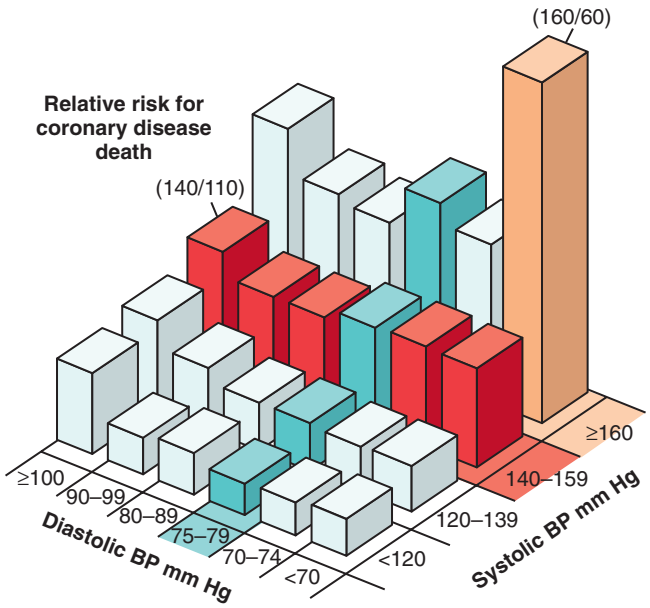

FIGURE 12-13 Joint influences of systolic blood pressure (BP) and diastolic BP on the risk of fatal coronary heart disease in the Multiple Risk Factor Intervention Trial. (Neaton JD, Wentworth D: Serum cholesterol, blood pressure, cigarette smoking, and death from coronary heart disease: overall findings and differences by age for 316,099 white men, Arch Intern Med 152:56–64, 1992.)

risk of fatal coronary heart disease as a BP of 140/110 mm Hg (pulse pressure, 30 mm Hg) (Fig. 12-13).

In older persons with isolated systolic hypertension, lowering the systolic pressure from more than 160 to less than 150 mm Hg reduces the risks of stroke, myocardial infarction, and overall cardiovascular mortality; it also reduces heart failure admissions and slows the progression of dementia (level A evidence). Trial data do not yet exist in older persons to determine whether treatment of isolated elevations in systolic pressure below 140 mm Hg is beneficial. However, in the absence of such data, treatment may be warranted to prevent progression of systolic hypertension, if the patient can tolerate the treatment without side effects such as orthostatic hypotension.

The combination of a low-dose thiazide diuretic with a dihydropyridine CCB or an ACEI reduces the risk of cardiovascular events in older patients with isolated systolic hypertension (level A evidence). To prevent orthostatic hypotension, medication should be titrated to standing BP, and one low-dose medication should be started at a time.

Blood Pressure Lowering for Secondary Prevention of Stroke

Most neurologists do not recommend BP reduction during an acute stroke. After the acute phase, BP should be lowered with a thiazide diuretic, and an ACEI or additional drugs should be added as needed to achieve BP levels lower than 140/90 mm Hg; whether BP should be lowered further is unclear.

Hypertensive Disorders of Women

Oral contraceptives cause a small increase in BP in most women but rarely cause a large increase into the hypertensive range. If hypertension develops, oral contraceptive therapy should be discontinued in favor of other methods of contraception. Oral estrogen replacement therapy seems to cause a small increase in BP. In contrast, transdermal estrogen (which bypasses first-pass hepatic metabolism) seems to cause a small but consistent decrease in BP.

Hypertension, the most common nonobstetric complication of pregnancy, is present in 10% of all pregnant women. One third of these cases are caused by chronic hypertension, and two thirds are caused by preeclampsia, which is defined as an increase in BP to 160/110 mm Hg or greater after the 20th week of gestation accompanied by proteinuria (>300 mg/24 hours) and pathologic edema, sometimes accompanied by seizures (eclampsia) and the multisystem HELLP syndrome of hemolysis (H), elevated liver enzymes (EL), and low platelets (LP). Oral drug therapy should be initiated with any one of three preferred drugs: labetalol (400 to 2400 mg daily). Intravenous labetalol (0.5 to 2 mg/minute up to a cumulative dose of 300 mg) has replaced hydralazine as the drug of choice to treat severe preeclampsia/eclampsia.

Resistant Hypertension

Resistant hypertension, defined as persistence of usual BP above 140/90 mm Hg despite treatment with full doses of three or more different classes of medications in rational combination and including a diuretic, is the most common reason for referral to a hypertension specialist. In practice, most of these patients have pseudoresistant hypertension due to (1) *white-coat aggravation,* a white-coat reaction superimposed on chronic hypertension that is well controlled with medication outside the physician's office; (2) an inadequate medical regimen; (3) nonadherence; or (4) ingestion of pressor substances. Common shortcomings of the medical regimen include undertreatment of hypertension with monotherapy and use of clonidine, a potent central sympatholytic that causes rebound hypertension between doses particularly, with PRN dosing. Several common causes of pseudoresistant hypertension are related to the patient's behavior, including medication nonadherence, recidivism with lifestyle modification (e.g., obesity, high-salt diet, excessive alcohol intake), and habitual use of pressor substances such as sympathomimetics (e.g., tobacco, cocaine, methamphetamine, phenylephrine-containing cold or herbal remedies) or NSAIDs, with the latter causing renal sodium retention. Once these behavioral factors have been excluded, the search should begin for causes of secondary hypertension.

The most common forms of secondary hypertension are chronic kidney disease and primary aldosteronism. Significant impairment in renal function can be present with serum creatinine in the range of 1.2 to 1.4 mg/dL or even lower, particularly in older patients with little muscle mass. To avoid this pitfall, calculation of the glomerular filtration rate (GFR) by equations based on serum creatinine, age, weight, and measurement of the urinary albumin-to-creatinine ratio from a spot-urine specimen should be an essential part of the routine evaluation of every patient with hypertension. Either a loop diuretic (e.g., a furosemide) or a potent thiazide-type diuretic (e.g, chlorthalidone) may be required to control hypertension in patients with chronic kidney disease. The treatment of primary aldosteronism was discussed earlier.