

# Biobehavioral Approaches to the Treatment of Essential Hypertension

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Despite recent advances in the medical management of hypertension, chronically elevated blood pressure remains a major health problem in the United States, affecting almost 50 million Americans. It is widely recognized that lifestyle factors contribute to the development and maintenance of elevated blood pressure. This article critically reviews current approaches to the nonpharmacological treatment of high blood pressure and highlights outcome studies of exercise, weight loss and dietary modification, and stress management and relaxation therapies. Methodological issues in the assessment and treatment of hypertension are discussed, along with possible mechanisms by which lifestyle modification may reduce elevated blood pressure.

Hypertension (HTN) affects almost 50 million people in the United States (Burt et al., 1995; Joint National Committee, 1997), placing them at increased risk of stroke, myocardial infarction, congestive heart failure, kidney failure, and peripheral vascular disease. In addition, subtle cognitive impairments, which may be reversible with treatment, also have been shown to be associated with HTN (Blumenthal, Madden, Pierce, Siegel, & Appelbaum, 1993; Waldstein, Manuck, Ryan, & Muldoon, 1991).

HTN is defined as having systolic blood pressure (SBP) of 140 mm Hg or greater, having diastolic blood pressure (DBP) of 90 mm Hg or greater, or taking antihypertensive medication. Blood pressure (BP) in excess of 140/90 mm Hg is further categorized in terms of severity of HTN, defined as Stage 1 (140–159/90–99 mm Hg), Stage 2 (160–179/100–109 mm Hg), or Stage 3 ( $\geq$  180/110-mm Hg). Although BP less than 140/90-mm Hg was once considered normal, the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (Joint National Committee, 1997) now includes the category of “high normal,” defined as the pressure range 130–139/85–89 mm Hg. Only pressures below 120/80 mm Hg are considered optimal.

The prevalence of HTN is related to age, gender, and ethnicity. In both men and women, prevalence generally increases markedly across the life span. Although the prevalence in young adults is much greater in men than women, this difference progressively diminishes with age, ultimately reversing at around 50 years to a greater prevalence in women than men (Burt et al., 1995). For women, the more dramatic rise in BP associated with age is linked

to the occurrence of menopause (Staessen, Bulpitt, Fagard, Lijnen, & Amery, 1989). HTN is also more prevalent among African Americans than Americans of European heritage, especially in the southeastern United States (Hall et al., 1997).

Although HTN is defined by elevation of arterial BP, its clinical significance is derived from morbid events affecting the heart, brain, and kidneys. Complications such as myocardial infarction and stroke are not due directly to elevated BP but to the resulting structural changes in the heart and blood vessels. One of the structural consequences of HTN, left ventricular hypertrophy (LVH), is the strongest known predictor, other than advancing age, of cardiovascular morbidity and mortality. LVH predicts these clinical outcomes both in individuals with HTN (Casale et al., 1986) and in healthy individuals (Levy, Garrison, Savage, Kannel, & Castelli, 1990) independent of other conventional risk factors. Cardiovascular risk associated with LVH appears to be greater in women than men (Kannel & Wilson, 1995). The prevalence of LVH also is greater in African Americans than in their White counterparts, even controlling for the differences in HTN (Taylor, Borhani, Entwistle, Farber, & Hawkins, 1983).

Two decades ago, D. Shapiro and Goldstein (1982) provided a thorough review of psychosocial factors and HTN and noted that lifestyle factors were linked to the development of HTN and were targets for biobehavioral interventions. The primary biobehavioral approaches for the treatment of HTN include exercise, dietary modification such as salt restriction and caloric reduction to achieve weight loss, biofeedback, and stress management. The purpose of this review is to update the empirical evidence gathered since 1982 that nonpharmacological approaches reduce BP among individuals with HTN, with an emphasis on studies published since 1990, and to discuss possible mechanisms for such an effect. In addition, several important methodological issues are discussed, along with a brief overview of pharmacological approaches to HTN treatment, to place the nonpharmacological therapies in the context of the current medical management of HTN.

## Methodological Issues

Because of significant variability among BP measurements in a given individual, it is generally recommended that BP be measured

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two or more times on at least three separate occasions while the patient is seated in a quiet room and that an average reading be used. The auscultatory method, using a stethoscope and mercury sphygmomanometer, is still considered the methodology of choice for clinic BP measurement (Joint National Committee, 1997). The random-zero sphygmomanometer remains a standard in clinical research as it provides a means of eliminating the observer bias common in the measurement of BP using the auscultatory method. Automated BP-monitoring devices also eliminate observer bias and have become a popular instrument in clinical research. However, it has been recommended that studies using automated electronic BP monitors should provide evidence of the validity and reliability of the specific device used and that such devices should also be calibrated regularly against the mercury sphygmomanometer (D. Shapiro et al., 1996).

One critical methodological advance in the area has been the increased use of ambulatory blood pressure (ABP) monitoring. ABP technology has now been available for over 20 years, and its use in the clinical research setting to examine BP variations over the 24-hr period of normal daily routines has generated a number of important findings. The most well-established diagnostic aspect of ABP is that it is superior to clinic BP as a predictor of target-organ damage in HTN. A number of cross-sectional studies have documented that LVH is more closely related to ABP than to clinic BP (Devereux, Savage, Sachs, & Laragh, 1983; Verdecchia et al., 1990; White, Lund-Johansen, McCabe, & Omvik, 1989). Several studies have shown that the higher pressures of the 24-hr diurnal rhythm, associated with daytime work activity and stress, are notably good predictors of left ventricular mass (LVM; Devereux, Pickering, et al., 1983; Lemne, Lindvall, Georgiades, Fredrikson, & de Faire, 1995; Palatini et al., 1985). Nighttime sleep pressures are also related to LVM, and some ABP studies have indicated that sleep ABP shows the strongest association with LVM (Verdecchia et al., 1990).

The prognostic value of ABP also has been evaluated. There is evidence that ABP is a superior predictor of cardiovascular events, including myocardial infarction, cerebrovascular events, and mortality, than is clinic BP (Ohkubo et al., 1997; Palatini et al., 1992; Staessen et al., 1999; Verdecchia et al., 1994). One reason for the superior prognostic validity of ABP is that it transcends the problem of "white coat" HTN, in which BP is abnormally elevated in the clinic environment but otherwise tends to be normal during typical daily activities (Mallion, Baguet, Siche, Tremel, & De Gaudemaris, 1999; Pickering, Coats, Mallion, Mancia, & Verdecchia, 1999). The importance of 24-hr ABP monitoring is underscored by evidence that nighttime BP may be the best predictor of cardiovascular morbidity and mortality (Staessen et al., 1999). Therefore, the inclusion of ABP monitoring is highly recommended for studies designed to evaluate intervention effects on BP.

Most recently, studies have examined the significance of BP measured in the laboratory during mental stress. Mental stress BP may have prognostic significance over and above clinic BP, as it has been shown that hypertensive patients who exhibit elevated BP responses are more likely to develop LVH compared with patients who do not exhibit large BP responses (Kop, Gottdiner, Patterson, & Krantz, 2000; Sherwood et al., 2002) and patients who exhibit elevated BP in the laboratory also are more likely to exhibit myocardial ischemia during mental stress testing and during daily

life (Blumenthal et al., 1995). Mental-stress testing may therefore be considered a novel and potentially useful procedure for assessing BP in studies evaluating the effectiveness of interventions in patients with HTN.

### Pharmacotherapy

The development of effective drug therapies for HTN has grown exponentially over the past 2 decades, providing the modern clinician with an extensive arsenal of treatment options. The most commonly prescribed HTN drugs belong to one of four major classes: diuretics, beta-adrenergic blockers, calcium antagonists, and angiotensin-converting enzyme (ACE) inhibitors. A vast body of literature exists demonstrating that pharmacological therapy of mild to moderate HTN can significantly decrease the incidence of stroke, coronary artery disease, and overall cardiovascular mortality. Collins et al. (1990) conducted a meta-analysis of 14 randomized controlled trials of antihypertensive drug treatment in both community and hospital settings. Of the 37,000 patients included, drug therapy, in which diuretics and beta-blockers were the first-line medicines used, resulted in a mean reduction in DBP in treated patients relative to controls of 5–6 mm Hg. This reduction in average DBP was associated with reductions of 42% in stroke, 14% in coronary artery disease, and 21% in vascular mortality.

The Joint National Committee (1997) has recommended initiating drug therapy after failure of a 6-month lifestyle modification regimen or in more severe situations where the patient requires immediate drug treatment. The goal for therapy is to lower BP to less than 140/90-mm Hg. In individuals with uncomplicated HTN, diuretics and beta-adrenergic blockers are recommended as first-line pharmacological agents, a conclusion based on randomized, placebo-controlled trials that have demonstrated improved health outcomes with these medicines (Medical Research Council Working Party, 1992; Wilhelmsen et al., 1987).

Newer antihypertensive drugs, such as calcium channel blockers and ACE inhibitors, have been shown to be effective in reducing BP and are well-tolerated when used as monotherapy (Materson et al., 1993). However, their potential benefits in preventing cardiovascular morbidity and mortality are still being evaluated in randomized, controlled clinical trials (Davis et al., 1996). Therefore, current clinical evidence favors the use of diuretics and beta-blockers (Hennekens, 1998).

In some instances, adequate BP control is not achieved by reasonable dosages of the first drug. There are a variety of reasons for this situation, including failure to follow the prescribed therapy, secondary HTN, and drug interactions. Individual differences can also impact BP response to drug treatment. In several randomized, controlled studies, African American patients were less responsive to monotherapy with beta-blockers than with diuretics (Veterans Administration Cooperative Study Group, 1982; Wassertheil-Smoller, Oberman, Blaufox, Davis, & Langford, 1992). Moreover, beta-blocker therapy was less effective in African Americans compared with Whites, whereas no ethnic differences were observed in response to diuretic treatment. The conclusion from these studies is that diuretics should be the agents of choice among African Americans in the absence of conditions prohibiting their usage.

If BP is not reduced or if bothersome side effects develop, an agent from another class is given in place of the initial drug. In

situations where the first drug produces a partial response without major side effects, a second agent from another class is added. For the majority of patients, adherence to such a pharmacological regimen results in adequate HTN management.

### *Importance of Adherence*

Despite the proven benefits of drug therapy for HTN, however, control of BP remains challenging. Results of the Third National Health and Nutrition Examination Survey (Burt et al., 1995) indicate that only one quarter of Americans with HTN have their BP "controlled," defined as a level below 140/90 mm Hg. These suboptimal results are largely a result of poor medication adherence, which is a consequence of numerous factors including adverse side effects, expense, changes in clinical providers, and a lack of sufficient understanding of the risks associated with untreated HTN. Moreover, in general, the more complex the medication regimen, the poorer the compliance. Thus, regimen simplification, when possible, is an obvious strategy for improving adherence. Once-daily dosing, for example, has been found to be associated with better compliance than more frequent dosing (e.g., Leenen et al., 1997).

Attempts to identify individual predictors of adherence, however, have produced mixed results. In addition, research on the effectiveness of behavioral interventions to improve medication adherence has been limited. What is known is based primarily on research in primary care settings. Simple educational materials, for example, have generally been shown to be ineffective in improving adherence to medication (e.g., Binstock & Franklin, 1988; Kirscht, Kirscht, & Rosenstock, 1981). In contrast, a number of strategies have been shown to significantly improve adherence to medication. These include social support; family member education and support of the patient; home BP monitoring; behavioral contracting; use of pill packs; and telephone check-in calls to identify problems, provide ongoing education, and reinforce compliance (Binstock & Franklin, 1988; Kirscht et al., 1981; Morisky, De Muth, Field-Fass, Green, & Levine, 1985). In turn, improved medication adherence has been found to be related to decreased BP (e.g., Binstock & Franklin, 1988; Morisky et al., 1985). However, the feasibility of such interventions on a large scale remains to be proven.

Adherence aside, it is important to recognize that pharmacological treatments do not preclude the use of lifestyle modification to lower BP. Indeed, there is evidence that behavioral interventions may reduce or eliminate the need for drug therapy in some patients (Glasgow, Engel, & D'Lugoff, 1989). Shapiro et al. (D. Shapiro, Hui, Oakley, Pasic, & Jamner, 1997) demonstrated that the addition of a cognitive-behavioral intervention to the standard drug treatment for Stage 1-2 HTN was highly effective in lowering BP and in reducing the required dosage of medications to adequately control BP.

### *Summary*

Reducing BP by drug therapy results in a demonstrable reduction in cardiovascular morbidity and mortality. However, maintaining patients on treatment and effectively reducing their BP has proved difficult in clinical practice. Although diuretics and beta-blockers remain the first-line agents for hypertension drug therapy,

randomized trials currently in progress are studying cardiovascular disease outcomes associated with BP reduction using newer classes of antihypertensive agents. Regardless of the effectiveness of drug therapies for HTN, lifestyle interventions are an important initial strategy for lowering BP in most patients with HTN, and also may reduce the amount of medication required to successfully lower BP. A stepped care approach integrating pharmacological and behavioral-lifestyle modifications is presented in Figure 1.

### *Exercise Therapy*

Physical activity and aerobic exercise have received much attention and been the subject of numerous reviews (American College of Sports Medicine, 1994; Arroll & Beaglehole, 1992; Halbert et al., 1997; Kelley, 1999; Kelley & McClellan, 1994; Linden & Chambers, 1994; Siegel & Blumenthal, 1991; Tipton, 1991). A variety of observational studies have demonstrated a significant association between higher levels of fitness and lower BP. Several large longitudinal studies have demonstrated an inverse relation between physical fitness and BP, including studies of University of Pennsylvania alumni (Paffenbarger, Thorne, & Wing, 1968), Harvard alumni (Lee, Hsieh, & Paffenbarger, 1995; Paffenbarger, Wing, Hyde, & Jung, 1983), and self-referrals to a preventive medicine clinic (Blair, Goodyear, Gibbons, & Cooper, 1984).

A large number of interventional studies also have been published in which individuals with normal BP or HTN have BP measurements obtained before and after an exercise program. However, the majority of these studies did not have an adequate control group for comparison. Although most of these researchers observed a drop in BP, it is evident from controlled studies that BP frequently has shown significant decreases in nonexercising control groups because of such factors as regression to the mean and habituation to BP measurement after multiple measurements have been made. Thus, uncontrolled studies are difficult to interpret, and they do not provide adequate scientific evidence for the value of exercise.

Other studies of exercise interventions have used nonrandom controls (Seals & Reiling, 1991; Tanaka et al., 1997). In one study (Seals & Reiling, 1991) 34 men and women, 24 of whom completed at least 6 months of exercise training, underwent BP measurement at rest and during 24 hr of ABP monitoring. Participants were not randomly assigned to exercise or control conditions but rather were allowed to choose between training and maintained physical activity. After 6 months, exercisers achieved 3-4 mm Hg clinic BP reductions, which were not different from controls. Similarly, changes in ABP were small and not statistically significant. Other studies also have shown relatively small BP reductions using ABP monitoring (Fortmann, Haskell, & Wood, 1988; Gilders, Voner, & Dudley, 1989; Van Hoof et al., 1989).

Several studies of exercise interventions have used subjects who serve as their own controls. In one design, investigators observe subjects for a period of time before the exercise intervention, with the initial observation period serving as the control (Ketelhut, Franz, & Scholze, 1997; Kiyonaga, Arakawa, Tanaka, & Shindo, 1985; Seals, Silverman, Reiling, & Davy, 1997). Using this approach, Ketelhut et al. (1997) studied 10 male patients with HTN who exercised for 18 months and exhibited a decrease in resting BP from 139/96 to 133/91 mm Hg. In another study design,

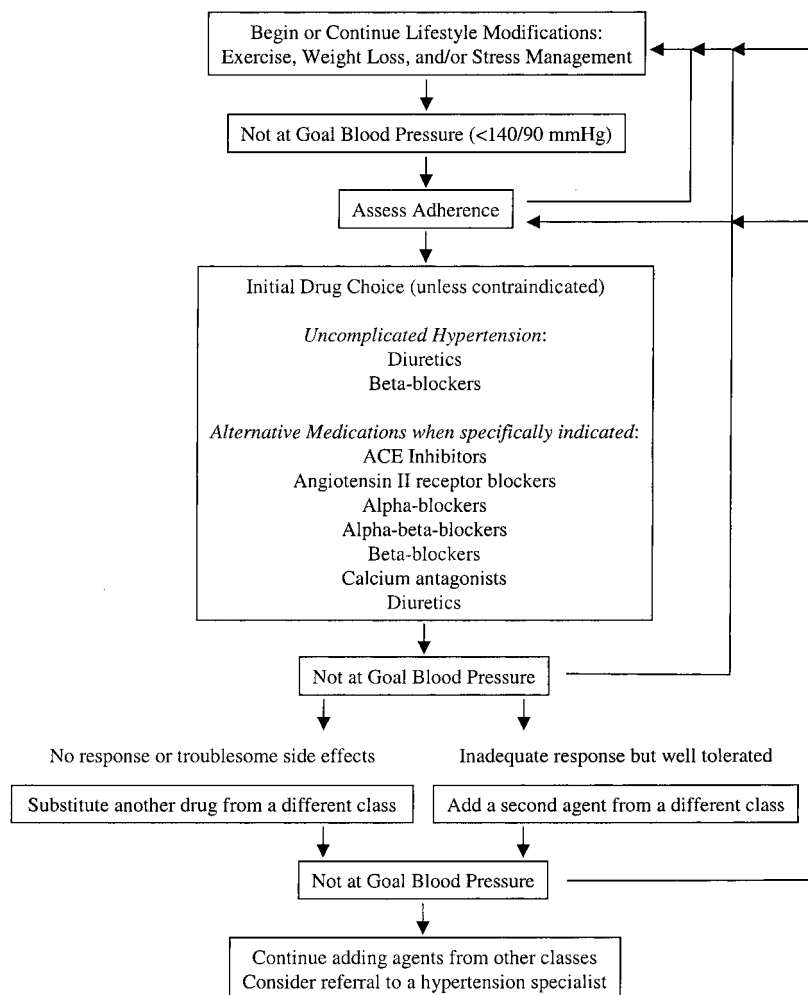


Figure 1. Algorithm for the treatment of hypertension. ACE = angiotension-converting enzyme.

initially sedentary subjects go through an exercise program and then return to a sedentary lifestyle (Roman, Camuzzi, Villalon, & Klenner, 1981; Somers, Conway, Johnston, & Sleight, 1991). For example, Roman et al. (1981) reported that 27 women with HTN underwent 3 months of exercise, 3 months of sedentary behavior, and then another 3 months of exercise. BPs were 182/113 mm at baseline, decreased to 161/97-mm after the first exercise period, increased to 179/113 mm after the sedentary period, and then fell again to 159/95 mm after the second exercise period. No data were presented about weight or dietary changes, however.

Several studies have used a Latin square design (Marceau, Kouame, Lacourciere, & Cleroux, 1993; Nelson, Jennings, Esler, & Korner, 1986). Marceau et al. (1993) assigned 11 patients with HTN to each of three 10-week interventions: sedentary behavior, bicycling 3 days per week at low intensity, and bicycling 3 days per week at moderate intensity. No changes occurred in weight or urinary sodium excretion. Both training intensities produced comparable 5-mm Hg reductions in SBP and DBP, although the low-intensity training appeared to reduce BP during waking hours, whereas the moderate-intensity training reduced BP during the evening and sleep (and also was associated with greater weight

loss). However, there were no BP differences during 60 min of supine rest, during 10 min of rest sitting, or during 2 min of submaximal exercise compared with the sedentary condition. Taken together, these studies provided suggestive evidence that aerobic-exercise training may result in significant reductions in BP. However, these studies only included participants who completed the protocol, often failed to account for important confounders such as weight loss, and demonstrated that BP will frequently fall in control groups, emphasizing a need for randomized controls.

#### Randomized Controlled Trials

Since 1990, there have been seven randomized controlled trials of exercise training in adults with HTN (see Table 1). Additional randomized trials were not included because results were uninterpretable because of major methodological limitations such as failure to control for concurrent medication use (e.g., Kokkinos et al., 1995) or uncertainty about BP status at the time of study enrollment (e.g., Cononie et al., 1991).

In a well-controlled study of 27 hypertensive men (J. E. Martin, Dubbert, & Cushman, 1990), exercisers exhibited a BP reduction

Table 1  
Summary of Randomized Controlled Trials of Exercise in Hypertensive Patients Since 1990

Authors	Intervention	Duration	N	Pretx	Posttx	Change in
				SBP/DBP (mm Hg)	SBP/DBP (mm Hg)	SBP/DBP (mm Hg)
Martin, Dubbert, & Cushman, 1990	Four 30-min sessions per week of walking, jogging, or cycling at 60%–80% max.	10 weeks	27 (EX: 10)	136.6/94.8 (EX) 134.9/93.7 (control)	130.1/85.2 (EX) 135.8/94.4 (control)	–6.4/–9.6 <sup>a</sup>
Blumenthal, Siegel, & Appelbaum, 1991	Aerobic exercise: three 35-min sessions per week of walking, jogging, or cycling at > 70% max.; strength training: two to three 30-min sessions per week of circuit weight training	16 weeks	99 (aerobic: 39) (strength: 31)	142/95	133/89 (aerobic) 136/89 (strength)	–8/–6 (aerobic) –7/–6 (strength)
Gordon, Scott, & Levine, 1997	Exercise: three to five 30–45-min sessions per week of walking at 60%–85% max.; diet: counseling to achieve loss of 10% body weight; goal of ≤ 20% calories from fat and ≤ 2,300 mg sodium	12 weeks	55 (EX: 14) (diet: 15) (EX + diet: 19)	145/96 (EX) 141/93 (diet) 145/95 (diet + EX)	—	–9.9/–5.9 (EX) –11.3/–7.5 (diet) –12.5/–7.9 (EX + diet)
Moreira, Fuchs, Ribeiro, & Appel, 1999	Three 30-min sessions per week of cycle ergometry at either 20% (I) or 60% (II) max.	10 weeks	28	I. clinic: 155.9/98.4 ABP: 137.2/92.1 II. clinic: 153.5/96.9 ABP: 144.4/ 93.3	I. clinic: — ABP: 135.2/89.3 II. clinic: — ABP: 138.6/ 90.6	—
Young, Appel, Jee, & Miller, 1999	Four 30-min sessions per week of walking and aerobic dance at 40%–60% heart rate reserve	12 weeks	62	138.2/75.4 (aerobic) 141.7/76.6 (control)	—	–8.4/–3.2
Cooper, Moore, McKenna, & Ridloch, 2000	Five 30-min sessions per week of brisk walking	6 weeks	90 (EX: 48)	Clinic: 155.5/ 97.1 (EX) 155.4/98.2 (control) ABP: 139.8/ 89.5 (EX) 135.7/87.6 (control)	Clinic: — ABP: 137.0/ 87.7 (EX)	Clinic: — ABP: –2.8/ –1.9
Blumenthal et al., 2000	Exercise: three to four 35-min sessions per week of cycle ergometry, walking, and jogging at 70%–85% heart rate reserve; weight management (WM): exercise + behavioral weight loss program	26 weeks	133 (EX: 54) (WM: 55)	Clinic: 141.0/93.6 ABP: 142.2/ 87.6	Clinic: 133.7/ 89.3 (EX) 135.2/87.6 <sup>b</sup> (WM) ABP: 142.4/ 87.4 (EX) 140.0/83.7 <sup>b,c</sup> (WM)	Clinic: –4.4/ –4.3 (EX) –7.4/–5.6 (WM) ABP: +0.8/ –0.6 (EX) –2.6/–3.0 (WM)

Note. Pretx = pretreatment; SBP = systolic blood pressure; DBP = diastolic blood pressure; Posttx = posttreatment; max. = maximum; EX = exercise group; ABP = ambulatory blood pressure. Dash indicates *not applicable*.

<sup>a</sup> EX significantly lower than control (DBP not SBP). <sup>b</sup> EX and WM significantly lower than control (SBP and DBP). <sup>c</sup> WM significantly lower than EX (for DBP).

from 137/95 to 130/85 mm Hg after 10 weeks, whereas the control group showed no change (135/94- to 136/94-mm Hg). The changes in DBP, but not in SBP, were significantly different. However, both groups showed comparable changes in body weight and aerobic fitness. Gordon and colleagues (N. F. Gordon, Scott, & Levine, 1997) randomized 55 sedentary, overweight patients with high-normal BP or Stage 1 or 2 HTN to exercise only, diet only

(calorie reduction), or both exercise and diet for 12 weeks. Clinic BP reductions in the combination group (12.5/7.9-mm Hg) were larger than in diet only (11.3/7.5 mm Hg) and in exercise alone (9.9/5.9 mm Hg). Because results failed to reach statistical significance, the authors concluded that the BP lowering effects of exercise and weight loss were not additive. In the absence of a true no-treatment control condition, however, the BP changes are dif-

difficult to interpret. The absence of a no-treatment control condition also characterized several other recent studies. Young, Appel, Jee, and Miller (1999) randomized 62 older adults (age 60–80 years) with high-normal or Stage 1 HTN to either a 12-week moderate aerobic exercise program or a T'ai Chi light-activity control condition. There were no group BP differences after 12 weeks of training. However, the findings are difficult to interpret in the absence of a no-treatment control group because the T'ai Chi control group may have had elements (e.g., relaxation) that had BP-lowering effects. In addition, the aerobic exercise group failed to achieve significant improvements in maximal aerobic capacity, and data analysis failed to adjust for potential baseline group differences. The BP changes also were greatest during the initial 6 weeks of treatment, raising a question about the stability of the BP measurements and possible regression to the mean. Moreira, Fuchs, Ribeiro, and Appel (1999) randomized 28 sedentary hypertensive patients to 10 weeks of aerobic exercise at either 20% or 60% of their maximal workload. Mean 24-hr ABPs fell to a comparable degree in both groups, and the reductions were due to changes in daytime, not evening, BPs. As the authors noted, results could be interpreted as demonstrating either that both intensities have an antihypertensive effect or that neither had an antihypertensive effect.

Cooper, Moore, McKenna, and Riddoch (2000) randomized 90 patients with BPs 150–180/91–110 mm Hg to an aerobic exercise program or to a nonexercise control condition for 6 weeks. Exercise participants were asked to expend 150–200 kcal per day (equivalent of 30 min of brisk walking) at least 5 days per week. Although exercisers showed significant reductions in both systolic and diastolic ABP and controls showed small, nonsignificant increases, differences between exercisers and controls were not significant.

Two studies from Duke also provided a mixed picture. In an initial study (Blumenthal, Siegel, & Appelbaum, 1991), 99 patients with mild HTN were randomly assigned to 4 months of aerobic exercise, strength training, or a waiting list control. After treatment, all groups achieved comparable 5–10-mm reductions in BP. In a second study (Blumenthal et al., 2000), patients were randomly assigned to 6 months of aerobic exercise, to an aerobic exercise plus a behavioral weight-loss program, or to a waiting list control group. A 4-mm reduction in resting clinic SBP and DBP was observed in the exercise-only condition compared with 7 mm Hg for SBP and 5 mm Hg for DBP in the weight-management condition. Larger BP reductions also were observed for the weight-management condition relative to exercise only with ABP monitoring during routine activities of daily living, particularly for DBP. Additionally, BP levels were lower for both intervention groups relative to controls during mental stress and submaximal exercise testing. Weight management also was associated with larger DBP reductions than was exercise only during mental stress and submaximal exercise.

The reasons for the discrepancy in the effects of exercise on BP in the two Duke studies may be attributed to important methodological differences, including different patient characteristics (< 20% above ideal body weight vs. 10%–50% above ideal body weight) and a more extended exercise program (three times per week for 4 months vs. four times per week for 6 months). Most significantly, although the within-group BP changes were comparable among the exercisers in the two studies, the waiting list

control group in the initial study exhibited a significant BP reduction after 4 months, whereas the BP for the waiting list control group in the more recent study remained unchanged. This difference could be attributed to improved reliability of clinic BP measurement in the 1999 study, which incorporated a more rigorous BP-screening protocol.

### Summary

Despite claims by a number of highly respected organizations such as the American College of Sports Medicine (1994) that exercise will elicit a 10-mm Hg reduction in both SBP and DBP, results from well-controlled studies offer a more cautious appraisal. Results from randomized trials have been inconsistent, and certainly the magnitude of effects has been far more modest. Moreover, most of the studies have been limited because of high drop-out rates, unplanned crossover, imprecise measurement of BP or aerobic fitness, or failure to precisely measure other potential confounders (e.g., body weight), and few studies included adequate numbers of women. Better designed studies with greater methodological rigor generally demonstrated smaller exercise-related BP reductions than studies with less rigorous controls. A recent meta-analysis suggested that a 2% reduction in resting SBP and a 1% reduction in DBP were more likely (Kelley, 1999). Furthermore, findings indicate that although mild–moderate exercise by itself is generally not associated with significant weight loss, the addition of a behavioral weight-loss program to an exercise intervention may result in even greater BP reductions than the reductions observed with exercise alone.

### Weight Loss and Dietary Modification

The role of diet in HTN has become an increasingly complex area of study, as recent research has implicated a number of individual nutrients, in addition to obesity, in the development of HTN. The Joint National Committee (1997) made several dietary recommendations to prevent and manage HTN, including losing weight if overweight, limiting alcohol consumption, reducing sodium intake, and maintaining an adequate intake of dietary potassium. These suggestions were based on a growing body of both observational data, indicating a relationship between dietary factors and BP, and interventional research, documenting the efficacy of dietary modification in reducing BP.

#### Weight Loss

The association between obesity and BP is well-established, both cross-sectionally and longitudinally (see review by Jeffery, 1991). Moreover, numerous interventional studies have examined the effect of weight loss on BP. However, not all such studies were designed to compare weight loss alone with a usual diet–usual lifestyle control condition, making it difficult to clarify the specific effect of weight loss on BP in a number of studies. Instead, some interventions target multiple dietary components and/or multiple lifestyle components (e.g., Pérez-Stable et al., 1995); compare weight loss with another intervention, such as exercise (e.g., N. F. Gordon et al., 1997); include a dietary intervention for all groups (e.g., Neaton et al., 1993); or allow for changes in antihypertensive medication use. For example, Langford et al. (1991), in the Trial of

Antihypertensive Interventions and Management, examined the DBP response of nine combinations of drugs and diets and included a placebo-weight loss group and a placebo-usual diet group. However, in this study, 20% of placebo-usual diet participants were on step-up or open-label medication therapy by the end of study. Thus, even though the weight loss group exhibited a BP reduction of  $-11.5/-8.8$  mm Hg, this was not significantly different than the control group, which not surprisingly displayed a decrease in BP as well.

By and large, however, results of such studies have shown significant reductions in BP resulting from weight loss in hypertensive individuals (see reviews by Mulrow et al., 2000; Staessen, Fagard, & Amery, 1988). Moreover, studies such as the Trials of Hypertension Prevention (Stevens et al., 1993, 2001) have gener-

ally found a similar pattern of results among normotensive individuals. Table 2 lists those randomized trials of weight loss in overweight hypertensive patients that have included a usual diet control group and did not specifically restrict sodium.

In terms of magnitude of change, MacMahon, Cutter, Brittain, and Higgins (1987) pooled results from a number of interventional studies and estimated that a weight loss of 9.2 kg is associated with a reduction of 6.3-mm Hg SBP and 3.1-mm Hg DBP. More recent trials have supported these estimates, with clinically meaningful reductions of 7–10-mm Hg SBP and 6–7-mm Hg DBP following weight loss of roughly 8 kg (Blumenthal et al., 2000; Dengel, Galecki, Hagberg, & Pratley, 1998). Thus, not only has the association between body weight and BP been well-documented but in addition interventional research has strongly supported the effi-

Table 2

Summary of Randomized Controlled Trials of Weight Loss in Hypertensive Patients Since 1990

Authors	Intervention	Duration	N	Pretx	Posttx	Change in
				SBP/DBP (mm Hg)	SBP/DBP (mm Hg)	SBP/DBP (mm Hg)
Singh, Rastogi, Mehta, Mody, & Garg, 1990	WL (low energy usual diet, with weekly compliance checks)	3 months	416 (WL: 104)	— <sup>a</sup>	—	$-16^b/-13^b$
Andersson, Elam, Wallin, Bjorntorp, & Andersson, 1991	WL (individually adjusted energy-restricted diet with goal loss of 7% of body weight; visits to the dietician every 4 weeks, on average)	3–5 months (M = 4)	20	146/93	142/86 <sup>c</sup> (WL) 146/94 (control)	—
Jalkanen, 1991	WL (1,000–1,500 kcal/day, weekly discussions for first 6 months, every 3 weeks thereafter, educational materials)	12 months	49	152/101 (WL) 155/102 (control)	144/90 (WL) 140/91 (control)	$-8/-11^d$
Kawamura et al., 1993	WL (450 kcal/day in hospital metabolic ward)	2 weeks	20	152/93 versus 149/90 (groups not specified)	Figure shows significant decreases in SBP and DBP for WL group versus control, but values are not reported.	
Singh et al., 1995	WL (1,600 kcal/day, counseling by dietician, with no group differences in sodium intake or physical activity)	16 weeks	217	152.5/99.9 <sup>a</sup> (WL) 154.8/100.5 (control)	—	$-10.5^b/8.0^b$
Whelton et al., 1998	WL (group counseling on nutrition and exercise with goal of $\geq 10$ -lb weight loss)	8 months active intervention; 15–36 months of follow-up	585 (WL: 147)	130/72 <sup>a</sup> (WL) 128/72 (control)	Hazard ratio for combined outcome measures <sup>e</sup> = .64 <sup>b</sup>	
Blumenthal et al., 2000	WL (behavioral change program: weekly group meetings + aerobic exercise; goal of 0.5–1.0 kg/week weight loss through reduced calorie and fat intake)	6 months	133 (WL: 55)	141/94	—	$-7^b/-6^b$

Note. Includes trials with a usual diet control group that did not specifically restrict sodium. Prettx = pretreatment; SBP = systolic blood pressure; DBP = diastolic blood pressure; Posttx = posttreatment; WL = weight loss. Dash indicates *not applicable*.

<sup>a</sup> All participants on a single antihypertensive medication. <sup>b</sup> WL significantly lower than control. <sup>c</sup> Posttreatment DBP was significantly lower than pretreatment BP for WL, with no change in control; statistics comparing treatment with control are not reported. <sup>d</sup> WL not significantly different than control. <sup>e</sup> Outcome measures = diagnosis of high blood pressure (BP), treatment with antihypertensive medication, or cardiovascular event during follow-up.

cacy of weight loss in reducing BP in both normotensive and hypertensive individuals who are overweight.

### *Sodium Restriction*

Sodium chloride intake also has been implicated in the development of HTN, especially on the basis of observations of increased BP in cultures with higher intake of dietary salt (Midgley, Matthew, Greenwood, & Logan, 1996). However, results of within-population studies of the relationship between sodium and BP have been somewhat mixed (Midgley et al., 1996). For example, no support for a relationship between sodium and BP was found in the Scottish Heart Health Study (Smith, Crombie, Tavendale, Gulland, & Tunstall-Pedoe, 1988). In contrast, the International Study of Salt and Blood Pressure (Stamler, 1997), a recent worldwide epidemiological study that examined both within- and cross-population relationships between sodium and BP, found a significant linear relationship between 24-hr urinary sodium excretion, a proxy measure of dietary sodium intake, and SBP. The estimated effect of a 100 mmol per day increase in sodium was a 3–6 mm-Hg increase in SBP and a 0–3-mm Hg increase in DBP. Moreover, an increase in sodium intake of 100-mmol per day was associated with a 30-year increase of 10–11-mm Hg SBP and 6-mm Hg DBP.

These findings were supported by recent reviews of both epidemiological studies and randomized controlled trials of sodium reduction and BP, particularly SBP (Cutler, Follmann, & Allender, 1997; He & Whelton, 1997; Midgley et al., 1996). In a review of 32 studies with outcome data for 2,635 subjects, Cutler et al. (1997) observed a dose–response relationship in which a 100 mmol 24-hr sodium reduction was associated with a decrease of 5.8-mm Hg SBP and 2.5-mm Hg DBP for hypertensives and 2.3-mm Hg SBP and 1.4-mm Hg DBP for normotensives. More recently, Graudal, Galloe, and Garred (1998) performed a meta-analysis of studies examining the effects of reduced sodium on BP and found a similar pattern of results, with a reduction of 3.9-mm Hg SBP and 1.9-mm Hg DBP across 58 trials of hypertensive patients, but a reduction of only 1.2-mm Hg SBP and 0.23-mm Hg DBP in 56 trials of normotensive persons.

This issue is complicated, however, by the fact that individuals vary greatly in their BP response to sodium, with salt-sensitive individuals exhibiting a rise in BP in response to high sodium intake and salt-resistant individuals showing no rise (Sullivan, 1991). Furthermore, some evidence has suggested that restricting dietary sodium too severely may actually have detrimental effects on other variables, such as lipids (e.g., Egan, Weder, Petrin, & Hoffman, 1991) and even cardiovascular morbidity and mortality (e.g., Alerdman, Madhavan, Cohen, Sealey, & Laragh, 1995).

Taken together, the research suggests that for hypertensive individuals who are salt sensitive, there does appear to be a moderately beneficial effect on BP as a result of reducing a high-sodium intake. However, this appears to hold true neither for normotensive individuals nor for individuals who are salt resistant. Moreover, although population-wide changes in BP, even of the small magnitude attained in sodium reduction interventions, could have significant public health implications (Chait et al., 1993), general recommendations to reduce sodium may not be warranted until further research clarifies the longer term impact of sodium

restriction on metabolic factors and cardiovascular morbidity and mortality.

### *Potassium Supplementation*

Because dietary potassium intake has been found to be inversely related to BP in epidemiological data (e.g., Rodriguez, Labarthe, Huang, & Lopez-Gomez, 1994), numerous studies have examined the effects of increased potassium intake on BP, with mixed results (e.g., Brancati, Appel, Seidler, & Whelton, 1996; Grimm et al., 1990). Whelton et al. (1997) recently performed a meta-analysis of 33 randomized controlled trials, including a total of 2,609 subjects, in which potassium supplementation was the only difference between the intervention and control conditions. Analysis of the pooled data showed that potassium supplementation was associated with significant reductions of 3-mm Hg SBP and 2-mm Hg DBP. Moreover, there was a relationship between the effect of potassium and sodium intake, highlighting the importance of sodium–potassium balance. Specifically, greater potassium-related reductions in BP were associated with higher levels of urinary sodium excretion. Similarly, the effect of potassium supplementation on BP was greater in the trials including a majority of African Americans, who tend to be more salt sensitive. Extending these findings, a recent study by West et al. (1999) examined the effects of three dietary interventions (low salt, high salt, and high potassium with high salt) and found similar reductions in BP resulting from either reduced salt intake or increased potassium intake only in salt-sensitive individuals, with no changes in BP across diets in salt-resistant individuals. Of note, the effects of high salt intake were buffered by the high potassium diet in the salt-sensitive individuals.

Thus, on average, increased potassium intake appears to have a weak positive effect on BP, which is strengthened among salt-sensitive individuals. Although this magnitude of change may be relatively small, as in the case of other dietary changes, population-wide changes in BP of this magnitude could produce significant public health benefits. However, further research should attempt to clarify whether this can safely be achieved through dietary supplements. In the meantime, general recommendations may be best limited to increasing dietary intake of potassium through fruits and vegetables.

### *Calcium Supplementation*

Early studies of environmental factors related to BP that suggested a relationship between hard water and HTN prompted interest in the contribution of minerals, including calcium and magnesium, to elevated BP. A recent meta-analysis of epidemiological studies of the association between dietary calcium intake and BP (Cappuccio et al., 1995) evaluated 23 population studies that included 38,950 people. Results of this analysis indicated an inverse association between dietary-calcium intake and BP. This same group also conducted a meta-analysis of data from 22 randomized clinical trials of the effects of dietary-calcium supplementation on BP (Allender et al., 1996). The effect of calcium supplementation on BP across the pooled data was a reduction of slightly less than 1.0-mm Hg SBP and 0.2-mm Hg DBP, with larger decreases in SBP among hypertensives than among normo-



tensives. This negligible effect does not appear to merit recommending calcium supplementation to reduce BP at this time.

### *Magnesium Supplementation*

Epidemiological studies have shown lower levels of dietary magnesium intake to be related to elevated BP as well as to the prevalence of HTN (Harlan & Harlac, 1995; Kesteloot & Joossens, 1988; Witteman et al., 1989). In addition, hypertensive patients have been found to have reduced serum and intracellular levels of magnesium compared with normotensives (Kawano, Matsuoka, Takishita, & Omae, 1998). These findings suggest that magnesium supplementation may have a beneficial effect on BP. Until recently, however, few randomized, controlled trials of magnesium supplementation had been conducted, leaving the Joint National Committee (1997) to conclude that there was "no convincing data currently [to] justify recommending an increased magnesium intake in an effort to lower BP" (p. 2423). Indeed studies that have examined the relationship between magnesium supplementation and BP generally have been small, although results often have suggested a beneficial effect of magnesium supplementation on BP (e.g., Motoyama, Sano, & Fukuzaki, 1989; Widman, Wester, Stegmayr, & Wirell, 1993). One of the few larger randomized trials of magnesium supplementation on BP was the Trials of Hypertension Prevention (Yamamoto et al., 1995), which found no effect of magnesium supplementation on BP in healthy adults with high-normal DBP. However, a recent randomized, cross-over study of 60 hypertensive individuals did find a small but significant reduction in BP resulting from 8 weeks of magnesium supplementation (Kawano et al., 1998). It remains unclear whether the failure to find a relationship between magnesium supplementation and BP in some studies is due to a real lack of association or whether the association is small and only evident at higher levels of BP. In either case, the evidence does not support recommending increased magnesium intake as a preventive measure or treatment for HTN at the current time.

### *Alcohol Restriction*

Alcohol consumption generally has been shown to be associated with elevated BP in epidemiological studies (see Beilin, Puddey, & Burke, 1996). Although many studies have found a linear relationship between alcohol consumption and BP, in some studies a threshold effect of two or three drinks has been observed. In fact, in a review of 30 cross-sectional population studies by MacMahon (1987), about 40% of studies found higher BP among nondrinkers as compared with individuals consuming one to two drinks per day. It is not entirely clear why there have been mixed findings in this area. However, recent results of the Atherosclerosis Risk in Communities Study (Fuchs, Chambless, Whelton, Nieto, & Heiss, 2001) suggest that the association between alcohol consumption and BP may vary across different groups. This study found, for example, that although alcohol consumption of  $\geq 210$  g per week was an independent risk factor for HTN in all race and gender groups, only in Black men was consumption of low to moderate amounts of alcohol associated with a higher risk of hypertension.

In addition to findings from cross-sectional studies, observational studies have found a direct relationship between change in alcohol consumption and change in BP (T. Gordon & Doyle, 1986;

T. Gordon & Kannel, 1983; Kromhout, Bosschieter, & Coulander, 1985). These findings have prompted the Joint National Committee (1997) to recommend limiting alcohol intake to no more than 1 oz (29.5 ml) of ethanol per day.

However, much of the research on alcohol restriction and BP has focused on alcoholics during and after detoxification (Aguilera et al., 1999; Saunders, Beevers, & Paton, 1981), and fewer studies have examined the relationship in light to moderate drinkers. Despite these limitations, the overall findings in studies of alcohol restriction and BP do indicate that reducing alcohol intake can lower BP, particularly in heavy drinkers. Keil, Liese, Filipiak, Swales, and Grobbee (1998) reviewed the literature on alcohol and BP over the past 30 years and concluded that a direct and causal relationship exists between chronic intake of  $\geq 30$ –60 g of alcohol per day and elevated BP and that reducing alcohol intake is effective in lowering BP. From these data, it was estimated that, above 30 g of alcohol per day, for each additional 10 g consumed, SBP could be expected to increase by 1–2 mm Hg and DBP by 1 mm Hg. Although a recently conducted controlled randomized trial of moderate alcohol restriction on BP (Cushman et al., 1998) found that a decrease of 1.3 drinks per day produced nonsignificant changes in BP, the baseline consumption of alcohol was not considered heavy (440 g/week; Bulpitt & Shipley, 1999). Therefore, recommending moderate alcohol consumption appears warranted, but complete abstinence in nonproblem drinkers does not appear necessary, especially in light of the beneficial effects of moderate alcohol consumption on coronary artery disease risk (Joint National Committee, 1997).

### *Summary*

Of all the dietary interventions, weight loss for the overweight individual appears the most promising in terms of its potential impact on BP. However, modifications in sodium, potassium, and alcohol intake may be beneficial on a population-wide basis for certain groups. Specifically, sodium reduction for salt-sensitive individuals who consume a high-sodium diet and are hypertensive may be beneficial. Increased intake of dietary potassium, especially for groups with typically low intake of potassium or high intake of sodium, or with a greater tendency toward salt sensitivity, may also be an important public health step. Finally, recommending reduced alcohol consumption among heavy drinkers appears warranted, although the resulting reduction in BP for any given individual may be relatively small. Increased intake of calcium and magnesium do not at this time appear to confer a significant beneficial effect on BP. However, epidemiological data do suggest an inverse relationship between dietary intake of these minerals and BP. It may be that this relationship is the result of confounding factors. For example, individuals with lower intake of magnesium and calcium also may consume more sodium or alcohol or may be more overweight.

An overall healthy pattern of eating may indeed be the best defense against high BP. Recent results of the Dietary Approaches to Stop Hypertension study (Appel et al., 1997) support this idea. This clinical trial of the effects of dietary patterns on BP included 459 participants with normal BP or mild HTN who were randomly assigned to 8 weeks of a control diet (typical U.S. diet), a diet rich in fruits and vegetables, or a combination diet rich in fruits, vegetables, and low-fat dairy foods and low in saturated fat and

total fat. Both special diets reduced BP compared with the control diet, although the combination diet was most effective in reducing BP. Moreover, although both normotensives and hypertensives benefited from the special diets, hypertensives received a greater benefit. For hypertensive individuals, the combination diet reduced SBP by 11.4 mm Hg and DBP by 5.5 mm Hg over the control diet. Given the efficacy of modifying diet in reducing BP and the relative lack of adverse side effects associated with dietary modification, this type of lifestyle intervention is clearly warranted for both prevention and treatment of high BP. Dietary interventions can serve as a preventive measure by reducing BP in normotensive individuals, as initial therapy in Stage 1 HTN, and as a complement to antihypertensive medication in patients with established HTN (Appel, 1999).

### Biofeedback, Relaxation Therapies, and Stress Management

Observational studies have suggested that chronic real-life stress may contribute to the development of HTN (Baum, 1990; Rose et al., 1979; Schnall et al., 1990; Theorell et al., 1991). Moreover, laboratory studies provided evidence that heightened behavioral or mental stress-induced cardiovascular responses may characterize individuals at risk for the development of HTN (Fredrikson & Matthews, 1990); individuals with normal BP who have hypertensive parents also typically exhibit greater BP reactivity to laboratory stressors than normotensives with no family history (Muldoon, Terrell, Bunker, & Manuck, 1993). Gender is also related to heightened BP responsiveness, with men exhibiting greater SBP responses than women (Saab, 1989) and postmenopausal women showing greater BP reactivity than premenopausal women (Owens, Stoney, & Matthews, 1993). Some studies have observed greater BP reactivity in African Americans than in Caucasians, but this finding has been inconsistent (Light, Obrist, Sherwood, James, & Strogatz, 1987). There is consistent evidence of ethnic differences in vascular responses to stress, however, with African Americans showing abnormal elevations in systemic vascular resistance, which may account, in part, for the greater prevalence of HTN among African Americans relative to Caucasians (Sherwood, May, Siegel, & Blumenthal, 1995).

In addition to these cross-sectional studies, evidence from several prospective studies also was consistent with an etiological role for stress reactivity in the development of HTN (Kasagi, Akahoshi, & Shimaoka, 1995; Light, Dolan, Davis, & Sherwood, 1992; Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998; Menkes et al., 1989) and hypertensive end-organ disease. It has been reported that the magnitude of BP response during stress is directly related to LV mass, suggesting a relationship between BP reactivity and the etiology of LVH (Hinderliter, Light, Girdler, Willis, & Sherwood, 1996). In a prospective study of borderline hypertensive White men, Georgiades, Lemne, de Faire, Lindvall, and Fredrikson (1997) reported that high BP reactivity to mental arithmetic and a hand-grip stressor was predictive of significantly greater LV mass at 3-year follow-up than in the low-BP-reactive males, and in a retrospective study of adolescents, LVH was found to be related to an aggregate measure of BP responses (Murdison et al., 1998). Taken together, both naturalistic and laboratory studies provided strong evidence for the role of stress in the development of HTN and end-organ damage associated with

chronically elevated BP and, thus, provided the rationale for interventions designed to reduce stress in patients with HTN.

The effectiveness of interventions to reduce stress—yoga, meditation, relaxation, biofeedback, and cognitive-behavioral therapies—has been evaluated extensively (Eisenberg et al., 1993; Jacob, Chesney, Williams, Ding, & Shapiro, 1991; Kaufmann et al., 1988; Linden & Chambers, 1994) and critically reviewed by a number of international working groups (Joint National Committee, 1997; Spence, Barnett, Linden, Ramsden, & Taenzer, 1999).

Although studies conducted in the 1970s reported encouraging results (Jacob, Kraemer, & Agras, 1977; A. P. Shapiro, Schwartz, Ferguson, Redmond, & Weiss, 1977), the majority of those early studies suffered from major methodological shortcomings. Results tended to be more pronounced in patients with higher pretreatment BPs, suggesting that regression to the mean, at least partly, was responsible for the BP reductions. In addition, the largest declines in SBP were reported in studies that included only one baseline session and used patients that were new to the clinic environment. When patients were given the opportunity to habituate to the assessment environment, baseline BPs were lower and the intervention effects were smaller. When more methodologically sophisticated studies were conducted subsequently, the results from randomized controlled trials using stress-reducing interventions, especially single-method treatments, were less encouraging.

Another problem with the early studies was that many studies had small sample sizes. Jacob, Fortmann, Kraemer, Farquhar, and Agras (1985) estimated that a sample size of 30 or more patients per group is required to detect a change in SBP of 5 mm Hg at the 5% significance level with a statistical power of 0.80. In addition, the use of different therapies was not standardized and therapies were often combined, either with each other or with other interventions such as diet or pharmacological treatments, so that it was not clear to what to attribute BP changes. Three stress-reduction interventions have received the most attention: biofeedback, relaxation therapies, and stress management.

### *Biofeedback*

The American College of Physicians summarized the results from trials using SBP biofeedback, DBP biofeedback, or both and noted an average BP decline of 7.8/5.6 mm Hg (Health and Public Policy Committee, 1985). However, none of the studies reported had adhered to even the minimal qualitative requirements specified as necessary for valid conclusions (Sackett, Haynes, & Tugwell, 1985).

Subsequent reviews in which biofeedback was considered as a single-treatment method reported biofeedback to have only limited BP-reducing effects (Eisenberg et al., 1993; Jacob et al., 1991). Since 1990, there have been four randomized controlled studies of biofeedback as a single treatment in patients with HTN (Table 3). Hahn and colleagues (Hahn et al., 1993) found significant decreases in BP as compared with a control group after only a single biofeedback session, and the effect persisted throughout the eight treatment sessions. However, more recent studies are less encouraging. Blanchard et al. (1996) reported that an 8-week thermal biofeedback-training program had no effect on either clinic or ABP measurements. Hunyor et al. (1997) found that almost 50% of the hypertensive patients in their study were able to lower their BP in the laboratory using thermal biofeedback. However, the

Table 3

Summary of Randomized Controlled Trials of Relaxation, Stress Management, or Biofeedback Therapies in Hypertensive Patients Since 1990

Authors	Intervention	Duration	N	Pretx	Posttx	Change in
				SBP/DBP (mm Hg)	SBP/DBP (mm Hg)	SBP/DBP (mm Hg)
Bennett, Wallace, Carroll, & Smith, 1991	Stress management training (SMT) or SMT + Type A behavior modification (TAM)	6 months	44 (SMT: 15) (TAM: 15)	149.2/92.9 (TAM)	140.0/86.4 <sup>a</sup> (TAM)	—
				155.9/93.0 (SMT)	147.9/85.4 <sup>a</sup> (SMT)	
				151.2/93.5 (control)	153.1/94.6 (control)	
Hahn et al., 1993	Thermal biofeedback (two sessions/week) + one 20-min progressive-relaxation self-training session	4 weeks	19 (biofeedback: 11)	150.0/101.4 (biofeedback) 147.9/102.1 (control)	—	−20.6/14.4 <sup>a</sup>
Johnston et al., 1993	10 individual stress management (SM) sessions including passive relaxation, meditation, and instructions of how to use relaxation techniques in everyday life	6 months	96 (SM: 48)	Clinic: 140.1/ 92.5 (SM)	Clinic: 140.7/ 92.4 <sup>b</sup> (SM)	—
				141.0/91.4 (control)	141.8/91.8 (control)	
				ABP: 133.5/92.6 (SM)	ABP: 132.7/ 93.8 <sup>b</sup> (SM)	
McGrady, 1994	Relaxation training and thermal biofeedback (one session/week)	20 weeks	101 (relaxation: 70)	132.5/85.8 (relaxation)	126.5/82.6 <sup>a</sup> (relaxation)	—
				130.9/85.6 (control)	130.0/86.6 (control)	
Schneider et al., 1995	Transcendental meditation (TM) or progressive muscle relaxation (PMR)	3 months	111 (TM: 36) (PMR: 37)	145.4/93.7 (TM)	—	−10.9/−5.6 <sup>c</sup> (TM)
				144.3/89.2 (PMR)		−4.9/−2.5 <sup>c</sup> (PMR)
				150.4/91.7 (control)		
Blanchard et al., 1996	Thermal biofeedback (seven sessions for hand and nine sessions for foot warming)	8 weeks	42 (biofeedback: 21)	142.1/93.2 (biofeedback)	140.0/91.3 <sup>b</sup> (biofeedback)	—
				140.0/90.1 (control)	141.9/91.1 (control)	
Yung & Keltner, 1996	Relaxation—three treatment groups: stretch release (SR), tense release (TR), cognitive relaxation (COG); two control conditions: placebo attention (PA), test only control (TOC)	8 sessions	30 (SR: 6) (TR: 6) (COG: 6)	153.8/87.8 (SR)	132.8/78.7 (SR)	—
				155.5/87.9 (TR)	132.3/79.1 (TR)	
				154.9/87.7 (COG)	133.1/76.3 (COG)	
				154.1/94.7 (PA)	138.1/88.7 (PA)	
			154.5/90.3 (TOC)	146.1/84.9 (TOC)		
Hunyor et al., 1997	Biofeedback (eight sessions of continuous blood pressure feedback)	4 weeks	56 (biofeedback: 28)	153/97 (biofeedback) 154/98 (control)	—	SBP −5 <sup>b</sup>
Henderson, Hart, Lal, & Hunyor, 1998	Biofeedback (8 laboratory and 12 home-training sessions of continuous blood pressure feedback)	4 weeks	30 (biofeedback: 16)	154/98 (biofeedback)	144/91 (biofeedback)	−9/−7 <sup>b</sup>
				152/96 (control)	147/93 (control)	
Batey et al., 2000	37 contact hours of SM including relaxation, stress reduction techniques, cognitive approaches, communication skills, time management, anger management, etc.	18 months	562 (SM: 242)	125.3/83.9 (SM) 124.1/83.6 (control)	—	−4.2/−5.53 <sup>b</sup>
Linden, Lenz, & Con, 2001	1-hr sessions of individualized SM including autogenic training, biofeedback, cognitive therapy, anxiety management, and Type A reduction	10 sessions	60 (SM: 27)	Clinic: 152.0/ 97.9 (SM)	—	Clinic: −6.9/−5.4 <sup>b</sup>
				154.1/98.9 (control)		ABP: −6.1/ −4.3
				ABP: 154.7/98.5 (SM)		
				150.6/95.5 (control)		

Note. Pretx = pretreatment; SBP = systolic blood pressure; DBP = diastolic blood pressure; Posttx = posttreatment. Dash indicates *not applicable*.

<sup>a</sup> Posttx significantly different than pretx. <sup>b</sup> Treatment not significantly different than control. <sup>c</sup> Treatment significantly different than control.

placebo group also lowered their BP to a comparable degree, and the biofeedback sessions had no effect on ABP levels. Henderson, Hart, Lal, and Hunyor (1998) found a clinically significant decrease of BP levels in the active biofeedback group after 4 weeks of home-training exercise, but the placebo group also exhibited lowered BP, so the Time  $\times$  Group interaction was not significant. Thus, even when biofeedback is associated with reduced BP, the effect is usually not significantly different from that of a placebo or sham biofeedback interventions.

### *Relaxation Therapies*

In the Hypertension Intervention Pooling Project, Kaufmann et al. (1988) reviewed 12 randomized controlled trials of relaxation therapies in the treatment of HTN. The studies used either relaxation therapy alone, relaxation in combination with biofeedback, or relaxation in combination with other treatments such as dietary counseling or cognitive restructuring. The meta-analysis showed a significant, although modest, decrease in DBP for patients who were not pharmacologically treated, but no significant improvement for medicated patients. The effect on SBP was not significant. It was also evident that patients with higher pretreatment BP levels showed the greatest change.

Since 1990, there have been three randomized controlled trials using relaxation in the treatment of HTN (see Table 3). In a study by McGrady (1994), relaxation sessions were combined with thermal biofeedback techniques. The study showed significant BP reductions in the treatment group as compared with the control group. Schneider et al. (1995) compared transcendental meditation and progressive muscle relaxation (PMR) with a lifestyle-modification education control program and with each other among African Americans with HTN. Adjusted for significant baseline differences and compared with controls, transcendental meditation and PMR lowered both SBP and DBP. The reductions in the transcendental meditation group were significantly greater than in the PMR group for both SBP and DBP. In a study by Yung and Keltner (1996), four relaxation techniques were compared with control conditions. It was concluded that the "muscle tension-release" procedure proved most effective in lowering BP but that both muscular and cognitive relaxation procedures significantly reduced BP compared with control conditions.

### *Stress Management*

Stress management consists of techniques that reduce excessive stress arousal by changing cognitive and emotional responses to events. These can include cognitive restructuring, adaptive emotional-learning strategies, imagery, and psychosocial or mental relaxation. Most early studies on stress-management interventions showed consistent, although modest, reductions in BP (see McCaffrey & Blanchard, 1985), whereas a few studies demonstrated a remarkable degree of success, persisting over several years after cessation of therapy (Patel, 1997; Patel & Marmot, 1988). Intervention effects are highly correlated to the pretreatment BP levels, however, emphasizing the need for multiple baseline measurements to acquire stable pretreatment BP levels and avoid exaggerated regression to the mean effects.

Since 1990, there have been four randomized controlled studies using stress management techniques in the treatment of hyperten-

sion (see Table 3). Bennett, Wallace, Carroll, and Smith (1991) treated unmedicated hypertensives with either a stress management intervention or a Type A management program. Both interventions showed BP lowering effects as compared with the control condition. Johnston et al. (1993) found that BP dropped significantly during the 12-week baseline period when patients habituated to measurements of BP, but neither resting nor ABP was changed by the end of the treatment. It was concluded that stress management of the type advocated for treating mild HTN is ineffective in lowering BP in patients who are well-habituated to BP measurement. A major implication from this study is that previously reported BP reductions associated with stress reducing interventions may have reflected adaptation effects. The stress management intervention might just have helped the process of adaptation to measurement, lowering the BP readings for individuals who were not well-adapted to BP measurement procedures. Thus, it is critical that future studies include an extended baseline assessment over 3–4 weeks to minimize the potentially confounding effects of adaptation to measurement.

Batey et al. (2000) evaluated stress management as one of seven nonpharmacological approaches in Phase I Trials of Hypertension Prevention (TOHP-I) for efficacy in lowering DBP in healthy men and women aged 30 to 54 years with DBP 80–89 mm Hg. Participants were randomized to stress management or a control group at four clinical centers. The only statistically significant effect was a 1.36-mm Hg reduction in DBP relative to controls at the end of the trial for stress management participants who completed at least 61% or more of the intervention sessions.

In a recent study by Linden, Lenz, and Con (2001), an individualized stress management intervention had significant BP reducing effects on ABP levels, although office BP levels were not different between treatment group and controls. The same effect was replicated when the waiting list control group was treated and assessed, and results further improved with increased effect size at follow-up.

### *Single Versus Multicomponent Treatments*

Jacob et al. (1991) conducted a meta-analysis of stress-reducing therapies in the treatment of HTN. The analysis included studies in which subjects did not substantially change medication dose during treatment, relaxation or meditation was a component in the treatment, subjects did not have normal BP, children or geriatric patients were excluded, and the sample included at least 6 or more subjects. A total of 75 treatment groups and 41 control groups were thereby included in the analysis. It was concluded that single-component interventions, such as meditation or relaxation therapy, showed small effects or no reduction in BP levels ( $-5.7$  to  $+3.5$  mm Hg for SBP and  $-3.1$  to  $+2.3$  mm Hg for DBP).

In a subsequent meta-analysis by Eisenberg et al. (1993), more than 80 studies applying cognitive-behavioral techniques to adults with HTN were examined. The majority of studies were considered to have general methodological problems, including lack of data regarding specific individual characteristics such as age, race, gender, and medication in relation to baseline BP or BP change following intervention. Only 26 studies met formal criteria for randomized controlled trials with data sufficient to be included in an analysis (i.e., patients had been randomly assigned to an experimental or control group, and the outcome variables had been

reported in detail). Eisenberg et al. also concluded that there were no significant effects on BP from single-component interventions (SBP effects between  $-1.5$  mm Hg and  $+2.9$  mm Hg; DBP effects between  $-0.8$  mm Hg and  $+1.2$  mm Hg), whereas combination therapies showed more promising results ( $-13.5$  mm Hg for SBP and  $-3.4$  mm Hg for DBP).

More recently, Linden and Chambers (1994) conducted a meta-analysis comparing 90 studies on stress-reduction in the treatment of HTN with 30 pharmacological and 47 behavioral interventions, adjusting all treatment effects for baseline pretreatment BP levels. The pharmacological treatment group consisted of studies using diuretics, beta-blockers, or calcium-channel blockers as compared with placebo. The behavioral-intervention treatment group included studies of weight reduction, exercise, sodium restriction, alcohol restriction, calcium supplementation, or potassium supplementation. Results showed only small or no effects on BP reductions from the single-component stress-reducing therapies, whereas in contrast, the multicomponent stress management interventions reduced BP to a greater degree and over a longer period of time ( $-9.7/-7.2$  mm Hg), and individualized cognitive-stress management showed the largest BP reductions, with BP reductions on average of  $-15.2/-9.2$  mm Hg. A comparison between individualized stress management, exercise, and pharmacological treatments revealed that the three interventions were equally successful in lowering SBP levels, whereas pharmacological interventions were better in reducing DBP levels.

### Generalization of Treatment Effects

Most studies have used clinic BP measurements as both selection and outcome variables. However, because a small number of measurements taken in the clinic are unable to reflect the changes occurring during everyday life, there has been a growing interest in examining BP measurements obtained during an ordinary day. Although most previous studies examining the effects of stress reduction therapies on ABP in hypertensive patients have shown no significant reduction in ABP (Jacob et al., 1992; Johnston et al., 1993; Van Montfrans, Karemaker, Wieling, & Dunning, 1990), a recent study by Linden et al. (2001) showed significant reductions of ABP levels after an individualized stress management program as compared with controls. Of interest, the clinic office BPs were not significantly different between the treatment and control group.

### Summary

Many early studies conducted to investigate the effect of cognitive-behavioral interventions in the treatment of HTN suffered from serious methodological limitations. The majority of randomized controlled studies have shown relatively small BP reductions from single-component interventions such as relaxation and biofeedback, and the effects were usually not significantly different from that of a placebo or sham intervention. There is some evidence, however, that individualized multicomponent cognitive behavioral stress therapy may have a significant BP lowering effect.

### Mechanisms

Most behavioral intervention studies have focused on effectiveness and have not considered the physiological mechanisms that

might be responsible for the observed BP reductions. Because BP regulation is complex, involving multiple physiological systems, BP-lowering mechanisms are likely to reflect both intervention mode and individual difference characteristics. In the following section, we consider some of the BP regulatory mechanisms that may be altered by biobehavioral interventions. A summary of how each behavioral intervention may affect these mechanisms is depicted in Table 4.

### Sympathetic Nervous System and Hemodynamic Functioning

Because BP is the product of cardiac output (CO) and systemic vascular resistance (SVR), an abnormal elevation in one or both of these parameters provides the hemodynamic basis for elevated BP. In the early stages of HTN, several now-classic studies have described a hyperkinetic circulatory state, characterized by elevated CO and normal SVR (Julius & Conway, 1968; Lund-Johansen, 1967; Sannerstedt, 1966). A combination of heightened sympathetic and reduced parasympathetic activity has been shown to be responsible for such excessive circulatory stimulation in the initial stages of HTN (Julius, Pascual, & London, 1971). In documenting the natural history of HTN, Lund-Johansen (1967) described a progressive rise in vascular resistance, with abnormally elevated SVR defining the characteristic hemodynamic profile of established HTN. However, there are marked individual differences in the etiology and development of HTN, and although the early hyperkinetic circulatory state may characterize some instances of the onset of HTN, it is elevated SVR that is the dominant feature of HTN in the population as a whole.

Evidence that sympathetic nervous system activity is increased in patients with essential HTN, irrespective of hemodynamics, has become increasingly compelling as measures of sympathetic activity have grown more sophisticated and precise (Grassi, 1998). Therefore, interventions that reduce SVR and sympathetic activity may be optimal treatments for HTN because they should lower BP by abating the underlying disease process. Several studies have shown that BP reduction associated with physical fitness or exercise training is accompanied by a reduction in SVR (W. H. Martin et al., 1991; Meredith et al., 1990).

Other studies have reported a decrease in circulating catecholamines with exercise training (Jennings et al., 1986; Kiyonaga et al., 1985). Duncan et al. (1985) reported significant reduc-

Table 4  
*Potential Mechanisms Contributing to Blood Pressure Reduction by Behavioral Interventions*

Variable	Exercise	Weight loss	Stress management
Heart rate	↓	—	↓
Cardiac output	— or ↑	— or ↑	?
Systemic vascular resistance	↓	— or ↓	?
SNS activity	↓	↓	↓
Stress reactivity	— or ↓	?	— or ↓
Baroreceptors	↑	?	?
Insulin sensitivity	↑	↑	?

Note. ↓ = decrease; — = no change; ↑ = increase; ? = unknown; SNS = sympathetic nervous system.

tions in resting BP in trained hypertensives and normotensives and noted that the magnitude of BP reductions was related to the level of catecholamines. It has been well-established that exercise training is associated with lower resting heart rate and reduced heart-rate response to acute physical exercise (Duncan et al., 1985; Pavlik & Frenkl, 1975). Hagberg et al. (1984) reported that plasma catecholamine responses to matched submaximal exercise were attenuated in trained hypertensives, suggesting reduced sympathetic nervous system activity. If sympathetic nervous system activity may be reduced by behavioral interventions such as exercise or stress management, plasma renin activity (PRA) could also be reduced. Reduced PRA should then result in reduced vasoconstrictive influences from the renin-angiotensin system and faster elimination of salt and fluid by the kidneys, which may be important mechanisms contributing to BP lowering. Renal effects of behavioral interventions should be included in future investigations.

The effects of the sympathetic nervous system on the circulation are mediated by cardiac and vascular adrenergic receptors, which in turn regulate CO and SVR and, ultimately, determine BP. One hallmark of HTN is that the cardiovascular beta-adrenergic receptors are down-regulated, a phenomenon that is thought to be a consequence of their overstimulation by the sympathetic nervous system (Bertel, Buhler, Kiowski, & Lutold, 1980; Feldman, 1987; Sherwood & Hinderliter, 1993). Because vascular beta-adrenergic receptor activation leads to vasodilatation, their desensitization is one mechanism that contributes to the elevation of SVR in HTN. Therefore, interventions that impact vascular beta-adrenergic receptors by restoring their functionality should also help reduce SVR and lower BP. Evidence in support of this possibility comes from an isoproterenol infusion study in which endurance-trained men showed heightened vascular beta-adrenergic responsiveness (Svedenhag, Martinsson, Ekblom, & Hjendahl, 1991). However, in the same study, as well as other studies, fitness failed to show any association with *in vitro* measures of beta-adrenergic receptor function on lymphocytes.

### *Insulin*

Observational studies have shown that HTN very often occurs in conjunction with other cardiovascular risk factors in what has been referred to as "metabolic clustering" (Kannel, 1996). These frequently co-occurring traits include obesity, dyslipidemia, glucose intolerance, insulin resistance, and hyperuricemia. Obesity is strongly associated with elevated BP and other cardiovascular risks, particularly when excess fat is deposited in the abdominal area (Pouliot et al., 1994). Insulin resistance has been implicated as the link among these cardiovascular risk factors (De Fronzo, 1990), and data increasingly suggest a causal relationship between insulin resistance and elevated BP (Reaven, Lithell, & Landsberg, 1996). Lower insulin associated with reduced insulin resistance may reduce the renal sodium retention caused by insulin and may result in lower sympathetic nervous system activity (O'Hare, 1988). In obese patients, both weight loss and exercise have been shown to improve glucose metabolism and insulin resistance (Anderssen, Holme, Urdal, & Hjermand, 1995; Blumenthal et al., 2000; Dengel et al., 1998; Golay et al., 1985; Katznel et al., 1995; Niskanen et al., 1996; Su et al., 1995).

### *Baroreceptors*

HTN is associated with abnormally low levels of baroreflex control (Goldstein, 1983; Gribbin, Pickering, Sleight, & Peto, 1971; Parati et al., 1988). In fact, it was once hypothesized that impaired baroreflex buffering systems led to the onset of HTN, through loss of inhibitory feedback of sympathetic activity. However, with the development of improved techniques for measurement of sympathetic nerve activity, there is increasing evidence that baroreflex control of sympathetic activity is intact in HTN (Grassi, 1998; Meyrelles, Tinucci, Hollanda, & Mion, 1997). These findings suggest that with respect to baroreceptor function, HTN may be more closely related to impairment of vagal reflex control of heart rate than to altered sympathetic control.

Data on exercise training suggest that regular exercise training is associated with improved baroreceptor sensitivity (Jingu et al., 1988; Pagani et al., 1988; Somers et al., 1991). This effect is more pronounced in subjects who are inactive at baseline, and it requires 3 to 6 months of regular exercise. For example, in one study of 10 healthy middle-aged men (Sheldahl, Ebert, Cox, & Tristani, 1994) and in a second study in sedentary postmenopausal women (Davy, Willis, & Seals, 1997), 12 weeks of aerobic training was not associated with changes in baroreflex control. However, in a study of 16 patients with borderline HTN (Somers et al., 1991), 6 months of aerobic training was associated with increases in baroreflex sensitivity and increased heart rate variability. Improved autonomic control has also been observed in older volunteers randomized to 4 (Jingu et al., 1988) or to 6 months of supervised training (Schuit et al., 1999). The effects of other lifestyle interventions on baroreceptor function remain largely unknown.

### *Cardiovascular Stress Reactivity*

Cross-sectional studies have shown that fit individuals are less reactive to emotional stressors with respect to heart rate and BP (Crews & Landers, 1987) and may recover more quickly than their unfit counterparts. Although several exercise intervention studies failed to find an attenuation of heart rate or BP responses to mental stressors (Albright, King, Taylor, & Haskell, 1992; Roskies, Seraganian, Oseasohn, Hanley, & Collu, 1986; Sinyor, Peronnet, Brisson, & Seraganian, 1988), several recent studies of normotensive men have shown reduced cardiovascular and catecholamine responses to standard laboratory challenges such as mental arithmetic (Blumenthal et al., 1988, 1990). Moreover, several studies of patients with borderline HTN (Rogers, Probst, Gruber, Berger, & Boone, 1996; Sherwood, Light, & Blumenthal, 1989) showed attenuated BP responses to mental stress. In the study by Sherwood et al. (1989), patients undergoing aerobic-exercise training experienced a significant reduction in DBP during rest and showed an attenuated DBP response to a competitive behavioral task. These changes were believed to be a function of altered vascular tone, and it was suggested that increased sensitivity of vascular beta-adrenergic receptors and/or a proliferation of skeletal muscle precapillary blood vessels might have been responsible for the reductions in DBP. Because opioid peptides are involved in the regulation of BP reactivity (McCubbin, 1993), exercise may also alter the central opioid system. Indeed, there may be multiple mechanisms for reductions in mental-stress-induced BP. In a recent study comparing exercise alone and in combination with a

behavioral weight-loss program (Georgiades et al., 2000), both treatment groups exhibited lower SBP levels compared with controls during mental stress, and the addition of a weight reduction program appeared to enhance the effects of exercise, resulting in lower DBP levels than exercise alone.

### Clinical Significance and Future Research Directions

Although there have been numerous studies that have documented an association between lifestyle factors and BP, there have been very few randomized controlled trials of lifestyle modification in individuals with HTN. Moreover, inadequate attention to methodological details has impeded progress in this area. Rigorous methodological controls including careful medical screening; precise documentation of hypertensive status; well-described, reproducible interventions; and randomized designs are imperative. Consideration of individual differences should also be included, as there appears to be considerable variability in response to treatment. The most important lifestyle behavior to modify may be different for different patients, so that identifying which treatment will be most effective for which patient will be an important area for future research. In addition, consideration of mechanisms by which exercise may reduce BP should be included in the design. Animal studies also offer considerable promise, particularly for better understanding mechanisms mediating behaviorally induced changes in BP. Although animal studies permit greater experimental control, methodological and conceptual issues (e.g., confounding stress and exercise in paradigms that use swimming, generalizing from certain animal strains to human populations) should also not be overlooked. Finally, investigations of the interactive effects of lifestyle behaviors and medication may be valuable for the practical management of patients, as well as for better understanding the mechanisms of BP regulation.

The clinical significance of BP reductions in patients with HTN who undergo lifestyle modification has not been systematically evaluated (see special section of the *Journal of Consulting and Clinical Psychology*, June 1999; Kendall, 1999). Although changes in such clinical markers as the presence of LVH have been shown to be modified (Hinderliter et al., in press; Kokkinos et al., 1995; Turner, Spina, Kohrt, & Ehsani, 2000), to our knowledge, no studies of behavioral interventions in patients with HTN have examined the impact of such interventions on "hard" clinical endpoints such as stroke, myocardial infarction, or death. The feasibility of conducting a study with such clinical endpoints is an issue, as such a clinical trial would involve many participants in a multicenter clinical trial who would be required to initiate and maintain lifestyle modifications over a relatively long follow-up period. Nevertheless, such an approach could yield important information. Cook, Cohen, Herbert, Taylor, and Hennekens (1995) performed an analysis of the effects of a population-wide reduction in DBP of only 2 mm Hg and found that such a decrease could lower the risk of stroke and transient ischemic attack (TIA) events by 15% and CAD by 6%. These figures translate into preventing an estimated 35,000 strokes and TIA events and almost 70,000 CAD events annually in U.S. residents aged 35–64 years. BP reductions induced by lifestyle changes may be of a comparable order of magnitude, so there is considerable reason to believe that such reductions induced by the kinds of lifestyle modifications described in this review may have significant clinical significance.

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