Office Evaluation of Hypertension

A Statement for Health Professionals by a Writing Group of the Council for High Blood Pressure Research, American Heart Association

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The approach to management of hypertension has changed radically in recent years. In the past, major emphasis was placed on detection of curable forms of hypertension and treatment of premalignant and malignant hypertension. Prospective randomized double-blind trials of treated and untreated patients have demonstrated conclusively that reduction of blood pressure with antihypertensive agents protects against major cardiovascular complications such as stroke, congestive heart failure, renal damage, dissecting aneurysm, progression of hypertension to a more severe state, and, in some studies, complications of coronary artery disease.

Present evidence indicates the higher diastolic blood pressure is, the greater the benefit of treatment. However, there is agreement that all patients with diastolic blood pressures averaging 90 mm Hg or more when measured three times on each of three or more office visits should be evaluated and receive nonpharmacological therapy. Pharmacological therapy is indicated for patients whose diastolic pressure remains greater than or equal to 95 mm Hg despite nonpharmacological therapy and for many whose diastolic pressure remains in the 90–94 mm Hg range during nonpharmacological therapy. The major emphasis therefore is to detect and effectively treat the large numbers of inadequately treated or untreated patients with persistent hypertension of this degree.

It is now recognized that systolic blood pressure may be even more important than diastolic blood pressure as an index of prognosis. The treatment goal should therefore include normalization of systolic blood pressure (<140 mm Hg) as well as diastolic blood pressure. There are no data from prospective trials that show treatment of isolated systolic hypertension (≥160 mm Hg with diastolic blood pressure <90 mm Hg) will lessen cardiovascular risk.

Even in the absence of diastolic hypertension, patients with persistent elevation of systolic blood pressure (≥160 mm Hg) deserve evaluation and nonpharmacological, if not pharmacological, treatment.

Objectives for evaluation of the hypertensive patient are listed in Table 1.

Evaluation of Hypertensive Adults

Evaluation of a hypertensive patient gives the physician an opportunity to discover curable causes for hypertension and its effect on target organs. Based on these observations, predictions about prognosis and appropriate treatment can be made. Evaluation also allows the physician an opportunity to discover other unrelated diseases.

The vast majority of patients with hypertension have primary or essential hypertension. Fewer than 5% of hypertensive patients have some identifiable curable cause such as hypothyroidism, acromegaly, Cushing’s syndrome, primary aldosteronism, pheochromocytoma, renovascular disease, or coarctation of the aorta. About 5% of patients with hypertension have an identifiable cause that is not curable—usually bilateral chronic renal parenchymal disease.

An appropriate history and physical examination are essential to evaluation. The history should describe duration and course of blood pressure elevation, associated cardiovascular problems, degree of blood pressure fluctuation, and responses to various treatments. The family history of hypertension, renal disease, cardiovascular problems, and diabetes mellitus should be noted. The physical examination should be complete, including measurement of height and weight and two or more blood pressure measurements with the patient standing and either supine or seated. Special attention should be given to the ophthalmoscopic examination, looking for arteriolar narrowing, arteriovenous compression, hemorrhages, exudates, and papilledema. Bruits in the neck and abdomen should be noted. The heart should be examined for rate, rhythm, size, precordial heave, clicks, murmurs, and third or fourth heart sounds. The abdominal examination
should focus on palpating masses, organs, and aneurysms. The extremities should be examined for peripheral arterial pulsations, bruits, and edema. A neurological examination for evidence of remote stroke should be administered.

A variety of laboratory examinations are recommended for routine evaluation of the hypertensive patient. Minimal laboratory examinations should include a hemoglobin measurement, hematocrit, a urinalysis with microscopic examination, an electrocardiogram (ECG), and an automated analysis of blood biochemical elements to include as a minimum serum potassium, uric acid, calcium, creatinine, glucose, and total and high-density lipoprotein (HDL) cholesterol. If either total or HDL cholesterol is abnormal, serum triglycerides should be measured and low-density lipoprotein (LDL) cholesterol calculated. A chest x-ray is ordinarily of little value in the initial evaluation unless the patient has known chest or cardiac disease or has smoked cigarettes for a long period.

Accurate, indirect blood pressure measurements are crucial in evaluation of the hypertensive patient. Persons who measure blood pressure should understand the need for 1) proper application of the appropriate size cuff and bladder, 2) a well-calibrated and properly functioning sphygmomanometer, and 3) knowledge of the proper technique for measuring blood pressure to avoid such problems as auscultatory gap, terminal digit preference, deflation of the cuff at a rate that is too rapid or too slow, and incorrect positioning of the arm. These matters are discussed in detail in the American Heart Association’s task force report, Recommendations for Human Blood Pressure Determination by Sphygmomanometers.* Blood pressure should be measured under standardized conditions in regard to exercise, excitement, food intake, and bladder distention, and with minimal apprehension. A pressor response is frequently observed when blood pressure is measured by a physician or nurse. This response usually becomes much less prominent with repeated measurements but occasionally persists in the office setting. Thus, it is difficult or impossible to estimate a patient’s true blood pressure with one or two casual blood pressure measurements. A major question concerns how many blood pressure measurements should be recorded for the best estimate of the patient’s representative blood pressure value. There is little argument that repeated measurements of blood pressure over several days are better than one or two measurements on one day. Nevertheless, it becomes impractical to have the patient return several times. As a minimum, the physician should take at least three measurements on three separate visits to estimate blood pressure severity for most patients with mild or moderate hypertension. When blood pressure is very high, time should not be lost in repeated measurements on separate days.

It is important to measure blood pressure with the patient upright because orthostatic hypotension may be present, especially in the elderly. Occasionally, orthostatic hypertension may also be observed. Blood pressure should be measured in both arms on one occasion to document equal blood pressure in each arm.

Home blood pressure measurements done properly with a mercury manometer, a calibrated aneroid sphygmomanometer, or an electronic device are valuable to the physician in determining significance of blood pressure elevation and necessity or adequacy of treatment. Home blood pressure measurements are vital in the care of the patient with accelerated or malignant hypertension.

There are many devices for home blood pressure measurement. The most reliable are aneroid sphygmomanometers with a conventional stethoscope or a stethoscope attached to a microphone beneath the blood pressure cuff. Several semiautomated electronic units use detection of Korotkoff’s sounds or oscillometry to estimate intra-arterial blood pressure. The accuracy of these systems varies when compared with a mercury manometer and becomes less so with heavy use. Thus, this equipment should be checked regularly against a mercury sphygmomanometer to ensure the reliability of the information obtained. Many of these devices are discussed in the May 1987 issue of Consumer Reports.

There is also great interest in 24-hour ambulatory blood pressure monitoring. Prospective studies from such measurements indicate that they are better predictors of cardiovascular morbidity and mortal-

<table>
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<th>Examinations</th>
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<td>To determine status of target organs and evaluate severity and prognosis.</td>
<td>History and physical, urinalysis, serum creatinine, ECG</td>
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<tr>
<td>To search for other risk factors for atherosclerosis.</td>
<td>History and physical, glucose, HDL, total cholesterol, triglycerides</td>
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<td>To determine the patient’s general state of health.</td>
<td>History and physical, hematocrit or CBC</td>
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<tr>
<td>To search for secondary causes, especially curable forms.</td>
<td>History and physical, urinalysis, serum K+, plasma glucose</td>
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<tr>
<td>To determine if hypertension is persistent.</td>
<td>Physical (repeated blood pressure measurements and ophthalmoscopic exam)</td>
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ECG, electrocardiogram; HDL, high-density lipoprotein; CBC, complete blood count.

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Risk Profile

In addition to average blood pressure, the patient’s age, sex, race, and presence or absence of target organ damage are major factors in determining the prognosis for cardiovascular mortality and morbidity in the hypertensive patient. For instance, a young black man with persistently elevated blood pressure in all measurements has a relatively poor chance of escaping complications into the sixth decade without treatment. On the other hand, a middle-aged white woman with borderline blood pressure elevation has relatively little likelihood of morbidity or mortality over the next few years.

Even these comments are inadequate to assess the patient’s overall cardiovascular risk. In addition to age, sex, race, and systolic and diastolic blood pressure, evidence from the 18-year follow-up in the Framingham study indicates that total serum cholesterol and HDL cholesterol, history of cigarette smoking, glucose intolerance, and the presence of left ventricular hypertrophy by ECG add to the physician’s ability to predict the likelihood of a major cardiovascular event. Booklets on gender- and age-specific risk of coronary heart disease and stroke are available from the American Heart Association.

With attention to the overall risk profile, the physician can identify modifiable risk factors that should be treated in addition to hypertension. Such risk profiles are powerful predictors of cardiovascular disease. For instance, the 6-year risk of cardiovascular disease occurring in a 45-year-old man with a systolic blood pressure of 165 mm Hg may vary from 31/1,000 to 372/1,000, depending on other risk factors. These variables and each patient’s specific needs should be considered.

Evaluation of Hypertensive Children

All children should have yearly blood pressure measurements. As in adults, proper cuff size is important for accurate measurement of blood pressure in children. Errors can be minimized by selecting the largest cuff that will fit the child’s arm.

As age increases, systolic blood pressure increases more than diastolic. However, body size also influences blood pressure and at a given age, tall or heavy children have higher blood pressures than small or slender children. Age- and sex-specific blood pressure distributions have been published by the National Heart, Lung, and Blood Institute’s (NHLBI) Second Task Force on Blood Pressure Control in Children.* Blood pressure greater than the 90th percentile for age may be normal if the child is tall or has increased lean body mass for age. (See appendix.) The 90th percentile for height and weight for each age are included.

Medical evaluation of a child with hypertension is largely directed toward ruling out secondary hypertension. The probability that blood pressure elevation is secondary is directly related to severity of hypertension and inversely related to age.

When evaluating a child’s blood pressure, a family history should be obtained, as children are at increased risk when primary hypertension is present in one or both parents. Use of umbilical artery catheters in sick newborns may result in renovascular hypertension due to platelet and fibrin emboli in the renal arteries; thus, it is important to obtain a neonatal history.

Physical examination should include blood pressure measurements in both arms and legs, palpation of peripheral pulses, ophthalmoscopic examination, cardiac auscultation, examination of the abdomen for masses and bruits, and search for physical signs of an endocrine disorder.

If the patient is obese or has a family history of primary hypertension, a urinalysis may be all that is indicated initially. Diagnostic studies in patients who are not obese will depend on the severity of hypertension. Urinalysis, urine culture, complete blood count, blood urea, creatinine, and electrolytes are indicated. Other studies for selected patients include intravenous pyelography, renal ultrasonography, and measurements of urinary and plasma catecholamines, plasma renin activity, serum and urinary aldosterone, and urinary and plasma corticoids.

It should be remembered that the most common causes for hypertension from infancy to age 6 are renal parenchymal diseases, coarctation of the aorta, and renal artery stenosis; from 6–10 years, the most common causes are renal artery stenosis, renal parenchymal diseases, and primary hypertension; in adolescence, primary hypertension and renal parenchymal disease are the most common causes of hypertension. Obviously, investigation of hypertensive children should rule out renal problems. The NHLBI task force recommends that when drug therapy is considered for a hypertensive child, an echocardiogram is justified for establishing baseline left ventricular mass to determine possible target organ damage that may be reversible.

Identifying Curable Forms of Hypertension

There is less emphasis now on detection of potentially curable forms of hypertension such as coarctation of the aorta, pheochromocytoma, primary aldosteronism, Cushing’s syndrome, and renovascular hypertension. The rationale is that these conditions are

are rare and most can be suspected on the basis of history, physical examination, and laboratory tests. Patients with primary aldosteronism or renovascular hypertension will respond to appropriate medical treatment and will therefore be protected against cardiovascular complications. Consequently, there is a growing opinion that the search for curable forms of hypertension should be directed chiefly toward patients with moderate or severe hypertension that does not respond to antihypertensive agents. Exceptions are hypertensive patients less than 30 years old since curable forms of hypertension are more common in this age group. In patients over 35, the prevalence of curable hypertension is so low that the returns often do not justify the cost and effort of an elaborate workup unless there is evidence from the history, physical examination, or routine laboratory work for suspecting its presence.

Screening for curable forms of hypertension should therefore be more selective than in the past as in young patients or when a routine examination reveals a suspicious finding. This discussion considers screening procedures for curable forms of hypertension. It should be noted that the most common cause for potentially curable hypertension in women of childbearing age may be the use of oral contraceptives and in men of any age, dietary indiscretion, including excessive alcohol consumption.

Coarctation of the Aorta

Coarctation of the aorta is present in about 40% of children with hypertension who are under the age of 5. The diagnosis can be made by detecting a decreased and/or lagging femoral pulse and by measuring blood pressure in both arms and one leg. A bruit over the back, rib notching, and indentation of the descending aorta (‘‘3+’’ sign) on chest x-ray are frequently present in older children. It is important that diagnosis and surgical repair take place before the child is 5–6 years old. Delay in surgery beyond that time may result in failure of blood pressure to return to normal.

Pheochromocytoma

This condition can occur at any age but its incidence is highest in young adults. The majority of patients are symptomatic and usually experience severe headaches with palpitations and inappropriate sweating with or without facial pallor and tremor of the upper extremities. The episodes may last from 5 minutes to several hours and can occur once a year or up to several times daily. The patient may be normotensive between symptomatic paroxysms, but more frequently, hypertension is persistent even though symptoms are intermittent. Patients with pheochromocytoma often exhibit a hypermetabolic state with recent weight loss although they are not all thin. Elevation of fasting blood glucose is not uncommon. Orthostatic hypotension is seen occasionally.

Rarely, pheochromocytoma is part of a multiple endocrine neoplasia syndrome that can include medullary carcinoma of the thyroid and adenomas of the parathyroid gland with primary hyperparathyroidism (multiple endocrine neoplasia type II or Sipple’s syndrome). This is usually familial; adrenal tumors are nearly always bilateral.

When suggestive symptoms or signs are present, special diagnostic tests screen for the presence of pheochromocytoma. The most reliable screening tests are measurement of plasma catecholamines (norepinephrine and epinephrine) in the resting and fasting state or measurement of metanephrines in a 24-hour urine specimen. Urinary vanillylmandelic acid and free catecholamines are less reliable.

When plasma catecholamines are between 500 and 2,000 ng/l in a patient whose symptoms suggest pheochromocytoma, the failure of 0.3 mg of clonidine given orally to suppress plasma catecholamines by 50% or more after 3 hours may indicate pheochromocytoma (clonidine suppression test). If blood pressure is normal, glucagon can be administered intravenously in a dose of 1 mg to stimulate secretion of catecholamines from a tumor. A rise in plasma catecholamines to more than 2,000 ng/l suggests pheochromocytoma even without a significant pressor response. Phentolamine should be immediately available to counteract a possible hypertensive crisis.

The tumor should be localized by computed tomographic (CT) scanning before surgical removal. 131-I-metaiodobenzylguanidine scintiscans are helpful when CT scanning fails to localize a tumor for patients with diagnostic elevations of plasma catecholamines or urinary metanephrines.

Primary Aldosteronism

This condition, which is due to a functioning adenoma or hyperplasia of the adrenal cortex, is rare. Primary aldosteronism is usually associated with mild to moderate elevation of blood pressure but can cause severe and resistant hypertension. While usually asymptomatic, it can cause muscular weakness, polyuria, nocturia, polydipsia, tetany, paresthesia, and headache.

The clue to the presence of primary aldosteronism is low serum potassium. A serum potassium level of less than 3.5 meq/l in a patient who is not taking oral diuretics or less than 3 meq/l in a patient who is receiving diuretics should arouse the clinician’s suspicion. If the patient has been taking a diuretic, it should be discontinued for at least 2 weeks. At the end of that time, if urinary potassium is less than 20 meq/24 hr, the diagnosis of primary aldosteronism is untenable even though serum potassium may be low. On the other hand, if urinary potassium is more than 40 meq/24 hr, further diagnostic studies to rule out primary aldosteronism are indicated. These include measurement of urinary aldosterone before and after 3 days of sodium loading (12 g of sodium chloride daily in addition to dietary intake). In primary aldosteronism, urinary aldosterone excretion will remain greater than or
equal to 8 μg/24 hr with urinary sodium greater than or equal to 250 meq/24 hr. Measurement of urinary sodium is essential to ensure adequate sodium intake. Nonsuppressible urinary aldosterone is a more important diagnostic feature than suppressed renin activity and urinary aldosterone is a more reliable index than plasma aldosterone. Nevertheless, plasma renin activity should be measured because very high levels may produce such severe aldosteronism that urinary aldosterone would not be suppressed by sodium loading.

Most, but not all, aldosterone tumors can be visualized by careful CT scanning. Sometimes a differential measurement of adrenal vein aldosterone concentrations is necessary to distinguish between an aldosterone-producing tumor and bilateral adrenal hyperplasia. The radioiodocholesterol scan has not been helpful because of low sensitivity. Furthermore, the isotope is expensive and not readily available.

**Renovascular Hypertension**

The prevalence of renovascular hypertension is probably no more than 2% of the hypertensive population, but it is the most common type of potentially curable hypertension except possibly those induced by oral contraceptives or excessive ingestion of alcohol. Not all patients with this condition are suitable candidates for surgery or balloon dilatation.

Renovascular disease may occur at any age. Before age 40, it is usually due to a fibrous dysplasia of the renal artery; it is more common in women than men and usually involves the right renal artery more often than the left, although it is bilateral in up to 30% of patients. After age 40, and especially after 50, the disease is usually atherosclerotic and lesions occur in the left renal artery more often than the right, although it can be bilateral in more than 50% of patients. Men are affected more often than women and atherosclerosis in other vascular beds, notably cerebral, coronary, and lower extremities, is the rule.

The history can yield important clues to this diagnosis:

- Abrupt onset of moderate to severe hypertension at any age.
- Hypertension resistant to a rational triple drug regimen.
- Diastolic hypertension greater than or equal to 110 mm Hg in patients less than 30 years old.
- Onset of diastolic hypertension after age 55.
- Acquired resistance to a previously effective regimen, especially in older patients.
- Acute flank pain with or without hematuria followed by onset of hypertension.

Clues to renovascular hypertension on physical examination include:

- A systolic-diastolic bruit in the epigastrium and/or one or both upper quadrants. This finding occurs in up to 60% of patients with fibrous dysplasia of the renal artery but rarely in atherosclerotic disease. A systolic bruit with no diastolic component is much less specific and can be heard in many elderly patients who do not have renal artery stenosis.
- Advanced hypertensive retinopathy (retinal hemorrhages and exudates with or without papilledema). In one series, nearly 40% of patients with group III or IV retinopathy had renovascular hypertension.
- Evidence of atherosclerotic occlusive disease of the lower extremities and/or carotids (e.g., bruit) increases the possibility of atherosclerotic renal vascular disease.

Screening tests for renovascular disease include the rapid sequence intravenous urogram or a dynamic renal flow scan with technetium-99m diethylenetriaminepentaacetic acid (DTPA). Features suggesting renal artery stenosis are a disparity in renal size of 1.5 cm or more and/or a disparity in appearance time of contrast medium on the intravenous pyelogram or a difference in perfusion between the two kidneys on the 99mTc-DTPA scan. Late hyperconcentration of contrast in the ischemic kidney is a less common finding on the intravenous pyelogram. These two modalities are similar in specificity and sensitivity; with either there is a 20% chance of missing a significant renal artery lesion. Consequently, a Seldinger-type renal arteriogram is indicated when there is a high index of suspicion that renal artery stenosis is present, even though screening tests are negative. In fact, screening tests can be omitted if clinical clues are sufficiently suggestive of renovascular hypertension. The intravenous digital subtraction angiogram, while less invasive than the conventional arteriogram, can miss orificial atherosclerotic lesions as well as branch lesions and does not permit evaluation of intrarenal vasculature. Consequently, it is not a substitute for the Seldinger arteriogram.

Visualization of a stenotic lesion on the arteriogram confirms diagnosis of renovascular disease, but does not necessarily indicate that the lesion is significant in causing hypertension ( renovascular hypertension). Renal vein renin determinations are often performed to find a ratio of plasma renin activity of 1.5 to 1.0 or more between the kidneys, favoring the ischemic side. Ideally, peripheral venous renin activity should be equal to or greater than renin activity from the nonischemic renal vein, indicating that renin secretion from the normal kidney is suppressed. However, this procedure has a false-negative rate of more than 50% in predicting surgical treatment outcome, especially in patients with fibrous dysplasia of the renal artery. Thus, it is no longer used as frequently as it once was. If the lesion appears to be very stenotic (≥ 75%) on the arteriogram, there is evidence of poststenotic dilatation and collateralization, and hypertension is less than 5 years’ duration, many authorities would proceed with surgical treatment or percutaneous transluminal angioplasty without renal vein renin
determinations or despite nonlalizing renal vein renin determinations.

Determining unstimulated peripheral venous plasma renin activity has not been a reliable screening test for renovascular disease. More recently, determination of peripheral plasma renin activity after stimulation with an oral dose of captopril has been reported to be a reliable index to the presence of renovascular disease and renovascular hypertension. However, confirmation is needed before this test is recommended for routine screening or prediction of outcome of surgery or transluminal angioplasty. Administration of 25 mg of captopril 1 hour before isotope renography with 99mTc-DTPA and 131I-orthoiodohippurate reportedly enhances predictability of the renogram in selecting patients with unilateral renal artery stenosis for surgery or percutaneous transluminal angioplasty.

Atherosclerotic renal artery lesions, if bilateral or occurring in an artery of a single kidney, can jeopardize total renal function. Frequently, the indication for surgery or transluminal angioplasty is preservation of renal function, even though hypertension can be well controlled medically.

Transluminal angioplasty has been more successful in treating fibrous dysplasias of the renal artery than atherosclerotic lesions where recurrence has been high, especially when the lesions occur in the orifice of the renal artery rather than more distally. Medical treatment of renovascular hypertension is often effective, but close follow-up of individual renal function, usually by technetium scan, is indicated, especially for atherosclerotic lesions, because renal function may deteriorate despite good blood pressure control.

Organ Damage

A variety of target organs bear the brunt of hypertensive damage (end-organ damage), including the eyes, brain, heart and arteries, and kidneys. The extent of target organ damage or complications of hypertension is an important determinant of prognosis and often influences urgency of treatment and drugs selected for treatment.

The Eyes

Examination of the optic fundi can detect longstanding changes of chronic hypertension and acute changes resulting from accelerated or malignant hypertension. If pupillary constriction precludes visualization of the entire fundus, dilatation may be accomplished with topical mydriatics.

Arteriosclerotic vascular changes of chronic hypertension include arteriovenous “nicking” (especially if they are at a distance from the optic disk), increased arteriolar light reflex, and arteriolar narrowing, irregularity, or tortuosity.

Accelerated hypertension is characterized by hemorrhages and fresh exudates, with or without papilledema. This clinical picture, now rare, requires prompt evaluation and institution of therapy as hypertensive end-organ damage may progress quite rapidly (days to weeks) in untreated accelerated hypertension. A recent study indicates that up to one half of white patients with accelerated hypertension may have renovascular hypertension. By contrast, renovascular hypertension is unusual in black patients, even with accelerated hypertension. Papilledema is the sine qua non for diagnosis of malignant hypertension (Keith-Wagener-Barker group IV) and is sometimes found with hypertensive encephalopathy. Retinal hemorrhages and exudates may also result from diabetes mellitus with or without hypertension and usually in association with retinal microaneurysms.

The Brain

Hypertension may cause damage to the brain in the form of strokes (cerebrovascular accidents) resulting from hemorrhage (intracerebral or subarachnoid), thrombosis, or thromboembolism (often originating from atherosclerotic plaques in the carotid arteries or aorta). Cerebral microaneurysms may be more frequent in adults with hypertension; rupture can lead to hypertensive intraparenchymal hemorrhage. Symptoms often ascribed by patients to blood pressure elevation, including headache and dizziness, do not correlate with blood pressure elevation except in unusual circumstances of accelerated or malignant hypertension.

The most common cause of dementia is Alzheimer’s disease, which is unrelated to atherosclerosis and is not more prevalent in hypertensive than normotensive patients. However, hypertensive atherosclerotic disease is an important cause of dementia in many patients. Two broad categories of vascular dementia are recognized: multi-infarct dementia and dementia ofBinswanger’s disease. In multi-infarct dementia, large and/or small (arteriolar) vessel atherothrombotic infarcts accumulate over time, resulting in dementia. Lacunae are small (<1 cm), deep infarcts from arteriolar occlusive disease that are common in hypertensive patients and can be clinically silent or produce lacunar stroke syndromes (e.g., pure motor or pure sensory stroke). Multiple lacunae can result in a “lacunar state” consisting of pseudobulbar palsy, corticospinal signs, and dementia. Magnetic resonance imaging is more sensitive than CT imaging for detecting lacunae.

Binswanger-type dementia is a less common form of vascular dementia usually related to hypertensive atherosclerotic disease of long penetrating white matter arteries. This produces a distinct pattern of subcortical periventricular white matter infarction visible in CT as diffuse periventricular hypodensity (leukoaraiosis) or in magnetic resonance imaging as diffuse periventricular increased T2 signal.

It is unclear how the site of vascular involvement in the hypertensive individual is determined. Clinical hallmarks of vascular dementia are the stuttering evolution combined with focal signs indicating brain infarction. There is some evidence that treating hypertension can prevent worsening of the multi-infarct state.

Hypertensive encephalopathy occurs in the setting of acute, severe blood pressure elevations (diastolic blood pressure often >140 mm Hg). It is usually characterized by papilledema, headache, confusion, or somnolence (which may progress to stupor or coma), and nonfocal or fleetingly focal neurological signs. Prompt lowering of blood pressure by appropriate intravenous agents is indicated.

**The Heart and Arteries**

The initial response of the heart to the pressure overload of systemic hypertension is concentric hypertrophy of the left ventricle. The degree of hypertrophy correlates with mean 24-hour blood pressure measured by ambulatory blood pressure monitoring.

The usual clinical indexes of left ventricular hypertrophy, which include physical examination (sustained apical impulse and fourth heart sound), ECG, and chest x-ray, are relatively insensitive. The echocardiogram is a more sensitive, noninvasive index of left ventricular hypertrophy than the ECG. The one-dimensional (M-mode) echocardiogram measures thickness of the left ventricular septum and posterior free wall; left ventricular mass can be calculated in grams from these measurements. When assessed in this fashion, left ventricular mass may regress toward normal after only a few weeks of antihypertensive treatment with drugs that inhibit sympathetic nervous system activity. The role of the echocardiogram in routine clinical evaluation of hypertensive patients has not been established, however.

Long-standing hypertension is a major risk factor for development of atherosclerotic coronary artery disease, which is usually manifested by angina pectoris or myocardial infarction. Reducing elevated blood pressure may reduce myocardial oxygen demand and thus ameliorate myocardial ischemia.

Long-standing hypertension may also lead to congestive heart failure as a result of either chronic pressure overload (hypertensive cardiomyopathy) or myocardial infarction (ischemic cardiomyopathy). In fact, the Framingham study found that the most frequent antecedent (and presumed cause) of congestive heart failure is systemic hypertension.

Hypertension accelerates the atherosclerotic process in the peripheral arteries, leading to increased incidence of occlusive disease in the lower extremities manifested by absence or diminution of peripheral arterial pulsations. Hypertension also predisposes to medial degeneration of the large and medium-sized arteries manifested by dilatation and tortuosity of peripheral arteries and aortic tortuosity and dilatation on roentgenogram of the chest. Osler’s sign (a palpable radial artery after temporary manual occlusion of the ipsilateral brachial artery) may indicate an especially stiff, noncompliant arterial tree in the elderly and is frequently associated with artifactual elevations in systolic blood pressure (pseudohypertension) obtained by cuff.

**The Kidney**

Uncontrolled hypertension can lead to kidney damage. Nephrosclerosis is characterized pathologically by sclerosis of the interlobular artery walls and afferent arterioles, effectively reducing renal blood flow. This can eventually culminate in end-stage renal failure. Progression of nephrosclerosis may be more rapid in hypertensive blacks than whites.

Unfortunately, the usual clinical indexes of renal function (blood urea, nitrogen, and serum creatinine) are relatively insensitive to early renal dysfunction as a consequence of hypertension; up to one half of renal function may be lost before serum creatinine is notably elevated. Urinalysis is also characteristically normal in the initial stages of nephrosclerosis. An early clinical indicator of hypertensive nephrosclerosis may be a modest elevation in serum uric acid.

It may be difficult to determine cause and effect in the patient with coexisting hypertension and renal insufficiency since hypertension is a frequent sequela of renal failure from any cause. Several clinical clues may be helpful. The history may indicate one antedates the other; for example, profuse proteinuria before onset of hypertension suggests primary renal disease with secondary hypertension. In mild to moderate hypertension, clinically evident azotemia is unusual even after many years.

In accelerated hypertension with associated progressive azotemia, prompt control of blood pressure is of great value in halting progression toward uremia. In the acute state, after blood pressure is lowered, kidney function may be unchanged or even deteriorate transiently, but in the long term, renal function may improve and exceed what it was before treatment.

Effective control of associated hypertension may also halt or slow progression of a variety of renal diseases, especially diabetic nephropathy. In essential hypertension, it is distinctly unusual for a patient taking antihypertensive medication to progress to accelerated or malignant hypertension.

**Summary**

The ultimate purpose of office evaluation of the hypertensive patient is to provide optimal management of blood pressure and associated risk factors. The workup includes a valid estimate of average blood pressure, including home blood pressure measurements, assessment of the degree of target organ damage, and identification of other risk factors, including family history. The history and physical examination should be directed to the principal target organs, including the optic fundi, central
nervous system, heart, and kidneys. Laboratory evaluation should include urinalysis, ECG, and determinations of blood hemoglobin/hematocrit, creatinine, potassium, glucose, and cholesterol, including HDL fraction.

This information will alert the physician to the possibility of curable forms of hypertension such as coarctation of the aorta, pheochromocytoma, primary aldosteronism, and renovascular hypertension. The office evaluation is also concerned with estimating prognosis and extent of organic damage, which is essential in planning management. The nurse or trained allied health professional should be used to the fullest possible extent both in evaluation and management of hypertensive patients.

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Appendix

Age- and Sex-Specific Blood Pressure Distributions in Children

If a child or adolescent has blood pressure greater than the 90th percentile for age, but is tall with weight proportional for age, or tall and lean, blood pressure may be normal. If the child has an increase in body weight, blood pressure may be normal if the increase is in lean body mass but abnormal if the child is obese.
AGE-SPECIFIC PERCENTILES OF BLOOD PRESSURE MEASUREMENTS IN BOYS AGES 1 YEAR TO 13 YEARS

AGE-SPECIFIC PERCENTILES OF BLOOD PRESSURE MEASUREMENTS IN GIRLS AGES 1 YEAR TO 13 YEARS

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<th>90TH PERCENTILE</th>
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AGE-SPECIFIC PERCENTILES OF BLOOD PRESSURE MEASUREMENTS IN BOYS AGES 13 TO 18 YEARS

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AGE-SPECIFIC PERCENTILES OF BLOOD PRESSURE MEASUREMENTS IN GIRLS AGES 13 TO 18 YEARS

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R W Gifford, Jr, W Kirkendall, D T O'Connor and W Weidman

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