

Hypertension Clinical Presentation

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History

The 2013 joint European Society of Hypertension (ESH) and the European Society of Cardiology (ESC) guidelines recommend that ambulatory blood-pressure monitoring (ABPM) be incorporated into the assessment of cardiovascular risk factors and hypertension.^[7, 8]

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

- Extent of end-organ damage (eg, heart, brain, kidneys, eyes)
- Assessment of patients' cardiovascular risk status
- Exclusion of secondary causes of hypertension

Patients may have undiagnosed hypertension for years without having had their BP checked. Therefore, a careful history of end-organ damage should be obtained. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) identifies the following as targets of end-organ damage^[3]:

- Heart: left ventricular hypertrophy, angina/previous myocardial infarction, previous coronary revascularization, and heart failure
- Brain: stroke or transient ischemic attack, dementia
- Chronic kidney disease
- Peripheral arterial disease
- Retinopathy

The JNC 7 identifies the following as major cardiovascular risk factors^[3]:

- Hypertension: component of **metabolic syndrome**
- Tobacco use, particularly cigarettes, including chewing tobacco
- Elevated LDL cholesterol (or total cholesterol ≥ 240 mg/dL) or low HDL cholesterol: component of metabolic syndrome
- Diabetes mellitus: component of metabolic syndrome
- Obesity (BMI ≥ 30 kg/m²): component of metabolic syndrome
- Age greater than 55 years for men or greater than 65 years for women: increased risk begins at the respective ages; the Adult Treatment Panel III used earlier age cut points to suggest the need for earlier action
- Estimated glomerular filtration rate less than 60 mL/min
- Microalbuminuria
- Family history of premature cardiovascular disease (men < 55 years; women < 65 years)
- Lack of exercise

Obtain a history of the patient's use of over-the-counter medications; herbal medicines such as herbal tea containing licorice (the issue is products containing licorice root; large amount of licorice in the US is licorice candy, but black licorice and over-the-counter licorice root supplements are increasingly available); **ephedrine/ephedra**; current and previous unsuccessful antihypertensive medication trials; oral contraceptives; ethanol; and illicit drugs such as cocaine. The patient's lifestyle factors should also be included, such as changes in weight, dietary intake of sodium and cholesterol, exercise level, and psychosocial stressors.^[9]

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 obstructive sleep apnea may be noted. A history of weakness suggests hyperaldosteronism. Kidney stones raise the possibility of hyperparathyroidism.

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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.^[3, 5] 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.

The patient should rest quietly for at least 5 minutes before the measurement. Blood pressure should be measured in both the supine and sitting positions, auscultating with the bell of the stethoscope. 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. Although somewhat controversial, the common practice is to document phase V (a disappearance of all sounds) of Korotkoff sounds as the diastolic pressure.

Ambulatory or home blood pressure monitoring provides a more accurate prediction of cardiovascular risk than do office blood pressure readings.^[46] "Non-dipping" is the loss of the usual physiologic nocturnal drop in blood pressure and is associated with an increased cardiovascular risk. 

A study by Wong and Mitchell indicated that independent of other risk factors, there is a link between the presence of certain signs of hypertensive retinopathy (eg, retinal hemorrhages, microaneurysms, cotton-wool spots) and an increased cardiovascular risk (eg, stroke, stroke mortality).^[47] Therefore, a fundoscopic evaluation of the eyes should be performed to detect any evidence of early or late, chronic or acute hypertensive retinopathy, including arteriovenous nicking or changes in the vessel wall (eg, copper wiring, silver wiring, SOT, hard exudates, flame-shaped hemorrhages, papilledema). Indeed, ocular changes can be the initial finding in an asymptomatic patient necessitating a primary care referral; acute and chronic changes may manifest in the eyes. Alternatively, a symptomatic patient may be referred to the ophthalmologist for visual changes due to hypertensive changes.

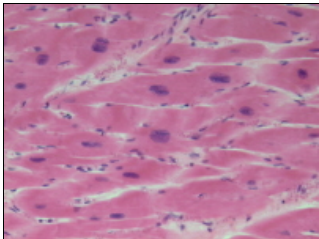
Palpation of all peripheral pulses should be performed. Absent, weak, or delayed femoral pulses suggests coarctation of the aorta or severe peripheral vascular disease. In addition, examine the neck for carotid bruits, distended veins, or enlarged thyroid gland.^[3, 5] Listen for renal artery bruit over the upper abdomen; the presence of a 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.

Hypertension and Cerebrovascular Disease

Blood pressure is a powerful determinant of risk for **ischemic stroke** and intracranial hemorrhage; in fact, 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 are associated with a significant incidence of strokes. The American Heart Association notes that individuals whose blood pressure level is lower than 120/80 mm Hg have about 50% the lifetime stroke risk of that of hypertensive individuals.

The main pathologic findings are in the heart, which shows an increase in mass caused principally by left ventricular hypertrophy. Histologically, the individual myocytes are enlarged and show nucleomegaly ("box car" nuclei) (see the image below). Hearts that are enlarged secondary to hypertension have an increased incidence of arrhythmia and death. Other cerebrovascular manifestations of complicated hypertension include hypertensive hemorrhage, hypertensive encephalopathy, lacunar-type infarctions, and dementia.



Hypertrophied cardiac myocytes with enlarged "box car" nuclei.

Hypertensive encephalopathy is one of the clinical manifestations of cerebral edema and microhemorrhages seen with dysfunction of cerebral autoregulation and is characterized by hypertension, altered mentation, and papilledema.

Hypertensive Emergencies

The history and physical examination determine the nature, severity, and management of the hypertensive event. The history should focus on the presence of end-organ dysfunction, the circumstances surrounding the hypertension, and any identifiable etiology. The physical examination should assess whether end-organ dysfunction is present (eg, neurologic, cardiovascular). BP should be measured in both the supine position and the standing position (assess volume depletion). BP should also be measured in both arms (a significant difference may suggest aortic dissection).

The most common clinical presentations of hypertensive emergencies are cerebral infarction (24.5%), **pulmonary edema** (22.5%), hypertensive encephalopathy (16.3%), and congestive heart failure (12%). Other clinical presentations associated with hypertensive emergencies include intracranial hemorrhage, **aortic dissection**, and **eclampsia**,^[48] as well as acute myocardial infarction. Hypertension is also one of several conditions that have been increasingly recognized as having an association with posterior reversible encephalopathy syndrome (PRES), a condition characterized by headache, altered mental status, visual disturbances, and seizures.^[49]

Hypertensive Heart Disease

Uncontrolled and prolonged BP elevation can lead to a variety of changes in the myocardial structure, coronary vasculature, and conduction system of the heart. These changes in turn can lead to the development of left ventricular hypertrophy (LVH), coronary artery disease, various conduction system diseases, and systolic and diastolic dysfunction of the myocardium, which manifest clinically as [angina](#) or [myocardial infarction](#), cardiac arrhythmias (especially atrial fibrillation), and [congestive heart failure \(CHF\)](#). Thus, hypertensive heart disease is a term applied generally to heart diseases—such as LVH, [coronary artery disease](#), cardiac arrhythmias, and CHF—that are caused by direct or indirect effects of elevated BP.

Although these diseases generally develop in response to chronically elevated BP, marked and acute elevation of BP can also lead to accentuation of an underlying predisposition to any of the symptoms traditionally associated with chronic hypertension.

In a study by Tymchak et al, patients presenting with acute heart failure as a manifestation of hypertensive emergency were more likely to be black and have a history of heart failure; they were also more likely to have higher B-type natriuretic peptide (BNP) and creatinine levels and lower left ventricular ejection fraction. Note that BNP is inversely proportional to the degree of a patient's obesity.^[50]



Hypertension in Pediatric Patients

Advances in the ability to identify, evaluate, and care for infants with hypertension, coupled with advances in the practice of neonatology in general, have led to an increased awareness of hypertension in modern neonatal ICUs (NICUs) since its first description in the 1970s.

The true incidence of hypertension in the pediatric population is not known, although various health data showed a decreasing trend between 1963 and 1988, followed by an upward trend. Hypertension is now commonly discovered in children, and the long-term health risks to these children may be substantial.

Systemic hypertension is less common in children than in adults, but the incidence of hypertension in children is approximately 1-5%. The presence of hypertension in younger children is usually indicative of an underlying disease process (secondary hypertension). In children, approximately 5-25% of cases of secondary hypertension are attributed to renovascular disease.

Hypertension in Pregnancy

Hypertension is the most common medical problem encountered during pregnancy, complicating 2-3% of pregnancies. Hypertensive disorders during pregnancy are classified into the 4 following categories, as recommended by the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy:

- Chronic hypertension
- Preeclampsia-eclampsia
- Preeclampsia superimposed on chronic hypertension
- Gestational hypertension (transient hypertension of pregnancy or chronic hypertension identified in the latter half of pregnancy); this terminology is preferred over the older but widely used term pregnancy-induced hypertension (PIH) because it is more precise.

A large, population-based study compared 26,651 pregnant women with hypertensive disorders to 213,397 pregnant women without hypertensive disorders to determine risk of end-stage renal disease. Results showed that the incidence of chronic kidney disease was almost 11-fold higher in the hypertensive group. This group also exhibited a 14-fold increased risk for end-stage renal disease. The risk was much greater for women with preeclampsia or eclampsia. This study highlights the importance of adequate follow-up of BP after pregnancy.^[51]

Primary Aldosteronism

Mineralocorticoid excess secondary to primary hyperaldosteronism is infrequently observed and is characterized by excessive production of aldosterone. Renal sodium retention, kaliuresis, hypokalemia, and hypochloremic metabolic alkalosis are the common manifestations. It should be considered in patients who have an exaggerated hypokalemic response to a thiazide diuretic or who have hypokalemia unprovoked by a diuretic. These patients develop increased intravascular volume, resulting in hypertension. The BP increase may vary from mild hypertension to marked elevation in primary hyperaldosteronism. Patients may have underlying adenoma or hyperplasia of the adrenal gland and rarely have an extra-adrenal source for aldosterone.

The incidence of primary hyperaldosteronism was 1.5% in one study.^[11] However, the true incidence of primary aldosteronism is still not clear, largely because of the lack of criterion-standard testing. In contrast, inappropriately high output of aldosterone for a given salt state of a patient (ie, not meeting criteria for a diagnosis of primary aldosteronism) is much more common, especially in patients with metabolic syndrome.^[52]

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