3. Screening for Hypertension

RECOMMENDATION
Screening for hypertension is recommended for all children and adults (see *Clinical Intervention*).

Burden of Suffering

Hypertension is usually defined as a diastolic blood pressure of 90 mm Hg or higher or a systolic pressure of 140 mm Hg or higher.[1] It is present in an estimated 43 million Americans and is more common in blacks and older adults.[1a] Hypertension is a leading risk factor for coronary heart disease, congestive heart failure, stroke, ruptured aortic aneurysm, renal disease, and retinopathy. These complications of hypertension are among the most common and serious diseases in the U.S., and successful efforts to lower blood pressure could thus have substantial impact on population morbidity and mortality. Heart disease is the leading cause of death in the U.S., accounting for nearly 740,000 deaths each year (287 deaths per 100,000 population), and cerebrovascular disease, the third leading cause of death, accounts for about 150,000 deaths each year (58/100,000).[2] Milder forms of hypertension predict progression to more severe elevations and development of cardiovascular disease.[1,3,4] Coronary heart disease mortality begins to increase at systolic blood pressures above 110 mm Hg and at diastolic pressures above 70 mm Hg.[5] The prevalence of unrecognized and uncontrolled hypertension, and the mortality from cardiovascular disease, have declined substantially in the U.S. in the past several decades.[1]

Treatable (also known as secondary) causes of hypertension such as aortic coarctation or renovascular disease also may be associated with severe consequences, including congestive heart failure, aortic rupture, or stroke.[6–9] There are no population data available for estimating the true prevalence of secondary hypertension. The incidence of coarctation of the aorta has been estimated at 0.2–0.6/1,000 live births and the prevalence at 0.1–0.5/1,000 children.[10–12]

Accuracy of Screening Tests

The most accurate devices for measuring blood pressure (e.g., intra-arterial catheters) are not appropriate for routine screening because of their invasiveness, technical limitations, and cost. Office sphygmomanometry (the blood pressure cuff) remains the most appropriate screening test for hypertension in the asymptomatic population. Although this test is highly accurate when performed correctly, false-positive and false-negative results (i.e., recording a blood pressure that is not representative of the patient's average blood pressure) do occur in clinical practice.[13] One study found that 21% of persons diagnosed as mildly hypertensive based on office sphygmomanometry had no evidence of hypertension when 24-hour ambulatory recordings were obtained.[14]

Errors in measuring blood pressure may result from instrument, observer, and/or patient factors.[15] Examples of instrument error include manometer dysfunction, pressure leaks, stethoscope defects, and cuffs of incorrect width or length for the patient's arm size. The observer can introduce errors due to sensory impairment (difficulty hearing Korotkoff sounds or reading the manometer), inattention, inconsistency in recording Korotkoff sounds (e.g., Phase IV vs. Phase V), and subconscious bias (e.g., "digit preference" for numbers ending with zero or preconceived notions of "normal" pressures). The patient can be the source of misleading readings due to posture and biologic factors. Posture (i.e., lying, standing, sitting) and arm position in relation to the heart can affect results by as much as 10 mm Hg.[15] Biologic factors include anxiety, meals, tobacco, alcohol, temperature changes, exertion, and pain. Due to these limitations in the test-retest reliability of blood pressure measurement, it is commonly recommended that hypertension be diagnosed only after more than one elevated reading is obtained on each of three separate visits over a period of one to several weeks.[1]

Additional factors affect accuracy when performing sphygmomanometry on children; these difficulties are especially common when testing infants and toddlers under 3 years of age.[16–18] First, there is increased variation in arm circumference, requiring greater care in the selection of cuff sizes.[19] Second, the examination is more frequently complicated by the anxiety and restlessness of the patient. Third, the disappearance of Korotkoff sounds (Phase V) is often difficult to hear in children and Phase IV values are often substituted. Fourth, erroneous Korotkoff sounds can be produced inadvertently by the pressure of the stethoscope diaphragm against the antecubital fossa. Finally, the definition of pediatric hypertension has itself been uncertain because of confusion over normal values during childhood. The definition of hypertension in childhood is essentially arbitrary, based on age-specific percentile.[18] Age-, sex-, and height-specific blood pressure nomograms for U.S. children and adolescents have been published more recently, based on data from 56,108 children aged 1–17 years.[20]

Self-measured (home) blood pressure and ambulatory blood pressure monitoring may provide useful information in special circumstances (e.g., research, persistent "white-coat" hypertension), but there is insufficient evidence at present to warrant their routine use in screening.[1,21–28]

Effectiveness of Early Detection

There is a direct relationship between the magnitude of blood pressure elevation and the benefit of lowering pressure. In persons with malignant hypertension, the benefits of intervention are most dramatic; treatment increases 5-year survival from near zero (data from historical controls) to 75%.[29] Over the past 30 years, the results of many randomized clinical trials of the effects of antihypertensive drug therapy on morbidity and mortality in adult patients (≥21 years of age) with less severe hypertension have been published.[30–32] The efficacy of treating hypertension is clear, as demonstrated in a number of older randomized controlled trials in adults with diastolic blood pressures ranging from 90 to 129 mm Hg.[33–38] For example, in the Veterans Administration Cooperative Study on Antihypertensive Agents, middle-aged men with diastolic blood pressure averaging 90 through 114 mm Hg experienced a significant reduction in "morbid" events (e.g., cerebrovascular hemorrhage, congestive heart failure) after treatment with antihypertensive medication.[34]

Persons with mild (Stage 1) to moderate (Stage 2)[1] diastolic hypertension (90–109 mm Hg) also benefit from treatment.[30,39–41] This was confirmed in the Hypertension Detection and Follow-Up Program, a randomized controlled trial involving nearly 11,000 hypertensive men and women, of whom 40% were black.[39] The intervention group received standardized pharmacologic treatment ("stepped care") while the control group was referred for community medical care. There was a statistically significant 17% reduction in 5-year all-cause mortality in the group receiving standardized drug therapy; the subset with diastolic blood pressure 90–104 mm Hg experienced a 20% reduction in mortality.[39] Deaths due to cerebrovascular disease, ischemic heart disease, and other causes were also significantly reduced in the stepped care group.[42] Similar effects on all-cause mortality and cardiovascular events have been reported in other randomized controlled trials, such as the Australian National Blood Pressure Study (initial diastolic blood pressure 95–109 mm Hg)[40] and the Medical Research Council (MRC) trial (diastolic blood pressure 90–109 mm Hg).[41] In these two trials, the relative reduction in rates of stroke or other trial endpoints with treatment was similar in those with diastolic blood pressures <95 or 95–99 mm Hg and those with higher diastolic blood pressures, although the absolute benefit was less due to smaller initial risk of stroke and other diseases at lower blood pressures. Both trials included untreated control groups and did not report a significant reduction in deaths from noncardiovascular causes in the actively treated groups, confirming that the benefit was due to antihypertensive treatment rather than to other medical care.

Earlier studies included some subjects over age 65 years, but in insufficient numbers to permit firm conclusions. Four large, randomized placebo-controlled trials have since demonstrated conclusively the benefit of antihypertensive treatment in elderly subjects (aged 60–97 years).[43–48] Three of these studies included persons with diastolic blood pressures of 90–120 mm Hg, and among them reported significant reductions in all-cause mortality,[46] cardiovascular mortality,[43,46] cardiovascular events,[47] and strokes.[46,47] The Systolic Hypertension in the Elderly Program (SHEP) trial included over 4,000 subjects ≥60 years of age with isolated systolic hypertension (systolic blood pressure ≥ 160 mm Hg, with diastolic blood pressure , 90 mm Hg), and reported significant reductions in the incidence of stroke, myocardial infarction, and left ventricular failure.[48] A meta-analysis combining these and other trials that included persons aged ≥60 years demonstrated that antihypertensive treatment in elderly persons significantly reduced mortality from all causes (–12%), stroke (–36%), and coronary heart disease (–25%), as well as stroke and coronary heart disease morbidity.[49] This meta-analysis suggested reduced benefit with increasing age, although differences were not statistically significant. A second meta-analysis of randomized controlled trials in persons over age 60 years concluded that absolute 5-year morbidity and mortality benefits derived from trials were greater for older than for younger subjects.[50] This meta-analysis calculated that 18 (95% CI, 14–25) elderly hypertensive subjects needed to be treated for 5 years to prevent one cardiovascular event.

Treatment of hypertension is associated with multiple benefits, including reduced coronary heart disease and vascular deaths, but meta-analyses suggest it produces the largest reductions in cerebrovascular morbidity and mortality.[30–32,49,50] Improved treatment of high blood pressure has been credited with a substantial portion of the greater than 50% reduction in age-adjusted stroke mortality that has been observed since 1972.[1,51,52]

Although the efficacy of antihypertensive treatment for essential (also called primary) hypertension has been well established in clinical research, certain factors may influence the magnitude of benefit from hypertension screening achieved in actual practice. Compliance with drug therapy may be limited by the inconvenience, side effects, and cost of these agents.[53,54] Serious or life-threatening drug reactions in the clinical trials were rare, but less serious side effects were common, resulting in discontinuation of randomized treatment (almost 20% by the fifth year in the MRC trial,[41] for example) or a substantial increase in patient discomfort.[34] Higher incidences of mild hypokalemia, hyperuricemia, and elevated fasting blood sugar have also been reported in treated individuals.[35] A population-based case-control study suggested an increased risk of primary cardiac arrest with certain diuretic regimens (e.g., higher doses, use without potassium-sparing therapy).[55] However, current drug regimens, including low-dose diuretics, are associated with fewer adverse effects and with favorable effects on quality of life.[55a] Newer classes of drugs (e.g., calcium channel blockers, angiotensin-converting enzyme inhibitors) have not been assessed in long-term trials with clinical endpoints. Their effects on cardiovascular morbidity and mortality may differ from the effects reported in the clinical trials cited above, which used diuretics or beta-blockers.

Whether hypertension screening is equally effective for other populations or with treatments other than drugs is less clear. The benefits of hypertension treatment are less well studied in certain population groups, such as children (see below), Native Americans, Asians and Pacific Islanders, and Hispanics. The effects of nonpharmacologic first-line therapy (i.e., weight reduction in overweight patients, increased physical activity, sodium restriction, and decreased alcohol intake) on cardiovascular morbidity and mortality are unstudied. Although these nonpharmacologic therapies can sometimes lower blood pressure in the short term,[1,56–62a] the magnitude of blood pressure reduction achieved is generally smaller than that achieved with drug therapy, and both the magnitude and duration of reduction in actual practice may be limited by biologic factors (e.g., varying responsiveness to sodium restriction) and the difficulties of maintaining behavioral changes (e.g., weight loss). Some of these interventions, such as sodium restriction, may also have adverse effects on quality of life.[63]

The detection of high blood pressure during childhood is of potential value in identifying those children who are at increased risk of primary hypertension as adults and who might benefit from earlier intervention and follow-up. Hypertensive vascular and end-organ damage may begin in childhood,[64–69] although it is unclear how strongly these pathophysiologic changes are associated with subsequent cardiovascular disease. Prospective cohort studies have shown that children with high blood pressure are more likely than other children to have hypertension as adults.[70–78] Correlation coefficients from these studies were generally less than 0.5, however, suggesting a limited role for high blood pressure in childhood as a predictor of adult hypertension. Although controlled trials in children show that short-term (up to 3 years) effects on blood pressure can be achieved with changes in diet and activity,[79–82] studies demonstrating long-term changes in blood pressure are lacking. There are no trials showing that lowering blood pressure in childhood results in reduced blood pressure in adulthood. A relationship between lowering blood pressure during childhood and improved morbidity and mortality in later life is unlikely to be demonstrated, given the difficulty of performing such studies.

A relatively high proportion of children with hypertension have secondary, potentially curable forms. Among children and adolescents whose hypertension was evaluated in primary care centers, an estimated 28% had secondary hypertension (e.g., renal parenchymal disease, coarctation of the aorta).[69] This contrasts with hypertensive adults seen in primary care settings, of whom only 7% are estimated to have secondary hypertension.[83] Screening children and adolescents may be justifiable if the morbidity of these conditions is improved by early detection and treatment. Many causes of secondary hypertension in childhood are detectable by careful history-taking (e.g., preterm birth, umbilical artery catheter, chronic pyelonephritis, renal disease, bronchopulmonary dysplasia; symptoms of cardiac, renal, endocrinologic, or neurologic disease) or physical examination (e.g., murmur, decreased femoral pulses, abdominal bruit).[69,84] Characteristic symptoms and signs, such as those of aortic coarctation, are often overlooked, however.[85–87] Numerous surgical case series suggest that delay in surgical repair of aortic coarctation increases the likelihood of irreversible hypertension,[88–94] although none of these series controlled for other differences between persons presenting early versus late in life. Uncontrolled studies indicate that some important causes of hypertension for which definitive cures are available, including coarctation and renovascular disease, may not be diagnosed until complications such as congestive heart failure, aortic rupture, or stroke occur.[6–9] Prognosis with early surgical intervention is improved compared with historical controls.[88,95]

Recommendations of Other Groups

Recommendations for adults have been issued by the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure,[1] and similar recommendations have been issued by the American Heart Association.[96] These call for routine blood pressure measurement at least once every 2 years for adults with a diastolic blood pressure below 85 mm Hg and a systolic pressure below 130 mm Hg. Measurements are recommended annually for persons with a diastolic blood pressure of 85–89 mm Hg or systolic blood pressure of 130–139 mm Hg. Persons with higher blood pressures require more frequent measurements. The American College of Physicians (ACP)[97] and the American Academy of Family Physicians (AAFP)[98] recommend that all adults 18 years and older be screened for hypertension every 1–2 years. The AAFP policy is currently under review. The ACP also recommends screening at every physician visit for other reasons, and that those in high-risk groups (e.g., diastolic 85–89 mm Hg, previous history of hypertension) be screened on an annual basis. The Canadian Task Force on the Periodic Health Examination recommends that all persons aged 21 years and over receive a blood pressure measurement during any visit to a physician (“case finding”).[99]

The American Academy of Pediatrics (AAP),[100] the National Heart, Lung, and Blood Institute,[18] the AAFP,[98] Bright Futures,[101] the American

Medical Association,[102] and the American Heart Association[103] recommend that children and adolescents receive blood pressure measurements every 1 or 2 years during regular office visits. The Canadian Task Force found insufficient evidence to recommend for or against routine blood pressure measurement in persons under age 21 years.[99] The AAP recommends against universal neonatal blood pressure screening.[104]

Discussion

It is clear from several large randomized clinical trials that lowering blood pressure in hypertensive adults is beneficial and that death from several common diseases can be reduced through the detection and treatment of high blood pressure. Estimates suggest that an average diastolic blood pressure reduction of 5–6 mm Hg in everyone with hypertension could reduce the incidence of coronary heart disease by 14% and the incidence of strokes by 42%.[30,31] At the same time, it is important for clinicians to minimize the potential harmful effects of detection and treatment. For example, if performed incorrectly, sphygmomanometry can produce misleading results. Some hypertensive patients thereby escape detection (false negatives) and some normotensive persons receive inappropriate labeling (false positives), which may have certain psychological, behavioral, and even financial consequences.[105] Treatment of hypertension can also be harmful as a result of medical complications, especially related to drugs. Clinicians can minimize these effects by using proper technique when performing sphygmomanometry, making appropriate use of nonpharmacologic methods, and prescribing antihypertensive drugs with careful adherence to published guidelines.[1,106–108]

The diastolic blood pressure above which therapy has been proven effective (i.e., diastolic blood pressure . 90 mm Hg) is to a large extent based on the artificial cutpoints chosen for study purposes rather than on a specific biologic cutpoint defining increased risk. The coronary heart disease mortality risk associated with blood pressure occurs on a continuum that extends well below the arbitrarily defined level for abnormal blood pressure, beginning for systolic blood pressure above 110 mm Hg and for diastolic pressure above 70 mm Hg.[5] Nevertheless, many organizations outside the U.S. have been reluctant to recommend drug therapy for persons with diastolic blood pressures below 100 mm Hg who lack additional risk factors.[106,108–111] Drug treatment of mild hypertension is of particular concern for young adults: the evidence for therapeutic benefit comes primarily from several older trials[34,36,38] that included only a few individuals in their 20s, the potential adverse effects of decades of antihypertensive therapy are undefined, and the absolute benefits in young adults are likely to be limited given their small risk of stroke and coronary heart disease.

For persons with mild hypertension, most recommendations suggest including age and/or the presence of other cardiovascular disease risk factors or concomitant diseases (e.g., smoking, obesity, renal disease, peripheral vascular disease) to modify treatment decisions.[1,106,108–111]

Tracking studies and pathophysiologic evidence suggest there may be some benefit from early detection of primary hypertension in childhood, but there is insufficient evidence to support routine screening solely for this purpose. The lack of evidence is of concern because it is unclear whether a policy of routinely screening all children and adolescents to detect primary hypertension would achieve sufficient clinical benefit later in life to justify the costs and potential adverse effects of widespread testing and treatment. Potentially curable causes of hypertension, which account for a relatively large proportion of cases in young children, are often overlooked on history and physical examination, with rare but potentially catastrophic consequences. Evidence from case series and multiple time series indicate that early detection of secondary hypertension in childhood is of substantial benefit to the small number of patients affected.

CLINICAL INTERVENTION

Periodic screening for hypertension is recommended for all persons ≥21 years of age ("A" recommendation). The optimal interval for blood pressure screening has not been determined and is left to clinical discretion. Current expert opinion is that adults who are believed to be normotensive should receive blood pressure measurements at least once every 2 years if their last diastolic and systolic blood pressure readings were below 85 and 140 mm Hg, respectively, and annually if the last diastolic blood pressure was 85–89 mm Hg.[1] Sphygmomanometry should be performed in accordance with recommended technique.[1] Hypertension should not be diagnosed on the basis of a single measurement; elevated readings should be confirmed on more than one reading at each of three separate visits. In adults, current blood pressure criteria for the diagnosis of hypertension are an average diastolic pressure of 90 mm Hg or greater and/or an average systolic pressure of 140 mm Hg or greater.[1] Once confirmed, patients should receive appropriate counseling regarding physical activity (Chapter 55), weight reduction and dietary sodium intake (Chapter 56), and alcohol consumption (Chapter 52). Evidence should also be sought for other cardiovascular risk factors, such as elevated serum cholesterol (Chapter 2) and smoking (Chapter 54), and appropriate intervention should be offered when indicated. The decision to begin drug therapy may include consideration of the level of blood pressure elevation, age, and the presence of other cardiovascular disease risk factors (e.g., tobacco use, hypercholesterolemia), concomitant disease (e.g., diabetes, obesity, peripheral vascular disease), or target-organ damage (e.g., left ventricular hypertrophy, elevated creatinine).[1,106,108] Antihypertensive drugs should be prescribed in accordance with recent guidelines[1,106,108] and with attention to current techniques for improving compliance.[53,54]

Measurement of blood pressure during office visits is also recommended for children and adolescents ("B" recommendation). This recommendation is based on the proven benefits from the early detection of treatable causes of secondary hypertension; there is insufficient evidence to recommend for or against routine periodic blood pressure measurement to detect essential (primary) hypertension in this age group. Sphygmomanometry should be performed in accordance with the recommended technique for children, and hypertension should only be diagnosed on the basis of readings at each of three separate visits.[18] In children, criteria defining hypertension vary with age.[18] Age-, sex-, and height-specific blood pressure nomograms for U.S. children and adolescents have been published.[20]

Routine counseling to promote physical activity (Chapter 55) and a healthy diet (Chapter 56) for the primary prevention of hypertension is recommended for all children and adults.

The draft update of this chapter was prepared for the U.S. Preventive Services Task Force by Carolyn DiGuiseppi, MD, MPH, based in part on material prepared for the Canadian Task Force on the Periodic Health Examination by Alexander G. Logan, MD, FRCPC, and Christopher Patterson, MD, FRCPC.

REFERENCES

1. Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. The fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. Bethesda: National Institutes of Health, 1993. (NIH Publication no. 93-1088.)

1a. Burt VL, Whelton P, Roccella EJ, et al. Prevalence of hypertension in the US adult population: results from the Third National Health and Nutrition Examination Survey, 1988–1991. Hypertension 1995;25:305–313.

2. National Center for Health Statistics. Annual summary of births, marriages, divorces, and deaths: United States, 1993. Monthly vital statistics report; vol 42 no 13. Hyattsville, MD: Public Health Service, 1994.

3. Dawber TF. The Framingham study: the epidemiology of atherosclerotic disease. Cambridge, MA: Harvard University Press, 1980.

4. Sagie A, Larson MG, Levy D. The natural history of borderline isolated systolic hypertension. N Engl J Med 1993;329:1912–1917.

5. 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. Multiple Risk Factor Intervention Trial Research Group. Arch Intern Med 1992;152:56–64.

6. Cheitlin MD. Coarctation of the aorta. Med Clin North Am 1977;61:655–673.

7. Hiner LB, Falkner B. Renovascular hypertension in children. Pediatr Clin North Am 1993;40:123–140.

8. Rocchini AP. Cardiovascular causes of systemic hypertension. Pediatr Clin North Am 1993;40:141–147.

9. Rodd CJ, Sockalosky JJ. Endocrine causes of hypertension in children. Pediatr Clin North Am 1993;40:149–164.

10. Report of the New England Regional Infant Cardiac Program. Pediatrics 1980;65(suppl):375–461.

11. Buyse ML, ed. Birth defects encyclopedia. St. Louis, MO: Blackwell Scientific Publications, 1990:156.
12. Mustard WT, Rowe RD, Keith JD, et al. Coarctation of the aorta, with special reference to the first year of life. Ann Surg 1955;141:429–436.
13. Tifft CP. Are the days of the sphygmomanometer past? Arch Intern Med 1988;148:518–519.
14. Pickering TG, James GD, Boddie C, et al. How common is white coat hypertension? JAMA 1988;259: 225–228.
15. Perloff D, Grim C, Flack J, et al. Human blood pressure determination by sphygmomanometry. Office of Scientific Affairs, American Heart Association. Circulation 1993;88:2460–2470.
16. Uhari M, Nuutinen M, Turtinen J, et al. Pulse sounds and measurement of diastolic blood pressure in children. Lancet 1991;338:159–161.
17. Nuutinen M, Turtinen J, Uhari M. Random-zero sphygmomanometer, Rose's tape, and the accuracy of the blood pressure measurements in children. Pediatr Res 1992;32:243–247.
18. Task Force on Blood Pressure Control in Children. Report of the Second Task Force on Blood Pressure Control in Children—1987. Pediatrics 1987;79:1–25.
19. Gomez-Marin O, Prineas RJ, Rastam L. Cuff bladder width and blood pressure measurement in children and adolescents. J Hypertens 1992;10:1235–1241.
20. Rosner B, Prineas RJ, Loggie JMH, et al. Blood pressure nomograms for children and adolescents, by height, sex, and age, in the United States. J Pediatr 1993;123:871–886.
21. Hoegholm A, Kristensen KS, Madsen NH, et al. White coat hypertension diagnosed by 24-h ambulatory monitoring: examination of 159 newly diagnosed hypertensive patients. Am J Hypertens 1992;5: 64–70.
22. James GD, Pickering TG, Yee LS, et al. The reproducibility of average ambulatory, home, and clinic pressures. Hypertension 1988;11:545–549.
23. Hall CL, Higgs CMB, Notarianni L, et al. Home blood pressure recording in mild hypertension: value of distinguishing sustained from clinic hypertension and effect on diagnosis and treatment. J Hum Hypertens 1990;4:501–507.
24. Bottini PB, Carr AA, Rhoades RB, et al. Variability of indirect methods used to determine blood pressure: office vs mean 24-hour automated blood pressures. Arch Intern Med 1992;152:139–144.
25. Pearce KA, Grimm RH Jr, Rao S, et al. Population-derived comparisons of ambulatory and office blood pressures: implications for the determination of usual blood pressure and the concept of white coat hypertension. Arch Intern Med 1992;152:750–756.
26. Kleinert HD, Harshfield GA, Pickering TG, et al. What is the value of home blood pressure measurement in patients with mild hypertension? Hypertension 1984;6:574–578.
27. Berenson GS, Dalferes E Jr, Savage D, et al. Ambulatory blood pressure measurements in children and young adults selected by high and low casual blood pressure levels and parental history of hypertension: the Bogalusa Heart Study. Am J Med Sci 1993;305:374–382.
28. Harshfield GA, Alpert BS, Pulliam DA, et al. Ambulatory blood pressure recordings in children and adolescents. Pediatrics 1994;94:180–184.
29. Hansson L. Current and future strategies in the treatment of hypertension. Am J Cardiol 1988;61: 2C–7C.
30. Collins R, Peto R, MacMahon S, et al. Blood pressure, stroke, and coronary heart disease. Part 2, short-term reductions in blood pressure: overview of randomised drug trials in their epidemiological context. Lancet 1990; 335:827–838.
31. MacMahon SW, Cutler JA, Furberg CD, et al. The effects of drug treatment for hypertension on morbidity and mortality from cardiovascular disease: a review of randomized, controlled trials. Prog Cardiovasc Dis 1986; 29(suppl):99–118.
32. Hebert PR, Moser M, Mayer J, et al. Recent evidence on drug therapy of mild to moderate hypertension and decreased risk of coronary heart disease. Arch Intern Med 1993;153:578–581.
33. Veterans Administration Cooperative Study Group on Antihypertensive Agents. Effects of treatment on morbidity in hypertension: results in patients with diastolic pressures averaging 115 through 129 mm Hg. JAMA 1967; 202:1028–1034.
34. Veterans Administration Cooperative Study Group on Antihypertensive Agents. Effects of treatment on morbidity in hypertension. II. Results in patients with diastolic pressures averaging 90 through 114 mm Hg. JAMA 1970; 213:1143–1152.
35. Veterans Administration Cooperative Study Group on Antihypertensive Agents. Effects of treatment on morbidity in hypertension. III. Influence of age, diastolic pressure, and prior cardiovascular disease: further analysis of side effects. Circulation 1972;45:991–1004.

36. Wolff FW, Lindeman RD. Effects of treatment in hypertension: results of a controlled study. J Chronic Dis 1966; 19:227–240.
37. Hypertension-Stroke Cooperative Study Group. Effect of antihypertensive treatment on stroke recurrence. JAMA 1974;229:409–418.
38. Smith WM. Treatment of mild hypertension: results of a ten-year intervention trial. Circ Res 1977;40(5 Suppl 1):I98–105.
39. Hypertension Detection and Follow-Up Program Cooperative Group. Five-year findings of the Hypertension Detection and Follow-Up Program. I. Reduction in mortality of persons with high blood pressure, including mild hypertension. JAMA 1979;242:2562–2572.
40. Management Committee of the Australian National Blood Pressure Study. The Australian therapeutic trial in mild hypertension. Lancet 1980;1:1261–1267.
41. Medical Research Council Working Party. MRC trial of treatment of mild hypertension: principal results. BMJ 1985;291:97–104.
42. Medical Research Council Working Party. Persistence of reduction in blood pressure and mortality of participants in the Hypertension Detection and Follow-Up Program. JAMA 1988;259:2113–2122.
43. Amery A, Birkenhager W, Brixko P, et al. Mortality and morbidity results from the European Working Party on High Blood Pressure in the Elderly Trial. Lancet 1985;i:1349–1354.
44. Amery A, Birkenhager W, Brixko P, et al. Efficacy of antihypertensive drug treatment according to age, sex, blood pressure and previous cardiovascular disease in patients over the age of 60. Lancet 1986;ii: 589–592.
45. Staessan J, Bulpitt C, Clement D, et al. Relation between mortality and treated blood pressure in elderly patients with hypertension: report of the European Working Party on High Blood Pressure in the Elderly. BMJ 1989; 298:1552–1556.
46. Dahlof B, Lindholm LH, Hansson L, et al. Morbidity and mortality in the Swedish Trial in Old Patients with Hypertension (STOP-Hypertension). Lancet 1991;338:1281–1285.
47. MRC Working Party. Medical Research Council trial of treatment of hypertension in older adults: principal results. BMJ 1992;304:405–412.
48. SHEP Cooperative Research Group. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension. Final results of the Systolic Hypertension in the Elderly Program (SHEP). JAMA 1991;265:3255–3264.
49. Insua JT, Sacks HS, Lau T-S, et al. Drug treatment of hypertension in the elderly: a meta-analysis. Ann Intern Med 1994;121:355–362.
50. Mulrow CD, Cornell JA, Herrera CR, et al. Hypertension in the elderly: implications and generalizability of randomized trials. JAMA 1994;272:1932–1938.
51. Garraway WM, Whisnant JP. The changing pattern of hypertension and the declining incidence of stroke. JAMA 1987;258:214–217.
52. Casper M, Wing S, Strogatz D, et al. Antihypertensive treatment and U.S. trends in stroke mortality, 1962 to 1980. Am J Public Health 1992;82:1600–1606.
53. McClellan WM, Hall WD, Brogan D, et al. Continuity of care in hypertension: an important correlate of blood pressure control among aware hypertensives. Arch Intern Med 1988;148:525–528.
54. National Institutes of Health. The physician's guide: improving adherence among hypertensive patients. Working Group on Health Education and High Blood Pressure Control. Bethesda: Department of Health and Human Services, 1987.
55. Siscovick DS, Raghunathan TE, Psaty BM, et al. Diuretic therapy for hypertension and the risk of primary cardiac arrest. N Engl J Med 1994;330:1852–1857.
55a. Neaton JD, Grimm RH Jr, Prineas RJ, et al. Treatment of mild hypertension study: final results. JAMA 1993;270:713–724.
56. National High Blood Pressure Education Program Working Group. National High Blood Pressure Education Program Working Group report on primary prevention of hypertension. Arch Intern Med 1993;153:186–208.
57. The Trials of Hypertension Prevention Collaborative Research Group. The effects of nonpharmacologic interventions on blood pressure of persons with high normal levels. Results of the Trials of Hypertension Prevention, Phase 1. JAMA 1992;267:1213–1220.
58. Langford HG, Davis BR, Blaufox MD, et al. Effect of drug and diet treatment of mild hypertension on diastolic blood pressure. Hypertension 1991;17:210–217.
59. Schotte DE, Stunkard AJ. The effects of weight reduction on blood pressure in 301 obese patients. Arch Intern Med 1990;150:1701–1704.

60. World Hypertension League. Physical exercise in the management of hypertension: a consensus statement by the World Hypertension League. J Hypertens 1991;9:283–287.
61. Cutler JA, Follmann D, Elliott P, et al. An overview of randomized trials of sodium reduction and blood pressure. Hypertension 1991;17(Suppl I):I27–I33.
62. Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? III. Analysis of data from trials of salt reduction. BMJ 1991;302:819–824.
62a. Blumenthal JA, Siegel WC, Appelbaum M. Failure of exercise to reduce blood pressure in patients with mild hypertension: results of a randomized controlled trial. JAMA 1991;266:2098–2104.
63. Wassertheil-Smoller S, Oberman A, Blaufox D, et al. The Trial of Antihypertensive Interventions and Management (TAIM) study. Final results with regard to blood pressure, cardiovascular risk, and quality of life. Am J Hypertens 1992;5:37–44.
64. Newman WP, Freedman DS, Voors AW, et al. Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis: the Bogalusa Heart Study. N Engl J Med 1986;314:138–144.
65. Schieken RM, Clarke WR, Lauer RM. Left ventricular hypertrophy in children with blood pressure in the upper quintile of the distribution. The Muscatine Study. Hypertension 1981;3:669–675.
66. Culpepper WS 3d, Sodt PC, Messerli FH, et al. Cardiac status in juvenile borderline hypertension. Ann Intern Med 1983;98:1–7.
67. Burke GL, Arcilla RA, Culpepper WS, et al. Blood pressure and echocardiographic measures in children: the Bogalusa Heart Study. Circulation 1987;75:106–114.
68. Prebis JW, Gruskin AB, Polinsky MS, et al. Uric acid in childhood essential hypertension. J Pediatr 1981;98:702–707.
69. Feld LG, Springate JE. Hypertension in children. Curr Probl Pediatr 1988;18:317–373.
70. Shear CL, Burke GL, Freedman DS, et al. Value of childhood blood pressure measurements and family history in predicting future blood pressure status: results from 8 years of follow-up in the Bogalusa Heart Study. Pediatrics 1986;77:862–869.
71. Michels VV, Bergstrahl EJ, Hoverman VR. Tracking and prediction of blood pressure in children. Mayo Clin Proc 1987;62:875–881.
72. Lauer RM, Clarke WR, Beaglehole R. Level, trend and variability of blood pressure during childhood. Circulation 1984;69:242–249.
73. Nelson MJ, Ragland DR, Syme SL. Longitudinal prediction of adult blood pressure from juvenile blood pressure levels. Am J Epidemiol 1991;136:633–645.
74. Kneisley J, Schork N, Julius S. Predictors of blood pressure and hypertension in Tecumseh, Michigan. Clin Exp Hypertens [A] 1990;12:693–708.
75. Gillman MW, Cook NR, Rosner B, et al. Identifying children at high risk for the development of essential hypertension. Pediatrics 1993;122:837–846.
76. St George IM, Williams SM, Silva PA. The stability of high blood pressure in Dunedin children: an eight year longitudinal study. NZ Med J 1990;103:115–117.
77. Nishio T, Mori C, Watanabe K, et al. Quantitative analysis of systolic blood pressure tracking during childhood and adolescence using a tracking index: the Shimane Heart Study. J Hypertens 1989; 7 (Suppl 1):S35–S36.
78. Yong L-C, Kuller LH. Tracking of blood pressure from adolescence to middle age: the Dormont High School Study. Prev Med 1994;23:418–426.
79. Hofman A, Hazebroek A, Valkenburg HA. A randomized trial of sodium intake and blood pressure in newborn infants. JAMA 1983;250:370–373.
80. Bal' LV, Shugaeva EN, Deev AA, et al. Results of a three-year trial of arterial hypertension prevention in a population of children aged 11–15 years by overweight control. Cor Vasa 1990;32:448–456.
81. Hansen HS, Froberg K, Hyldebrandt N, et al. A controlled study of eight months of physical training and reduction of blood pressure in children: the Odense schoolchild study. BMJ 1991;303:682–685.
82. Sinaiko AR, Gomez-Marin O, Prineas RJ. Effect of low sodium diet or potassium supplementation on adolescent blood pressure. Hypertension 1993;21:989–994.
83. Williams GH, Braunwald E. Hypertensive vascular disease. In: Braunwald E, et al, eds. Harrison's principles of internal medicine. New York: McGraw-Hill, 1987.
84. De Santo NG, Capasso G, Giordano DR, et al. Secondary forms of hypertension. Semin Nephrol 1989; 9:272–286.
85. Strafford MA, Griffiths SP, Gersony WM. Coarctation of the aorta: a study in delayed detection. Pediatrics 1982;69:159–163.

86. Thoele DG, Muster AJ, Paul MH. Recognition of coarctation of the aorta. A continuing challenge for the primary care physician. Am J Dis Child 1987;141:1201–1204.
87. Ward KE, Pryor RW, Matson JR, et al. Delayed detection of coarctation in infancy: implications for timing of newborn follow-up. Pediatrics 1990;86:972–976.
88. Westaby S, Parnell B, Pridie RB. Coarctation of the aorta in adults. Clinical presentation and results of surgery. J Cardiovasc Surg 1987;28:124–127.
89. Nanton MA, Olley RM. Residual hypertension after coarctectomy in children. Am J Cardiol 1976;37: 769–772.
90. Presbitero P, Demarie D, Villani M, et al. Long term results (15–30 years) of surgical repair of aortic coarctation. Br Heart J 1987;57:462–467.
91. Shinebourne EA, Tam ASY, Elseed AM, et al. Coarctation of the aorta in infancy and childhood. Br Heart J 1976;38:375–380.
92. Liberthson RR, Pennington DG, Jacobs ML, et al. Coarctation of the aorta: review of 234 patients and clarification of management problems. Am J Cardiol 1979;43:835–840.
93. Lawrie GM, DeBakey ME, Morris GC Jr, et al. Late repair of coarctation of the descending thoracic aorta in 190 patients. Arch Surg 1981;116:1557–1560.
94. Forfang K, Rostad H, Sorland S. Coarctation of the aorta. Follow-up of 218 patients operated on after 13 years of age. Acta Med Scand 1981;645:15–22.
95. Bobby JJ, Emami JM, Farmer RDT, et al. Operative survival and 40 years follow-up of surgical repair of aortic coarctation. Br Heart J 1991;65:271–276.
96. Grundy SM, Greenland P, Herd A, et al. Cardiovascular and risk factor evaluation of healthy American adults. A statement for physicians by an ad hoc committee appointed by the Steering Committee, American Heart Association. Circulation 1987;75:1340A–1362A.
97. Eddy D, ed. Common screening tests. Philadelphia: American College of Physicians, 1991.
98. American Academy of Family Physicians. Age charts for periodic health examination. Kansas City, MO: American Academy of Family Physicians, 1994. (Reprint no. 510.)
99. Canadian Task Force on the Periodic Health Examination. Canadian guide to clinical preventive health care. Ottawa: Canada Communication Group, 1994:636–648, 944–951.
100. American Academy of Pediatrics. Recommendations for preventive pediatric health care. Pediatrics 1995;96:373–374.
101. Green M, ed. Bright Futures: guidelines for health supervision of infants, children and adolescents. Arlington, VA: National Center for Education in Maternal and Child Health, 1994.
102. American Medical Association. AMA guidelines for adolescent preventive services (GAPS): recommendations and rationale. Chicago: American Medical Association, 1994.
103. American Heart Association. Integrated cardiovascular health promotion in childhood. A statement for health professionals from the Subcommittee on Atherosclerosis and Hypertension in Childhood of the Council on Cardiovascular Disease in the Young. Circulation 1992;85:1638–1650.
104. American Academy of Pediatrics. Routine evaluation of blood pressure, hematocrit, and glucose in newborns. Pediatrics 1993;92:474–476.
105. MacDonald LA, Sackett DL, Haynes RB, et al. Labelling in hypertension: a review of the behavioral and psychological consequences. J Chronic Dis 1984;37:933–942.
106. Haynes RB, Lacourciere Y, Rabkin SW, et al. Report of the Canadian Hypertension Society Consensus Conference. 2. Diagnosis of hypertension in adults. Can Med Assoc J 1993;149:409–418.
107. Ogilvie RI, Burgess ED, Cusson JR, et al. Report of the Canadian Hypertension Society Consensus Conference. 3. Pharmacologic treatment of essential hypertension. Can Med Assoc J 1993;149: 575–584.
108. Sever P, Beevers G, Bulpitt C, et al. Management guidelines in essential hypertension: report of the second working party of the British Hypertension Society. BMJ 1993;306:983–987.
109. Whitworth JA, Clarkson D, Dwyer T, et al. The management of hypertension: a consensus statement. Australian Consensus Conference 1993. Med J Aust 1994;160(suppl):S1–S16.
110. WHO / ISH Mild Hypertension Liaison Committee. 1993 guidelines for the management of mild hypertension. Memorandum from a World Health Organization / International Society of Hypertension meeting. Hypertension 1993;22:392–403.
111. Jackson R, Barham P, Bills J, et al. Management of raised blood pressure in New Zealand: a discussion document. BMJ 1993;307:107–110.