be treated aggressively. Whether regression in LVH leads to improvement in cardiovascular mortality and morbidity rates is not clear, although limited data support this hypothesis.

All the medications already listed for the treatment of hypertension have been shown to reduce LVH. Limited meta-analysis data suggest a slight advantage to ACE inhibitors.

Treatment of LV diastolic dysfunction

Certain classes of antihypertensives—ACE inhibitors, beta-blockers, and nondihydropyridine calcium channel blockers—have been shown (although not consistently) to improve echocardiographic parameters in symptomatic and asymptomatic diastolic dysfunction and the symptomatology of heart failure.

Use diuretics and nitrates with caution in patients with heart failure due to diastolic dysfunction. These drugs may cause severe hypotension by inappropriately decreasing the preload, which is required for adequate LV filling pressures. Delicate titration of diuretics is necessary if they are indicated.

Hydralazine has been shown to cause severe hypotension in patients with heart failure due to diastolic dysfunction.

By increasing intracellular calcium level, digoxin can worsen LV stiffness. However, a large randomized trial has not shown any increase in mortality rate.

Treatment of LV systolic dysfunction

Diuretics (predominantly loop diuretics) are used in the treatment of LV systolic dysfunction.

ACE inhibitors are used for preload and afterload reduction and prevention of pulmonary or systemic congestion. They have been shown to decrease morbidity and mortality rates in patients with heart failure due to systolic dysfunction. The aim should be to use the target dose or the maximum tolerable doses. ACE inhibitors also are indicated in patients with asymptomatic LV dilatation and dysfunction.

Beta-blockers (cardioselective or mixed alpha and beta), such as carvedilol, metoprolol XL, and bisoprolol, have been shown to improve LV function and decrease rates of mortality and morbidity from heart failure. Recent trials also have shown improvement in outcome of patients in NYHA class IV heart failure with carvedilol. These drugs should be started when the patient has no signs of fluid overload and is in compensated heart failure. Therapy should be initiated with low doses, increasing the dose of the beta-blocker very slowly and closely monitoring the patient for signs of worsening heart failure.

Low-dose spironolactone has been shown to decrease the rates of morbidity and mortality in patients in NYHA class III or IV heart failure who already are taking ACE inhibitors.

Treatment of cardiac arrhythmias

Treatment depends upon the specific arrhythmia and the underlying LV function.

Anticoagulation should be considered in patients with atrial fibrillation.

The management of secondary hypertension may result in cure by the surgical correction of the underlying problem, such as removal of a pheochromocytoma. Surgery may not be feasible in a substantial number of patients for whom medical therapy is instituted to control hypertension.

* Renovascular hypertension

The goals of therapy are maintenance of normal blood pressure and prevention of end-stage renal disease. The therapeutic options include medical therapy, percutaneous transluminal renal angioplasty, and surgical revascularization. These options must be individualized because no randomized studies document the superiority of one option over the other. The indications for surgery or angioplasty include an inability to control blood pressure while on a medical regimen, the need to preserve renal function, and intolerable effects of medical therapy.

ACE inhibitors are quite effective in patients with unilateral renal artery stenosis; however, avoid ACE inhibitors in patients with bilateral renal artery stenosis or stenosis of a solitary kidney. A diuretic can be combined with an ACE inhibitor. Because of their glomerular vasodilatory effect, calcium antagonists are effective in renal artery stenosis and do not compromise renal function.

* Pheochromocytoma

Following suspicion of pheochromocytoma, the presence of a tumor should be confirmed biochemically by measuring urine and plasma concentrations of catecholamine or their metabolites. In most situations, a CT scan or an MRI may be used to localize the tumor in the abdomen. In the absence of abdominal imaging, nuclear scan with metaiodobenzylguanidine (MIBG) may further help with the localization.

Surgical resection is the treatment of choice because hypertension is cured by tumor resection. In the preoperative phase, combined alpha- and beta-adrenergic blockade is recommended for hypertension control. Alpha-adrenergic blockade is initiated with phenoxybenzamine or prazosin, and, following adequate alpha-adrenergic blockade, beta-adrenergic blockade is initiated. These patients are often volume contracted and require saline or sodium tablets. Catecholamines can be reduced further by metyrosine. For adrenal pheochromocytoma, laparoscopic adrenalectomy is becoming the procedure of choice in suitable patients. Follow-up 24-hour urinary excretion studies of catecholamines should be performed 2 weeks following surgery (and periodically thereafter) to detect recurrence, metastases, or development of second primary lesion.

* Primary hyperaldosteronism

Hyperkalemia is an important clue to the presence of primary aldosteronism. However, in a subset of patients, the serum potassium concentration may be within the reference range. Measurement of PRA has been used as a screening test. A suppressed PRA value that fails to rise above 2 mg/mL/h after salt and water depletion is considered a positive test result. The best initial test is the determination of the aldosterone excretion rate during prolonged salt loading.

The appropriate therapy depends on the cause of excessive aldosterone production. A CT scan may help localize an adrenal mass, indicating adrenal adenoma. If the results of the CT scan are inconclusive, adrenal venous sampling for aldosterone and cortisol levels should be performed. Medical therapy is indicated in patients with adrenal hyperplasia, patients with adenoma who are poor surgical risks, and patients with bilateral adenomas. These patients are best treated with sustained salt and water depletion. Hydrochlorothiazide or furosemide in combination with either spironolactone or amiloride corrects hypokalemia and normalizes the blood pressure. Some patients may require the addition of a vasodilator or a beta-blocker for better control of hypertension.

Adrenal adenomas may be resected via a laparoscopic procedure. Surgical resection often leads to the control of blood pressure and the reversal of biochemical abnormalities. These patients may develop hypoaldosteronism during the postoperative follow-up period and require supplementation with fludrocortisone.

**HYPERTENSIVE EMERGENCIES**

**Frequency**

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Morbidity and mortality depend on the extent of end-organ damage on presentation and the degree to which BP is controlled subsequently. BP control may prevent progression to end-organ impairment.

One-year mortality rate for an untreated hypertensive emergency is greater than 90%.

Five-year survival rate among all patients presenting with a hypertensive crisis is 74%.

Median survival is 144 months for all patients presenting to the ED with a hypertensive crisis.

Males are at greater risk of hypertensive emergencies than females.

Hypertensive emergencies occur most commonly in middle-aged patients. The peak incidence occurs in those aged 40-50 years.

**Causes**

The most common hypertensive emergency is a rapid unexplained rise in BP in a patient with chronic essential hypertension.

Other causes

Renovascular hypertension

Eclampsia, pre-eclampsia

Acute glomerulonephritis

Pheochromocytoma

Antihypertensive withdrawal syndromes

Head injuries and CNS trauma

Renin-secreting tumors

Drug-induced hypertension

Burns

Vasculitis

Thrombotic thrombocytopenic purpura

Idiopathic hypertension

Postoperative hypertension

Coarctation of aorta

Patients with hypertension in the ED can be classified into 3 categories based upon their symptoms and the organ systems that are affected at the time of presentation.

1. Hypertensive emergency

Hypertensive emergency, also called hypertensive crisis, is severe hypertension with acute impairment of an organ system (eg, central nervous system [CNS], cardiovascular, renal). In these conditions, the blood pressure (BP) should be lowered aggressively over minutes to hours.

1. Hypertensive urgency

This is distinguished from hypertensive urgency, in which the BP is a potential risk but has not yet caused acute end-organ damage. These patients require BP control over several days to weeks.

1. Severe hypertension

The third category, severe hypertension, is elevated BP not yet leading to significant organ damage. In these patients, the hypertension does not necessarily require treatment during the ED visit but does require close follow-up with a primary care physician for long-term BP control. In these cases, beginning antihypertensive therapy in the ED may be appropriate and should be done in consultation with the patient's primary care physician, who will be caring for the patient after the ED visit.

**Emergency department considerations**

* Optimal control of hypertensive situations balances the benefits of immediate decreases in BP against the risk of significant decrease in end-organ perfusion. The emergency physician must be capable of the following:
* Appropriately evaluating patients with an elevated BP
* Correctly classifying the hypertension
* Determining the aggressiveness and timing of therapeutic interventions
* Making disposition decisions
* An important point to remember in the management of the patient with any degree of BP elevation is to "treat the patient and not the number."

**Pathophysiology**

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* Central nervous system

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 This results in transudate leak across capillaries and continued arteriolar damage. Subsequent fibrinoid necrosis causes normal autoregulatory mechanisms to fail, leading to clinically apparent papilledema, the sine qua non of malignant hypertension. The end result of loss of autoregulation is hypertensive encephalopathy.

* Cardiovascular system

The cardiovascular system is affected as increased cardiac workload leads to cardiac failure; this is accompanied by pulmonary edema, myocardial ischemia, or myocardial infarction.

* Renal system

The renal system is impaired when high BP leads to arteriosclerosis, fibrinoid necrosis, and an overall impairment of renal protective autoregulation mechanisms. This may manifest as worsening renal function, hematuria, red blood cell (RBC) cast formation, and/or proteinuria.

**History**

Focus history on the presence of end-organ damage, the circumstances surrounding the hypertension, and any identifiable etiology.

* Medications

Use of hypertensive medications and compliance

Use of illicit drugs (specifically alpha-adrenergic agents)

Other medication history

* Duration of hypertension

Duration of current symptoms

* Other medical problems (eg, prior hypertension, thyroid disease, Cushing disease, systemic lupus, renal disease)
* Date of last menstrual period
* CNS manifestations

Headaches (85%): Mild headache alone in association with elevated BP does not indicate a hypertensive crisis.

New-onset blurred vision (60%)

Weight loss (75%)

Nausea and vomiting

Weakness and fatigue (30%)

Confusion and mental status changes

* Cardiovascular manifestations

Symptoms of congestive heart failure (CHF)

Angina

Dissecting aneurysm

* Renal manifestations

History of hematuria

Oliguria

* Other manifestations

Abdominal pain

Shortness of breath

Visual disturbances

**Physical**

Use an approach based on organ systems to identify signs of end-organ damage.

1. CNS

Focal neurologic findings

Seizures, stupor, coma

Papilledema, hemorrhages, exudates, or evidence of closed-angle glaucoma

1. Cardiovascular

Lung auscultation for evidence of pulmonary edema

Signs of CHF, including extra heart sounds

Jugular venous distension

Peripheral edema

Check for equal and symmetric BP and pulses bilaterally.

Check for abdominal masses and bruits.

**Lab Studies**

Electrolytes, BUN, and creatinine for evidence of renal impairment CBC

Dipstick urinalysis to detect presence of hematuria or proteinuria as evidence of renal impairment

Microscopic urinalysis, evaluating for RBCs or RBC casts as evidence of renal impairment

Optional studies

Toxicology screen

Endocrine testing

Pregnancy test

**Imaging Studies**

Electrocardiogram (ECG) to assess for evidence of ischemia or infarction

Chest x-ray - Signs of CHF, pulmonary edema, or coarctation of aorta

Head CT scan - Indicated with abnormal neurologic exam to look for intracranial bleeding, edema, or infarction

Chest CT scan, transesophageal echo, or aortic angiogram - May be indicated for clinical suspicion of aortic dissection

**TREATMENT**

**Prehospital Care:**

Address the manifestations of a hypertensive emergency, such as chest pain or heart failure. Reduction of BP may not be indicated in the prehospital setting.

Oxygen, furosemide (Lasix), and nitrates all may be appropriate.

Under most circumstances, attempting to treat hypertension directly in the prehospital setting is unwise. In particular, rapid lowering of BP can critically decrease end-organ perfusion.

**Emergency Department Care:**

The fundamental principle in determining the necessary ED care of the hypertensive patient is the presence or absence of end-organ damage.

Initial considerations (if the patient is not in distress)

Place patient who is not in distress in a quiet room and reevaluate after an initial interview. In one study, 27% of patients with an initial DBP >130 mm Hg had their DBP fall below critical levels after relaxation without specific treatment.

Consider the context of the elevated BP (eg, severe pain often causes increase in BP).

Screen for end-organ damage

Use historical criteria, physical examination steps, lab studies, and diagnostic tests outlined in Workup.

Patients with end-organ damage usually require admission and rapid lowering of BP using intravenous (IV) medications. Suggested medication depends on the affected organ system.

Patients without evidence of end-organ effects may be discharged with follow-up.

The misconception remains that a patient never should be discharged from the ED with elevated BP. As a result of this belief, patients are given oral medicines, such as nifedipine, in an effort to lower BP rapidly before discharge. This is not indicated and may be dangerous.

Attempts to temporarily lower BP by using these medicines may result in a precipitous and difficult-to-correct drop in BP. Should this occur, end-organ hypoperfusion may result. Furthermore, patients who present with high BP may have had this elevation for some time and may need chronic BP control but may not tolerate rapid return of BP to a "normal" level.

Acute lowering of BP in the narrow window of the ED visit does not necessarily improve long-term morbidity and mortality rates. The follow-up recommended for these situations by The Joint National Committee on High Blood Pressure is outlined in Follow-up.

Rapid BP reduction is indicated in the following circumstances:

**Acute myocardial ischemia**

* Nitroglycerin IV
* Beta-blockers IV
* Angiotensin-converting enzyme (ACE) inhibitors IV

**CHF with pulmonary edema**

* Nitroglycerin IV
* Lasix IV
* Morphine IV

**Acute aortic dissection**

* Nitroprusside IV plus beta-blockers IV
* Alternative - Trimethaphan IV plus beta-blockers IV

**Cerebral vascular accident**

 Lowering BP is indicated in cardiac or renal compromise, DBP >130 mm Hg, hypertensive encephalopathy, or subarachnoid hemorrhage (may require BP control to prevent rebleeding even without other evidence of end-organ damage).

* Nitroprusside IV
* Labetalol IV
* Nimodipine IV
* Monoamine oxidase (MAO)-tyramine interactions with acute hypertension - Phentolamine IV

Reduce BP quickly (over minutes to hours) in the following settings:

**Pheochromocytoma**

* Phentolamine IV
* Nitroprusside IV
* Labetalol IV

**Hypertensive encephalopathy**

* Nitroprusside IV
* Trimethaphan IV
* Beta-blockers IV

**Eclampsia**

* Hydralazine IV
* Labetalol IV
* Magnesium IV

Lowering of BP acutely in the ED in clinical situations other than those listed here is controversial and generally should be avoided.

Once the diagnosis of a true hypertensive emergency is established and end-organ damage confirmed, the BP should be lowered by up to 25% of the mean arterial pressure (MAP) over minutes to hours. Treat a diastolic reading of 120 mm Hg or greater with an IV antihypertensive medication to prevent cerebral hemorrhage.

**Further Inpatient Care**

Patients with a true hypertensive emergency require the careful titration of IV medications for good control and a smooth reduction of their BP.

Close monitoring is required; therefore, an intensive care unit is the most suitable place for admission.

Other problems or comorbid conditions need to be addressed appropriately (ie, surgery for aortic dissection).

**Further Outpatient Care**

Hypertension is a chronic problem. The most important factor in a patient's overall risks of morbidity and mortality is appropriate long-term care.

If a patient presents with a high BP but ED evaluation reveals no evidence of end-organ damage, the patient does not need immediate treatment in the ED. Patient does require proper follow-up.

The Joint National Committee on High Blood Pressure has published a series of recommendations for appropriate follow-up, assuming no end-organ damage.

For a systolic BP 140-159 mm Hg/diastolic 90-99 mm Hg, confirm BP within 2 months.

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For systolic BP greater than 210 mm Hg/diastolic greater than 120 mm Hg, evaluate/refer immediately.

**Prevention**

Good long-term control of hypertension is the best method for prevention of acute hypertensive emergencies.

Patient education and close follow-up in patients who have had a hypertensive crisis are essential to prevent recurrent hypertensive emergencies.

Proper use of antihypertensive medications by primary care physicians is the major tool in avoiding development of hypertensive emergencies.

**Complications**

* Congestive heart failure
* Myocardial infarction
* Renal failure
* Retinopathy
* Cerebrovascular accident
* Abrupt lowering of the BP may result in inadequate cerebral or cardiac blood flow, leading to stroke or myocardial ischemia.

**Prognosis**

The 1-year mortality rate is higher than 90% for patients with untreated hypertensive emergencies.

Median survival duration is 144 months for all patients presenting to the ED with a hypertensive emergency.

Five-year survival rate among all patients presenting with hypertensive crisis is 74%.

Patient Education:

Patients need continuing education about antihypertensive medications and complications arising from inadequate BP control.

Dangers of uncontrolled hypertension must be stressed, including associated serious morbidity and death.

Education and maintenance of BP control are important to help prevent further complications.

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