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Contemporary view on hypertension: From diagnostics to treatment

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Abstract

Arterial hypertension is a persistent increase in resting systolic blood pressure and/or diastolic blood pressure than recommended thresholds. An increase in blood pressure without a known cause is the most common. Hypertension with a known cause is usually associated with obstructive sleep apnea, chronic kidney disease, primary aldosteronism, diabetes, or obesity. Usually, symptoms appear only with severe or prolonged course. Diagnosis is based on sphygmomanometry. Diagnosis allows to determine the cause, evaluate the lesion, and also identify other cardiovascular risk factors. Treatment includes lifestyle changes and medications, including diuretics, beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, and calcium channel blockers. In this article latest study are stated in terms of early identification, diagnosis and timely management of this disease. Knowing risk factors and predictors allows health care workers early identification and management of high blood pressure as well as reduce the burden associated with its complications.

Keywords: Hypertension; Screening; Risk factors; Diagnosis

1 Introduction

Approximately 75 million people in the United States suffer from hypertension. About 81% of these people are aware that they have hypertension, only 75% receive treatment, and only 51% adequately control blood pressure [1]. Among adults, hypertension is more common in African Americans (41%) than in Caucasians (28%) or Mexican Americans (28%), and African Americans have higher morbidity and mortality [2]. Blood pressure increases with age. Approximately two-thirds of people > 65 years of age have hypertension, and people with normal blood pressure at age 55 have a 90% risk of developing hypertension [3]. Because high blood pressure becomes so common with age, it may seem harmless, but high blood pressure increases the risk of morbidity and mortality [4]. Hypertension before pregnancy, or developing during pregnancy, has its own characteristics. Blood pressure in adults is classified as normal, high blood pressure, stage 1 (mild), or stage 2 or 3 hypertension. Normal blood pressure in infants and adolescents is much lower.

1.1 Etiology of hypertension

Arterial hypertension can be: Primary (from 85% of cases) and Secondary.

Primary arterial hypertension. Hemodynamic and physiological components (eg, plasma volume, activity of the renin-angiotensin system) are variable, indicating that primary hypertension is unlikely to have a single cause [5]. Even if one of the factors is initially responsible for the development of arterial hypertension, multiple factors are probably involved in maintaining high blood pressure (mosaic theory). In afferent systemic arterioles, dysfunction of ion pumps in the

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sarcolemma of smooth muscle cell membranes can lead to a chronic increase in vascular tone. Heredity is a predisposing factor, however the exact mechanism remains unclear [6]. Environmental factors (eg, dietary sodium, stress) seem to affect only genetically susceptible individuals at a younger age; however, in patients over 65 years of age, high sodium intake is likely to precipitate hypertension [7].

1.2 Secondary arterial hypertension

The usual reasons are: Diabetes; Obesity; Obstructive sleep apnea syndrome; Primary aldosteronism; Parenchymal kidney disease (eg, chronic glomerulonephritis or pyelonephritis, polycystic kidney disease, connective tissue disease, obstructive uropathy); Renovascular disease; Other rarer causes include pheochromocytoma, Cushing's syndrome, congenital adrenal hyperplasia, hyperthyroidism, hypothyroidism (myxedema), primary hyperparathyroidism, acromegaly, aortic coarctation, and mineralocorticoid excess syndromes other than primary aldosteronism. Excessive alcohol consumption and use of oral contraceptives are common causes of treatable hypertension. Taking sympathomimetics, non-steroidal anti-inflammatory drugs (NSAIDs), glucocorticosteroids, cocaine, or licorice tends to worsen blood pressure control [8,9,10]. Arterial hypertension is considered resistant when blood pressure remains above the target values despite the use of 3 different antihypertensive drugs [11]. Patients with resistant hypertension have a higher rate of morbidity and mortality from cardiovascular disease.

1.3 Pathophysiology of arterial hypertension

Since blood pressure corresponds to cardiac output (CO) × total peripheral vascular resistance (TPVR), pathogenetic mechanisms must include: Increased CB; Increased periphery resistance; both. In most patients, CV is normal or slightly elevated and TPVR is elevated. This model is typical for primary arterial hypertension, as well as for hypertension due to primary hyperaldosteronism, pheochromocytoma, renovascular disease and parenchymal kidney disease [12].

In other patients, CO increases (possibly due to venoconstriction in large veins), and TPVR does not match the increase in CO and is normal. In the future, OPSS increases and CO normalizes, probably due to self-regulation [13]. Some diseases that increase CO (thyrotoxicosis, arteriovenous fistula, aortic insufficiency), especially when stroke volume is increased, can cause isolated systolic hypertension [14]. Some elderly patients have isolated systolic hypertension with normal or low CO, probably due to stiffness of the aorta and its large branches. Patients with high, persistent diastolic pressure often have low CO [15]. Plasma volume tends to decrease as blood pressure increases; rarely, plasma volume remains normal or increases. Plasma volume, as a rule, is increased in primary aldosteronism or parenchymal kidney disease, and may be sufficiently reduced in arterial hypertension due to pheochromocytoma [16]. Renal blood flow gradually decreases as diastolic blood pressure increases, and arteriolosclerosis begins. The glomerular filtration rate (GFR) remains normal until abnormalities appear during disease progression; as a result, the filtration fraction increases [17]. Coronary, cerebral and muscle blood flow is maintained until the development of severe atherosclerosis in these vascular pools.

2 Pathological transfer of sodium

In many causes of hypertension, sodium transport through the cell wall is impaired due to damage or inhibition of the potassium-sodium pump (Na +, K + ATPase) or increased cell permeability by sodium ions. The result is an increase in intracellular sodium, which makes the cells more sensitive to sympathetic stimulation. Calcium follows sodium, so the accumulation of intracellular calcium can lead to hypersensitivity [18]. Because Na+,K+-ATPase can move norepinephrine back into sympathetic neurons (thus inactivating this neurotransmitter), inhibition of this mechanism may also enhance the effect of norepinephrine, increasing blood pressure [19]. Defects in sodium transport can be observed in children with normal blood pressure whose parents suffer from hypertension.

2.1 Sympathetic nervous system

Sympathetic stimulation increases blood pressure; it generally occurs more frequently in patients with high blood pressure and hypertension than in patients with normal blood pressure [20]. It is not known whether this hyperreactivity is present in the sympathetic nervous system or in the myocardium and vascular smooth muscle [21]. A high resting heart rate, which may result from increased activity of the sympathetic nervous system, is a well-known predictor of hypertension. In some hypertensive patients, circulating plasma catecholamine levels at rest are higher than normal.

2.2 Renin-angiotensin-aldosterone system

The renin-angiotensin-aldosterone system helps regulate blood volume and therefore blood pressure. Renin, an enzyme produced in the juxtaglomerular apparatus, catalyzes the conversion of angiotensinogen to angiotensin I. Angiotensin-

converting enzyme (ACE) breaks down this inactive product, mainly in the lungs, but also in the kidneys and brain, to angiotensin II, a highly active vasoconstrictor that also stimulates autonomic functions. Centers in the brain, increasing discharges in the sympathetic nerve and stimulating the release of aldosterone and vasopressin. Aldosterone and vasopressin cause sodium and water retention, raising blood pressure [22]. Aldosterone also enhances potassium excretion; low plasma potassium (< 3.5 mEq/L [< 3.5 mmol/L]) increases vasoconstriction through the closure of potassium channels. Angiotensin III, present in the bloodstream, stimulates the release of aldosterone as actively as angiotensin II, but has much less vasoconstrictive activity. Because the enzyme chymase also converts angiotensin I to angiotensin II, ACE-inhibiting drugs do not completely suppress angiotensin II production.

Renin secretion is controlled by at least 4 mechanisms, which are not mutually exclusive: The renal vascular receptor responds to changes in pressure in the wall of the afferent arteriole; The macula receptor detects changes in delivery rate or sodium chloride concentration in the distal tubules; Circulating angiotensin influences renin secretion through a negative feedback mechanism [23]. The sympathetic nervous system stimulates beta-mediated renin secretion (via the renal nerve); Angiotensin is generally responsible for the development of renovascular hypertension, at least initially, but the role of the renin-angiotensin-aldosterone system in primary hypertension has not been established [24]. However, African Americans and elderly hypertensive patients tend to have low renin levels. Elderly patients also tend to have low angiotensin II levels. Arterial hypertension due to chronic parenchymal kidney disease (renoprival hypertension) is the result of a combination of renin-dependent and volume-dependent mechanisms. In most cases, an increase in renin activity is not detected in the peripheral blood [25]. Arterial hypertension, as a rule, has a moderate stage and is sensitive to the balance of sodium and water.

3 Lack of vasodilators

A lack of vasodilators (eg, bradykinin, nitric oxide) rather than an excess of vasoconstrictors (eg, angiotensin, norepinephrine) may lead to hypertension. A decrease in nitric oxide levels due to arterial stiffness is associated with salt-sensitive hypertension, an excessive increase in systolic blood pressure by > 10-20 mmHg. After a high sodium load [26]. If the kidneys do not produce enough vasodilators (due to renal parenchymal disease or bilateral nephrectomy), blood pressure may rise. Vasodilators and vasoconstrictors (mainly endothelin) are also produced by endothelial cells. Thus, endothelial dysfunction greatly affects blood pressure levels.

3.1 Pathology and complications

At an early stage of arterial hypertension, no pathological changes are observed. Severe or prolonged hypertension damages target organs (primarily the cardiovascular system, brain, kidneys), increasing the risk of developing: Ischemic heart disease (CHD) and myocardial infarction (MI); heart failure; Stroke (especially hemorrhagic); kidney failure; Death.

The mechanism includes the development of generalized arteriolosclerosis and the acceleration of atherogenesis [27]. Arteriolosclerosis is characterized by medial hypertrophy, hyperplasia and hyalinization, which is especially pronounced in small arterioles, in particular in the eyes and kidneys. In the kidneys, changes narrow the lumen of the arterioles, increasing the peripheral vascular resistance, thus leading to even more pronounced arterial hypertension [28]. In addition, after initial arterial narrowing, any slight additional contraction of already hypertrophied smooth muscle reduces the lumen to a greater extent than with a normal arterial diameter [29]. These factors may explain why the duration of hypertension is inversely related to the success of specific treatment (eg, renovascular surgery) of secondary causes in restoring normal blood pressure.

Due to increased afterload, the left ventricle gradually hypertrophies, resulting in diastolic dysfunction. The left ventricle eventually dilates, resulting in dilated cardiomyopathy and heart failure with systolic dysfunction and is often exacerbated by atherosclerotic heart disease [30]. Dissection of the thoracic aorta is usually a consequence of arterial hypertension; almost all patients with abdominal aortic aneurysm have arterial hypertension.

3.2 Symptoms and signs of hypertension

Arterial hypertension is usually asymptomatic until complications develop in target organs. Uncomplicated hypertension can cause dizziness, facial flushing, headache, fatigue, nosebleeds, and irritability [30]. Severe hypertension (hypertensive crisis) can cause serious cardiovascular, neurologic, renal, and retinal symptoms (eg, symptomatic coronary atherosclerosis, heart failure, hypertensive encephalopathy, renal failure). The presence of a 4th heart sound is one of the earliest signs of hypertensive cardiomyopathy. Retinal changes may include narrowing of arterioles, hemorrhage, exudation, and papilledema in patients with encephalopathy (hypertensive retinopathy) [31].

Changes are classified (in accordance with the Keith-Wagener-Barker classification) into 4 groups (in order of worsening prognosis):

- Stage 1: Arteriole constriction only
- Score 2: Narrowing and sclerosis of arterioles
- Stage 3: Bleeding and exudates in addition to vascular changes
- Stage 4: papilledema

3.3 Diagnosis of arterial hypertension

Multiple blood pressure measurements for confirmation; Urinalysis and urinary albumin/creatinine ratio; if there are deviations from the norm, then consider the need to perform an ultrasound of the kidneys; Blood tests: fasting lipids, creatinine, potassium; Ultrasound of the kidneys with elevated creatinine [32]. Aldosteronism should be assessed if potassium levels are low. ECG: in the presence of left ventricular hypertrophy, echocardiography is necessary. Sometimes the determination of thyroid-stimulating hormone. Assess for pheochromocytoma or sleep disturbances if blood pressure is labile and rises suddenly or severe hypertension occurs

Arterial hypertension is diagnosed and classified using sphygmomanometry. The history, physical examination, and other tests help determine the etiology and determine if target organs are affected.

4 Blood pressure measurement

For formal diagnosis, blood pressure should be measured on average 2 or 3 times, at different times when the patient: Sits in a chair (not on an examination couch) for > 5 minutes, feet on the floor, back supported; Limbs are supported at heart level, clothing does not cover the cuff area; No exercise, caffeine intake, or smoking for at least 30 minutes; At the first visit, measure blood pressure in both arms, and on subsequent visits, measure the arm with the higher reading.

The appropriately sized cuff of the blood pressure monitor is worn over the upper arm. An appropriately sized cuff covers two-thirds of the biceps; the cuff chamber is long enough to wrap around >80% of the arm and the cuff chamber is at least 40% of the arm circumference wide. Thus, in obese patients, a larger cuff is required [33]. The practitioner inflates the cuff above the expected systolic pressure and releases the air gradually while listening to the brachial artery pulse. The pressure at which the first heart sound is heard during pressure reduction is the systolic blood pressure. The complete disappearance of tones indicates diastolic blood pressure. The same principles should be followed for measuring blood pressure in the forearm (radial artery) and thigh (popliteal artery). Mechanical instruments need to be calibrated periodically; automatic devices often show inaccurate data. Blood pressure is measured on both arms, because if the difference in blood pressure is > 15 mm Hg. on one arm compared to the other, a study of the vascular system of the upper body is necessary. To rule out coarctation of the aorta, especially in patients with a weakened pulse or a delayed pulse wave on the femoral artery, blood pressure is measured at the thigh (using a much larger cuff); with coarctation, blood pressure in the lower extremities is much lower [34]. If blood pressure is in the range of stage 1 hypertension or is markedly labile, it is desirable to measure blood pressure more frequently. Rarely, blood pressure readings can be high before hypertension becomes persistent; this phenomenon probably explains "white coat hypertension", in which blood pressure is elevated when measured in the doctor's office, but normal when measured at home or with ambulatory blood pressure monitoring [35]. However, a sudden rise in blood pressure alternating with normal readings is unusual and possibly indicates pheochromocytoma, a sleep disorder such as sleep apnea, or unrecognized drug use.

History. The history includes information about the duration of hypertension and previous blood pressure levels; any indication of a history or symptoms of coronary artery disease, heart failure, sleep apnea, or loud snoring; history or symptoms of another significant comorbid disorder (eg, stroke, renal dysfunction, peripheral arterial disease, dyslipidemia, diabetes, gout); as well as a family history of any of these disorders [36]. Lifestyle history includes information about physical activity, tobacco, alcohol, and stimulant drug use (prescribed and prohibited). In dietary preferences, attention is paid to the intake of salt and stimulants (eg, tea, coffee, caffeinated sodas, energy drinks).

Objective examination. An objective examination includes measuring height, weight, waist circumference; examination of the fundus to exclude retinopathy; auscultation to exclude noise in the neck and abdominal cavity; complete cardiological, neurological and respiratory examinations 37]. Palpation of the abdomen is performed to exclude an increase in the kidneys and to identify formations in the abdominal cavity. The pulsation of peripheral arteries is estimated; decreased or delayed femoral pulsation suggests aortic coarctation, especially in patients < 30. A unilateral murmur over the renal artery may be heard in lean patients with renovascular hypertension.

Examination. The more severe the hypertension and the younger the patient, the more extensive the examination should be [38]. As a rule, when arterial hypertension is newly diagnosed, then a routine examination is carried out for: Detection of target organ damage; Definitions of cardiovascular risk factors; Urinalysis and the ratio of urine albumin to creatinine; Blood tests (creatinine, potassium, sodium, fasting glucose, lipid profile, and often thyroid-stimulating hormone).

ECG. Ambulatory blood pressure monitoring, radionuclide imaging of the kidneys, chest x-ray, screening tests to exclude pheochromocytoma, and analysis of the renin-sodium profile are usually not required as part of a routine study. However, if "white coat hypertension" is suspected, home or outpatient blood pressure monitoring is indicated. In addition, ambulatory blood pressure monitoring may also be indicated if "occult hypertension" (a condition in which blood pressure measured at home is higher than office readings) is suspected, usually in patients with sequelae. Hypertension without signs of hypertension as measured in the doctor's office. Evaluation of peripheral plasma renin activity does not help in diagnosis or choice of drug [39]. Other tests may be needed depending on the results of the first tests and examination. If a urinalysis shows albuminuria (proteinuria), cylindruria, microhematuria, or if serum creatinine is elevated (≥ 1.4 mg/dL [124 micromol/L] in men and ≥ 1.2 mg/dL [106 micromol/L] in women), an ultrasound of the kidneys to assess the size of the kidneys can provide useful information. Patients with hypokalemia not associated with the use of diuretics are examined for primary aldosteronism and increased salt intake [40]. On an ECG, a wide P wave indicates atrial hypertrophy and, although nonspecific, may be one of the early signs of hypertension. Left ventricular hypertrophy, identified by a sustained apex beat and an increase in QRS voltage, with or without evidence of ischemia, may occur later. If any of these findings are present, echocardiography is often done. In patients with lipid abnormalities or symptoms of coronary heart disease, it may be informative to perform studies to identify other cardiovascular risk factors (eg, C-reactive protein). If coarctation of the aorta is suspected, chest x-ray, echocardiography, CT, or MRI help confirm the diagnosis. Patients with labile, markedly elevated blood pressure and symptoms such as headache, palpitations, tachycardia, increased sweating, tremor, and pallor should be screened for pheochromocytoma (eg, measurement of plasma free metanephrines) [41]. In these patients, and in patients with a history suggestive of sleep apnea, sleep testing should be seriously considered. Patients with symptoms suggestive of Cushing's syndrome, connective tissue disease, eclampsia, acute porphyria, thyrotoxicosis, myxedema, acromegaly, or central nervous system (CNS) disorders are also evaluated.

4.1 Prognosis for arterial hypertension

The higher the blood pressure values and the more severe the retinal changes, as well as if there are other signs of target organ damage, the worse the prognosis [42]. Systolic blood pressure is a predictor of fatal and non-fatal cardiovascular events to a greater extent than diastolic blood pressure [43]. Without treatment, 1-year survival is < 10% in patients with retinal sclerosis, cottony exudates, narrowing of the arterioles, and hemorrhage (grade 3 retinopathy) and < 5% in patients with the same changes and papilledema (retinopathy 4). Degrees). Ischemic heart disease is the most common cause of death among treated patients. Ischemic or hemorrhagic stroke is the most common consequence of the lack of adequate treatment of arterial hypertension [44]. However, effective control of hypertension prevents most complications and prolongs life.

Primary hypertension cannot be cured, but some causes of secondary hypertension can be treated. In all cases, blood pressure control can significantly limit adverse effects. Despite the theoretical effectiveness of the treatment, blood pressure is reduced to the desired level in only a third of patients with hypertension in the United States.

Treatment goals for the general population, including patients with kidney disease and diabetes: Blood pressure < 130/80 mmHg, regardless of age up to 80 years. Even the elderly and debilitated elderly can tolerate such low diastolic blood pressure as low as 60-65 mmHg without an increase in the frequency of cardiovascular events. Ideally, patients or family members who measure blood pressure at home should be trained in this under supervision, and the blood pressure monitor should be regularly calibrated [45]. Treatment of hypertension during pregnancy requires special attention because some antihypertensive drugs can harm the fetus.

Lifestyle change. Lifestyle changes are recommended for all patients with high blood pressure or any hypertension (see also Table 15. Nonpharmacological Interventions in 2017 Hypertension Guidelines). The best evidence-based non-pharmacological interventions for the prevention and treatment of hypertension include the following: Increased physical activity through a structured exercise program; Weight loss if overweight or obese; A healthy diet rich in fruits, vegetables, whole grains and reduced-fat dairy products, reduced in total fat and saturated fat; Reduction of dietary sodium intake to < 1500 mg/day (< 3.75 g sodium chloride) is optimal, but at least reduction to 1000 mg/day; Increased dietary potassium intake, unless contraindicated due to chronic kidney disease or use of drugs that reduce potassium excretion; Moderate drinking in those who drink up to ≤ 2 drinks daily for men and ≤ 1 drink daily for women (one

drink is about 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of distilled spirits) [46]. Smoking cessation. Diet changes can also help control diabetes, obesity, and dyslipidemia. Patients with uncomplicated hypertension do not need to limit their usual activities as long as blood pressure is under control.

4.2 Medications

The decision to use medication is based on blood pressure levels and the presence of atherosclerotic cardiovascular disease (ASCVD) or its risk factors [47]. The presence of diabetes or kidney disease is not considered separately as these diseases are part of the ASCVD risk assessment [48]. An important part of treatment is regular follow-up visits. If patients are not achieving target blood pressure, clinicians should strive to optimize adherence before changing or adding drugs.

4.3 Initial approach to treating high blood pressure

The choice of drugs is based on several factors. For non-African American patients, including those with diabetes mellitus, initial treatment may include either an ACE inhibitor, an angiotensin II receptor blocker, a calcium channel blocker, or a thiazide-type diuretic (chlorthalidone or indapamide). For African Americans, including those with diabetes, calcium channel blockers or thiazide-like diuretics are recommended initially, unless patients also had stage 3 or later chronic kidney disease. For black patients with stage 3 chronic kidney disease, an ACE inhibitor or an angiotensin II receptor blocker may be appropriate [49,50].

If 2 drugs are initially prescribed, a single tablet combination of either an ACE inhibitor or an angiotensin II receptor blocker with either a diuretic or a calcium channel blocker. Signs of a hypertensive crisis require immediate lowering of blood pressure with the use of parenteral antihypertensive drugs [51]. Some antihypertensive drugs are contraindicated in certain conditions (eg, beta-blockers in asthma) or indicated in particular in patients with hypertension in certain conditions (eg, calcium channel blockers in angina pectoris, ACE inhibitors, or angiotensin II receptor blockers in diabetes mellitus with proteinuria—see tables Initial Antihypertensive Drug Class Selection and Antihypertensive Therapy in High-Risk Patients).

If target blood pressure is not achieved within 1 month, assess adherence and reinforce the importance of follow-up treatment. If patients are compliant, the initial dose of the drug may be increased or a second drug added (selected from the drugs recommended for initial treatment). It should be remembered that ACE inhibitors and an angiotensin II receptor blocker should not be used together [52]. Often, therapeutic doses are gradually reduced. If the desired blood pressure level cannot be achieved with 2 drugs, then a third drug from the starting group is added. If the use of a third drug is not acceptable (eg, African Americans) or the drug is not well tolerated by the patient, then a drug from another class (eg, beta-blockers, aldosterone antagonists) can be prescribed. Patients with difficult-to-control blood pressure may benefit from consulting a hypertension specialist.

4.4 Initial choice of antihypertensive drug class

If initial systolic blood pressure is > 160 mm Hg, then 2 drugs should be used regardless of lifestyle. An adequate combination and dose are selected; many drug combinations are available as a single tablet, which improves patient compliance and is preferred. For the treatment of persistent hypertension (blood pressure remains above the target despite the use of 3 different antihypertensive drugs), 4 or more drugs are usually required. Multiple visits and changes in drug therapy are often required to achieve adequate control. Reluctance to titrate and add drugs to control BP must be overcome [53]. Lack of patient compliance, especially when lifelong therapy is required, may compromise adequate blood pressure control. Knowledge, empathy and support are integral parts of success.

4.5 Devices and physical interventions

Percutaneous catheter radiofrequency ablation of sympathetic nerves in the renal artery is approved in Europe and Australia for the treatment of resistant hypertension [55, 56, 57, 58]. Although initial studies seemed promising, a new large double-blind study has recently been conducted. This study included sham ablation procedures for the first time in a control group and failed to show the benefits of radiofrequency ablation. Therefore, sympathetic ablation should still be considered as an experimental treatment and is only performed in European or Australian centers with extensive experience [59, 60]. The second physical intervention involves stimulating the carotid baroreceptors with a device implanted around the carotid sinus. A battery is attached to the device, as is a pacemaker, which is used to stimulate baroreceptors in a dose-dependent manner and to lower blood pressure. A long-term analysis of a statistically valid study showed that baroreflex activation therapy remained effective in permanently lowering office BP in patients with resistant hypertension without major safety concerns. The 2017 American College of Cardiology/American Heart

Association guidelines concluded that studies have not provided sufficient evidence to recommend the use of these devices in the treatment of resistant hypertension.

5 Conclusion

Only about three-quarters of patients with hypertension in the United States are treated, and only half of them have adequate blood pressure (BP) control. Most cases of arterial hypertension have a primary form; only 5 to 15% of cases occur in association with other diseases (eg, renal parenchymal disease or vasorenal hypertension, obstructive sleep apnea, pheochromocytoma, Cushing's syndrome, congenital adrenal hyperplasia, hyperthyroidism). Severe or long-term hypertension damages the cardiovascular system, brain, and kidneys, increasing the risk of myocardial infarction, stroke, and chronic kidney disease. Arterial hypertension is usually asymptomatic until complications develop in target organs. When hypertension is first diagnosed, a urinalysis, urine albumin/creatinine ratio, blood tests (creatinine, potassium, sodium, fasting plasma glucose, lipid profile, and often thyroid-stimulating hormone) and ECG are done. Reduce blood pressure to < 130/80 mmHg. in all patients < 80 years of age, including those with impaired renal function or diabetes mellitus. Treatment includes lifestyle changes, especially a low-sodium, high-potassium diet, control of causes of secondary hypertension, and medications.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest.

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