suggested on the basis of a good history, physical examination, and routine laboratory tests.

Renal Parenchymal Hypertension

Chronic kidney disease is the most common cause of secondary hypertension. Hypertension is present in more than 85% of patients with chronic kidney disease and is a major factor causing their increased cardiovascular morbidity and mortality. The mechanisms causing the hypertension include an expanded plasma volume and peripheral vasoconstriction, with the latter caused by both activation of vasoconstrictor pathways (renin-angiotensin and sympathetic nervous systems) and inhibition of vasodilator pathways (nitric oxide). Renal insufficiency should be considered when proteinuria is found by dipstick or when the serum creatinine level is greater than 1.2mg/dL in women with hypertension or greater than 1.4 mg/dL in men with hypertension.

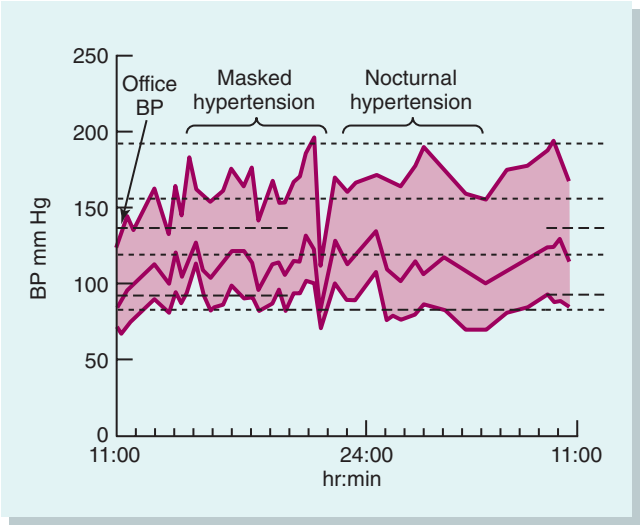

FIGURE 12-9 Twenty-four hour ambulatory blood pressure (BP) monitor tracing shows both masked hypertension and nocturnal hypertension in a 55-year-old man with stage 3 chronic kidney disease. Treatment with three different antihypertensive medications produced an office BP of 125/75 mm Hg, which seemed to be at goal. However, progressive hypertensive heart disease and deterioration of renal function suggested masked hypertension. Ambulatory monitoring revealed that the patient's treated BP was much higher out of the office, documenting both masked hypertension (ambulatory BP of 175/95 mm Hg) and sustained nocturnal hypertension (BP of 175/90 mm Hg). Additional medication was added. (Courtesy Ronald G. Victor, MD, Hypertension Division, Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, Tex.)

Renovascular Hypertension

Unilateral or bilateral renal artery stenosis is present in fewer than 2% of patients with hypertension in a general medical practice but in up to 30% of patients with medically refractory hypertension. The main causes of renal artery stenosis are atherosclerosis (85% of patients), typically in older adults with other clinical manifestations of systemic atherosclerosis, and fibromuscular dysplasia (15% of patients), typically in women between the ages of 15 and 50 years. Unilateral renal artery stenosis leads to underperfusion of the juxtaglomerular cells, resulting in renin-dependent hypertension even though the contralateral kidney is able to maintain normal blood volume. In contrast, bilateral renal artery stenosis (or unilateral stenosis with a solitary kidney) constitutes a potentially reversible cause of progressive renal failure and volume-dependent hypertension.

The following clinical clues increase the suggestion of renovascular hypertension: any hospitalization for urgent or emergent hypertension; recurrent *flash* pulmonary edema; recent worsening of long-standing, previously well-controlled hypertension; severe hypertension in a young adult or after 50 years of age; precipitous and progressive worsening of renal function in response to angiotensin-converting enzyme (ACE) inhibition or angiotensin-receptor blockade (ARB); unilateral small kidney by any radiographic study; extensive peripheral arteriosclerosis; or a flank bruit. The diagnosis is confirmed by noninvasive testing with MR or spiral CT angiography (Fig. 12-10).

Renal artery angioplasty often cures fibromuscular dysplasia. Atherosclerotic renal artery stenosis should be treated with intensive medical management of atherosclerotic risk factors (e.g., hypertension, lipids, smoking). Revascularization should be

TABLE 12-5 GUIDE TO EVALUATION OF SECONDARY HYPERTENSION

PROBABLE DIAGNOSIS	CLINICAL CLUES	DIAGNOSTIC TESTING
Renal parenchymal hypertension	Estimated GFR <60 mL/min/1.73m^2 Urine albumin : creatinine >30 mg/g	Renal ultrasound
Renovascular disease	New elevation in serum creatinine, significant elevation in serum creatinine with initiation of ACEIs or ARBs, refractory hypertension, flash pulmonary edema, abdominal bruit Coarctation of the aorta Arm BP > leg BP BP, chest bruits, rib notching on chest radiograph	MR or CT angiography, invasive angiogram Arm pulses > leg pulses, chest MR or CT, aortogram
Primary aldosteronism	Hypokalemia, refractory hypertension	Plasma renin and aldosterone, 24-hr urine potassium, 24-hr urine aldosterone and potassium after salt loading, adrenal CT scan, adrenal vein sampling
Cushing's syndrome	Truncal obesity, wide and blanching purple striae, muscle weakness, diabetes	24-hr Urine cortisol, dexamethasone suppression test, adrenal CT scan
Pheochromocytoma	Spells of paroxysmal hypertension, palpitations, perspiration, pallor, pain in the head	Plasma and 24-hr urine metanephrines and catecholamines, adrenal CT scan
Obstructive sleep apnea	Loud snoring, daytime somnolence, obesity, large neck	Sleep study

Modified from Kaplan NM: Clinical hypertension, ed 8, Philadelphia, 2002, Williams & Wilkins.

ACEIs, Angiotensin-converting enzyme inhibitors; ARBs, angiotensin-receptor blockers; BP, blood pressure; CT, computed tomography; GFR, glomerular filtration rate; MR, magnetic resonance.