



## Recommendations of the Canadian Consensus Conference on Non-pharmacological Approaches to the Management of High Blood Pressure, Mar. 21-23, 1989, Halifax, Nova Scotia\*

**Arun Chockalingam, MS, PhD; Darlene Abbott, RN, MSc; Martin Bass, MD, MSc, CCFP; Renaldo Battista, MD, MPH, ScD, CCFP, FRCPC; Roy Cameron, MS, PhD; Jacques de Champlain, MD, PhD, FRCPC; C. Edward Evans, MD, BS, MRCS, CCFP; John Laidlaw, MD, PhD, FRCPC; Betty Lou Lee, BA; Lawrence Leiter, MD, CM, FRCPC; Richard Lessard, MD, PhD, MPH, FRCPC; David MacLean, MD, FHS; James Nishikawa, MD, FRCPC; Simon Rabkin, MD, PhD, FRCPC; Carole Thibaudeau, BSc; Dorothy Strachan, MA**

**F**or some time the role of nonpharmacologic management, alone or in combination with drug treatment, has been controversial. When such controversies exist among researchers the task of health care professionals in promoting lifestyle changes in the community can be difficult.

To resolve such controversies and arrive at a consensus the Canadian Consensus Conference on Non-Pharmacological Approaches to the Management of High Blood Pressure was convened Mar. 21 to 23, 1989, in Halifax, NS. A large body of literature on the seven most discussed nonpharmacologic approaches was assembled by experts in each of the topic areas. The speakers presented current knowledge to a panel comprising Canadian experts from disciplines of health sciences as well as

from the general public. The panel assessed the evidence and arrived at this consensus document.

It was a pleasure to organize and conduct this conference. I thank every member of the Organizing Committee, the speakers (both international and Canadian) and all panel members. I am also grateful for the enormous support extended to this conference by both the sponsoring agencies and the supporting corporations.

These recommendations will have a major impact on the Canadian health care system, particularly in the containment of cardiovascular diseases.

Arun Chockalingam, MS, PhD  
Conference chairman  
September 1989

*\*Jointly sponsored by the Canadian Coalition for High Blood Pressure Prevention and Control; the Canadian Hypertension Society; the Department of National Health and Welfare; the Department of Health and Fitness, Nova Scotia; the Medical Research Council of Canada; the Heart and Stroke Foundation of Canada; and the Nova Scotia Heart Foundation*

*Dr. Chockalingam was chairman of the conference organizing committee and is at the Department of Community Medicine, Faculty of Medicine, Memorial University of Newfoundland, St. John's. The panelists were Ms. Abbott, Foothills Hospital, Calgary; Dr. Battista, Division of Clinical Epidemiology, Montreal General Hospital; Dr. Cameron, Health Studies, University of Waterloo, Ont.; Dr. de Champlain, Département de physiologie, Université de Montréal; Dr. Evans, Department of Family Medicine, Faculty of Health Sciences, McMaster University, Hamilton, Ont.; Dr. Laidlaw (consensus panel chairman), Canadian Cancer Society, Toronto; Ms. Lee, The Hamilton Spectator, Hamilton, Ont.; Dr. Leiter, departments of Medicine and Nutritional Science, University of Toronto; Dr. Lessard, Cité de la Santé de Laval, Laval, PQ; Dr. MacLean, Atlantic Health Unit, Bedford, NS; Dr. Nishikawa, Department of Clinical Epidemiology and Biostatistics, Faculty of Health Sciences, McMaster University, Hamilton, Ont.; Dr. Rabkin, Department of Medicine, Faculty of Medicine, University of British Columbia, Vancouver; Ms. Thibaudeau, LaPresse, Montreal; and Ms. Strachan (consensus panel facilitator), Ottawa.*

*Reprint requests to: Dr. Arun Chockalingam, Faculty of Medicine, Memorial University of Newfoundland, St. John's, Nfld., Canada A1B 3V6*

## INTRODUCTION

This report presents the recommendations of a multidisciplinary consensus panel on efficacy of seven nonpharmacologic approaches to the prevention and treatment of high blood pressure. It is particularly important to explore the value of such approaches because pharmacologic measures for the control of hypertension, though effective, are fraught with side effects.

High blood pressure is only one factor, albeit a very important one, in the genesis of cardiovascular disease (CVD). Hence, the control of high blood pressure must be seen in the context of a multifactorial approach to the prevention and treatment of CVD. As emphasized in previous consensus conferences, measures to achieve cessation of tobacco consumption and reduction of blood cholesterol levels must also be put into action if CVD is to be controlled.

The lowering of blood pressure by most pharmacologic agents is accompanied in hypertensive people by a decrease in CVD-related morbidity and mortality largely due to a decrease in the occurrence of stroke. Some nonpharmacologic measures will lower blood pressure. However, their efficacy in decreasing CVD-related morbidity and mortality has not been established, despite strong epidemiologic evidence that some lifestyle factors play an important role in the development of CVD.

The following recommendations are divided into eight sections, each of which considers one of the seven nonpharmacologic approaches to the treatment of high blood pressure; the eighth deals with combined pharmacologic and nonpharmacologic approaches. Within each section are recommendations for people who are normotensive, for those with high blood pressure, to health professionals and on areas for future study. In addition the last part of each section discusses the evidence behind the specific recommendations.

Finally, in reviewing the present recommendations, the following points should be noted:

- "Normotensive" means having normal blood pressure (systolic  $\leq$  140 mm Hg, diastolic  $\leq$  90 mm Hg); "hypertensive" means having high blood pressure.

- The recommendations are developed for adults.

- People are considered to be at risk of hypertension if their blood pressure is in the upper part of the normal range or if there is a family history of hypertension. In most instances the recommendations for at-risk people are the same as those for people with normal blood pressure who are not at risk for hypertension.

- Though it is usually the physician who initi-

ates measures for controlling hypertension, not infrequently other health professionals are involved (e.g., nurses, dietitians and pharmacists). Hence, the term "health professional" is used to describe the people providing care.

- Blood pressure should be measured using the guidelines developed by the Canadian Coalition for High Blood Pressure Prevention and Control.<sup>1</sup>

## RECOMMENDATIONS

### Weight/obesity

#### *People who are normotensive*

There is considerable evidence that increased body weight is associated with an increased risk of high blood pressure. Those with upper body obesity are at even greater risk. Because there is also some evidence that weight loss may reduce the risk of high blood pressure, all adult Canadians should aim for a body mass index (BMI) of 20 to 27. This range is described as a "generally acceptable range" in the 1988 publication from the Department of National Health and Welfare, *Promoting Healthy Weights: a Discussion Paper*,<sup>2</sup> and is associated with other health benefits. BMI is an estimate of body fatness and is equal to weight in kilograms divided by height in metres squared ( $\text{kg}/\text{m}^2$ ).

#### *People with high blood pressure*

There is evidence that weight loss will reduce blood pressure in people with high blood pressure. Hence, all overweight people with high blood pressure should be advised to reduce their weight. Weight loss programs should consist of both caloric restriction and increased physical activity, which may have benefits of their own. Such people may require referral to a dietitian or an appropriate weight reduction program.

The degree of weight loss necessary to achieve blood pressure control may vary, and blood pressure may become normal in people whose weight is still elevated. Thus, the initial target of weight loss should be normalization of blood pressure. A secondary target would be to lower the BMI into the range outlined earlier.

#### *Health professionals*

Health professionals should inform their patients or clients about the relation between body weight and blood pressure and the possible benefits of weight reduction. It may be desirable to refer the patient or client for appropriate counselling or treatment or both. Health professionals should monitor

the involvement in and tolerance of the patient or client for the weight loss program.

Care should be taken to ensure that a blood pressure cuff of appropriate size is used for obese people.

### *Future study*

We recommend the following.

- That studies be undertaken of the blood pressure response to weight loss in certain subgroups (e.g., those with upper body obesity).

- That the prevalence of adverse effects of caloric restriction and of weight loss-gain cycles be investigated.

- That the effect of weight loss on the doses of antihypertensive medications required to control blood pressure be studied.

### *The evidence*

The association between high blood pressure and increasing body weight has been demonstrated in a large number of cross-sectional studies.<sup>3-8</sup> The correlations have ranged from 0.25 to 0.30, with relative risks for high blood pressure ranging from 2 to 5.<sup>9</sup> Furthermore, there is cross-sectional evidence that people with android or upper body obesity are at even greater risk.<sup>10</sup> Longitudinal studies have also shown a positive correlation between change in body weight and change in blood pressure.<sup>11-13</sup> Finally, it has also been demonstrated in randomized controlled trials of up to 3 years that weight loss will decrease blood pressure even in people with normal blood pressure.<sup>14,15</sup> Thus it can be concluded that a reduction of excessive body weight can help prevent high blood pressure.

Eight randomized controlled trials<sup>16-23</sup> as well as a longitudinal study<sup>24</sup> have examined the effects of weight loss on people with high blood pressure. Although the methods and amount of weight loss achieved vary from study to study, the reports virtually uniformly report a beneficial effect of weight reduction at 6 months to 1 year follow up. The one study with negative findings<sup>21</sup> achieved a relatively lesser degree of weight loss and used a different method of final blood pressure determination. Thus, it can also be concluded that a reduction of excessive body weight can also be of benefit in the treatment of high blood pressure. However, the magnitude of the required weight loss is variable and cannot be predicted in a given person.

In the absence of fanges of desirable weight in the literature, specifically for blood pressure, we chose a range consistent with recent guidelines published by the Department of National Health and Welfare.<sup>2</sup>

## **Salt (sodium)**

For the purposes of this report the word salt, when appearing alone, is understood to include sodium unless otherwise indicated.

### *People who are normotensive*

Because the amount of salt presently in the Canadian diet exceeds individual needs, no harm has been associated with moderate salt reduction and there is evidence that salt intake is modestly correlated with high blood pressure in the general population, we recommend that people reduce their salt intake in the following ways.

- Choosing foods low in salt (e.g., fresh fruits and vegetables).

- Avoiding foods high in salt (e.g., processed meats, canned foods and snack foods).

- Not adding salt at the table and minimizing the amount of salt used in cooking.

These recommendations are especially important for those at risk of hypertension.

### *People with high blood pressure*

There is evidence that salt restriction can reduce blood pressure in some patients or clients. For patients or clients with a diastolic blood pressure between 90 and 99 mm Hg but without the complications of high blood pressure, salt reduction may be used as an initial therapeutic measure along with other nonpharmacologic measures as appropriate.

It has been shown that salt reduction can add to the blood-pressure-lowering effect of most drugs used in the treatment of high blood pressure. Hence, salt reduction in combination with drug therapy is advised for all other patients or clients with hypertension.

### *Health professionals*

Health professionals need to counsel their patients or clients on the relation between salt and blood pressure and provide them with information on the salt content of foods.

### *Government*

The government should require the labelling of all foods with their salt and sodium content.

### *The food industry*

The food industry should reduce its use of salt and sodium in food, especially in staples such as bread, and it should actively market low-salt foods.

## *Future study*

We recommend the following.

- That research on sodium sensitivity be conducted in humans.
- That long-term intervention studies on the effects of various levels of sodium restriction on blood pressure be undertaken.
- That basic research be conducted on the effects of dietary sodium and potassium on cardiovascular reactivity and function.
- That a database be developed that describes Canadian salt and sodium consumption and that a mechanism be created for monitoring this parameter.

## *The evidence*

Salt was the first environmental factor associated with high blood pressure. This association was made on the basis of several cross-sectional studies that suggested a direct relation between high blood pressure and sodium intake within, as well as across, populations. Previous recommendations to reduce dietary sodium intake in hypertensive patients came from numerous intervention studies carried out with small numbers of patients that suggested that sodium restriction tended to lower blood pressure and accentuated the hypotensive effects of most drugs in patients with high blood pressure. However, cross-sectional and intervention studies, including controlled studies of communities, have sometimes shown contradictory results.<sup>25-32</sup>

Two, more methodologically sound, studies have recently been published: the Intersalt Study<sup>8</sup> and the Australian National Health and Medical Research Council Dietary Salt Study.<sup>33</sup> These projects involved larger numbers of subjects than previous studies, and in both cases special care was taken to dissociate sodium effects from the influences of possible confounding factors such as potassium, weight loss and age. Overall, these studies support a causal relation between sodium intake and blood pressure levels. However, the strength of that relation appears to be weaker than that suspected from earlier data.

The Intersalt Study<sup>8</sup> cross-sectionally observed patients from 52 world-wide centres. After adjustment for other variables, urinary sodium excretion was found to be significantly correlated with systolic blood pressure in only 8 centres but was highly significantly correlated across the 52 centres; this correlation remained significant after patients with high blood pressure were removed from the analysis (Dr. Paul Elliott: personal communication, 1989).

The Australian Dietary Salt Study<sup>33</sup> was a randomized controlled trial of sodium versus placebo

supplementation of a low sodium diet in 111 untreated subjects with high blood pressure. The moderate sodium restriction attained led to significant reductions in both systolic and diastolic blood pressure on average. Individual responses varied markedly, being larger in older patients and in those with higher systolic blood pressure during the run-in period.

There is no evidence that moderate lowering of our present estimated sodium intake is harmful to most of the general population. This information, along with the results of recent studies and the possibility of high sensitivity to sodium in subgroups, led to the recommendation for a decrease in sodium intake for the general population. Because of the variations in individual habitual sodium intake and possible variation in individual sodium sensitivity, specific target intakes were not defined.

## **Alcohol**

### *People who are normotensive*

There is good evidence of a causal relation between excessive alcohol consumption and high blood pressure. Furthermore, it has been demonstrated that a reduction in alcohol intake leads to a lowering of blood pressure in normotensive people and those with high blood pressure. Consequently, the public is advised to avoid excessive intake of beer, wine and spirits. Excessive alcohol intake is defined as more than two standard drinks (20 to 30 g of alcohol) per day. Excessive alcohol intake includes binge drinking, which may be associated with an elevated risk of stroke. A standard drink is 120 ml (4 oz) of wine, 30 ml (1 oz) of liquor and 360 ml (12 oz) of beer.

This recommendation is especially important for those at high risk of hypertension.

### *People with high blood pressure*

In view of the evidence described above, hypertensive people should also avoid excessive alcohol intake. If blood pressure is still not controlled, abstinence may be helpful.

### *Health professionals*

When dealing with hypertensive patients or clients or those at risk of high blood pressure health professionals need to become skilled in the most effective methods of inquiring about alcohol consumption. Furthermore, they should provide advice on the possible effects of excessive alcohol consumption as well as on the means of reducing it. Community-based programs may be helpful.

## Government

The government should consider limiting the advertising of alcoholic beverages and the labelling of alcoholic beverages with a warning about their effects on blood pressure.

### Future study

We recommend the following.

- That research be conducted into the mechanism(s) whereby alcohol intake increases blood pressure.
- That research is needed to determine whether there are differences in the degree to which consumption of different alcoholic beverages increases blood pressure.
- That further research is necessary to determine whether the claim that CVD-related morbidity and mortality are higher at low to zero levels of alcohol intake than at moderate levels of consumption is valid.

### The evidence

There is substantial evidence confirming a causal association between high blood pressure and excessive alcohol consumption. The relation between alcohol consumption and elevated blood pressure has been demonstrated in nearly 40 cross-sectional studies from many countries around the world.<sup>34</sup> Some of these studies have been very large (over 80 000 subjects) and have demonstrated that the effect of alcohol was independent of several factors: age, obesity, cigarette smoking, exercise, education, tea and coffee consumption, and personality type.<sup>35-47</sup>

Direct evidence of the effect of alcohol on blood pressure comes from a number of cross-over trials that demonstrate that the addition and subtraction of alcohol from the diet can influence blood pressure in a predictable manner in patients with both normal and high blood pressure.<sup>48-51</sup> Clear, dose-response relations with considerable individual sensitivity to the pressor effects of alcohol have been demonstrated.

On a population basis the contribution of alcohol to hypertension has been estimated to range from 11%<sup>38</sup> to 20%.<sup>51</sup> The evidence reviewed here shows a remarkably consistent relation between alcohol consumption and blood pressure levels. Likewise, in longitudinal studies drinkers have been shown to be at greater risk for hypertensive CVD,<sup>52,53</sup> thus reinforcing the evidence that alcohol consumption predisposes to the development of hypertension. Binge drinking has also been associated with stroke in young men.<sup>54,55</sup>

## Physical exercise

### People who are normotensive

Though there are epidemiologic data that indicate that hypertension and CVD are less likely to develop in fit people, we know of no prospective controlled studies that demonstrate that physical activity prevents high blood pressure. None the less, regular physical activity appropriate to the person's capabilities seems prudent because of its value in weight regulation and its potential benefit in reducing CVD-related mortality.

### People with high blood pressure

People with high blood pressure should consult their physician before they undertake strenuous exercise. Appropriate physical activity is a useful component of weight management in the control of high blood pressure. Although there is evidence that regular aerobic activity may result in the lowering of blood pressure in patients or clients with mild hypertension, definitive recommendations must await further research to determine the intensity, frequency and duration of the activity required to lower blood pressure and to determine how long the benefits can be maintained. Activities such as weight-lifting are contraindicated in hypertensive patients or clients.

### Health professionals

Health professionals should encourage regular, appropriate physical activities, particularly those that can be incorporated into daily living, such as walking and stair-climbing. Health professionals who do not feel confident about prescribing such programs should refer their patients or clients to appropriate community resources.

### Future study

We recommend the following.

- That research be conducted to evaluate the benefits and risks of physical activity in the management of hypertension.
- That the intensity, frequency and duration of exercise required to lower blood pressure be investigated.
- That studies be undertaken to determine how long any exercise-induced reduction in blood pressure may be sustained.

### The evidence

There is epidemiologic evidence that people

who are physically active are at lower risk of coronary heart disease. A review of 43 longitudinal nonintervention studies suggested that the relative risk for physical inactivity as an independent factor for coronary heart disease ranges from 1.5 to 2.4 with a median of 1.9.<sup>56</sup> Physical inactivity is common, although estimates of prevalence vary widely.<sup>57</sup> A review of eight national surveys conducted in Canada and the United States suggests that approximately 20% of the population exercises at a level recommended for cardiovascular fitness and that 40% is virtually sedentary.<sup>57</sup>

There is also evidence from longitudinal studies indicating that regular exercise and physical fitness may be associated with a lower risk of hypertension.<sup>58</sup> For instance, Paffenbarger and colleagues<sup>13</sup> found that among 14 998 Harvard alumni men followed up for 6 to 10 years regular strenuous physical activity was related inversely to the risk of hypertension. A 12-year follow-up of more than 6000 men and women indicated that, when compared with those who were highly fit, the less fit people had a relative risk of hypertension of 1.52 after adjustments were made for sex, age and follow-up interval.<sup>59</sup> Data from the Framingham Offspring Study revealed a negative association between systolic blood pressure and exercise endurance that was independent of age, resting heart rate and BMI among men.<sup>60</sup> These correlation data lend plausibility to the notion that physical activity may have a salutary effect on blood pressure. There have been no controlled intervention studies demonstrating that physical activity prevents the development of high blood pressure.

There is some evidence that regular aerobic activity may lower blood pressure.<sup>58 61</sup> Martin and Dubbert<sup>62</sup> reviewed the effects of aerobic exercise on blood pressure and hypertension and found that in seven uncontrolled studies of 183 participants there was a reduction in mean levels of 19 mm Hg in systolic blood pressure and 14 mm Hg in diastolic blood pressure. Three partially controlled studies of 86 people showed a reduction in mean levels of 15 mm Hg in systolic blood pressure and 9 mm Hg in diastolic blood pressure. Six better controlled studies (including parallel group, randomized and nonrandomized controlled, and before-and-after intervention designs) of 65 hypertensive "exercised" people revealed a reduction in weighted mean levels of 8 mm Hg in systolic blood pressure and 6 mm Hg in diastolic blood pressure. Martin and Dubbert<sup>62</sup> noted that in these studies weight and sodium excretion were rarely monitored to isolate the independent effects of exercise. They also noted that in several of the better controlled studies untreated hypertensive controls showed lower blood pressures over time. Thus, aerobic activity may lower blood pressure

among hypertensive people, although more tightly controlled trials of longer duration are required before this conclusion can be made with confidence.

Some forms of exercise, particularly that involving explosive movements and isolation of single muscle groups, may be harmful to those with high blood pressure.<sup>63</sup>

## Calcium

### *People who are normotensive*

Although there is some evidence that low calcium intake is related to high blood pressure, it is inconclusive. Thus, we cannot recommend supplementation of calcium intake for the prevention of hypertension.

### *People with high blood pressure and health professionals*

Because the data on the relation between calcium intake and blood pressure are unclear and the evidence that calcium supplementation can lower the risk of hypertension is inconclusive, we do not recommend calcium supplementation for hypertensive people.

### *Future study*

We recommend the following.

- That research be conducted on the influence of calcium metabolism on the genesis of hypertension, particularly to explain the apparent discordant results between the epidemiologic association of low calcium intake and high blood pressure on the one hand and the association of high serum calcium levels and hypertension (e.g., in hyperparathyroidism) on the other.

- That research be done on the interrelations of dietary calcium, sodium, potassium and magnesium as they influence blood pressure.

### *The evidence*

Cross-sectional data have suggested that an inverse relation exists between dietary calcium intake and blood pressure such that low levels of calcium intake are associated with high blood pressure.<sup>6,64 67</sup> The difficulty with the interpretation of these data is that often other factors were not taken into account. After considering factors such as age, body weight and alcohol consumption, calcium intake was not always a significant factor in influencing blood pressure.<sup>68</sup> Some population studies have been unable to separate the effects of calcium from other dietary elements. One epidemiologic study conclud-

ed that it was "not possible to ascribe a causal relationship between calcium and blood pressure, however, due to the intricate network of covarying food intakes and the possible role that unmeasured social and cultural factors may play in the observed relations".<sup>67</sup> Another epidemiologic study that showed a significant univariate inverse association between reported calcium intake and blood pressure concluded that the high degree of intercorrelation among dietary factors "indicates that the independent role of any specific nutrient cannot be conclusively separated from the possible effects of other nutrients in this type of study".<sup>66</sup> Thus, the epidemiologic data are suggestive but not conclusive of a causal association between calcium intake and blood pressure.

The relation of calcium intake to blood pressure has been the subject of several randomized clinical trials.<sup>69-85</sup> These studies of calcium supplementation have had either a cross-over or a parallel-group design. Most were small (all of them with fewer than 100 patients and 14 with fewer than 50), short (the longest being 4 years and 16 being less than 1/2 year) and involved varying amounts of calcium supplementation and subjects with both normal and high blood pressure. The results of these 19 clinical trials showed conflicting results, with some indicating a significant decrease in blood pressure while others showed no significant change. A meta-analysis with the results of these randomized trials showed a decrease in systolic blood pressure of 1.5 mm Hg with calcium supplementation of 0.4 to 2.0 g/d. The meta-analysis did not separately analyse trials in people with high and normal blood pressure and included two abstracts (Dr. Jeffrey Cutler: personal communication, 1989). The results of long-term studies with larger sample sizes are awaited with interest because the inconsistent results of present trials do not provide strong supporting evidence for conclusive recommendations.

Epidemiologic and randomized controlled study data would be strengthened if a causal association made biologic sense. The low calcium intake-high blood pressure model does not readily explain the observation of hypertension with hypercalcemia, the efficacy of the calcium antagonist drugs in treating high blood pressure or the relation between calcium intake and other neurohumoral factors modulating blood pressure.

## Potassium

### *People who are normotensive*

There is evidence that populations that consume high-potassium foods have lower blood pressure. Though this relation has not been shown to be

causal, we recommend that people eat a diet rich in potassium (e.g., fresh/frozen fruits and vegetables, particularly potatoes, and low-fat dairy products). Such a diet clearly has other health benefits.

### *People with high blood pressure*

Short-term studies have shown that potassium-rich diets lower blood pressure in hypertensive patients or clients. Although a sustained effect has not yet been demonstrated, we recommend that these patients follow such a diet.

A potassium-rich diet in conjunction with a low-sodium diet is especially recommended for patients or clients taking thiazide diuretics, which tend to deplete body potassium.

### *Health professionals*

Clear information on potassium-rich foods and their preparation (e.g., steaming rather than boiling) should be provided to patients or clients.

### *Future study*

We recommend the following.

- That the potential protective effect of potassium on the development of hypertension and stroke should be explored.
- That long-term studies on the effect of increased dietary potassium on hypertension be conducted.

### *The evidence*

Support for recommending the maintenance of a potassium-rich diet for people with either normal or high blood pressure comes from population studies, clinical trials and experiments with animals.

Because people eat foods rather than nutrients, it is not surprising that potassium intake is closely linked with a variety of other nutrients. Generally, it has been found that populations with low potassium intake also have low fibre and calcium intake and high sodium and alcohol intake. Recent cross-sectional studies generally have shown an independent inverse relation between potassium intake (or urine excretion of potassium) and blood pressure.<sup>8,66,86,87</sup> In most studies the sodium:potassium ratio is also similarly correlated with blood pressure.

Experiments with hypertensive rats have shown that the administration of supplemental dietary potassium lowers blood pressure in these animals.<sup>88,89</sup>

Several randomized<sup>90-92</sup> trials and a nonrandomized<sup>93</sup> controlled trial of potassium supplementation (60 to 120 mmol/d) showed significant reductions in blood pressure of 2 to 11 mm Hg in patients

with high and normal blood pressure. These were all been short-term studies. As well, there is concern over the side effects of potassium supplementation in patients with renal disease and in those taking potassium-sparing diuretics or angiotensin-converting enzyme inhibitors. For these reasons it is premature to consider the use of potassium supplements in lowering blood pressure. On the other hand, hyperkalemia is not a concern with potassium in the diet. One randomized trial showed that an increase in dietary potassium had a blood-pressure-lowering effect comparable to that achieved by a reduction in dietary sodium.<sup>94</sup>

Potassium supplements also have been reported to reduce the incidence of stroke in rats.<sup>95</sup> A similar protective effect has been claimed in humans.<sup>96</sup>

Though it is difficult to make major changes in potassium intake by dietary means, it is estimated that a 10-mmol/d increase in potassium could yield a decrease of 1 to 2 mm Hg in mean systolic blood pressure.

## Relaxation/stress management

### *People who are normotensive*

It is widely believed that stress contributes to high blood pressure. Although it is well known that short-term stress will produce a temporary rise in blood pressure, there is little information on the role of chronic stress in the development of chronic high blood pressure. There is no convincing evidence that relaxation/stress management reduces the probability of hypertension. Hence, at present we cannot recommend that people participate in such training programs to prevent hypertension.

### *People with high blood pressure*

Studies of the impact of relaxation/stress management techniques in hypertensive patients or clients have yielded both positive and negative results. Hence, it is premature to recommend these techniques in the treatment of high blood pressure.

### *Health professionals*

Although the value of relaxation/stress management techniques in patients or clients with high blood pressure is uncertain, health professionals should be aware of and responsive to stress-related problems as they are perceived by such patients or clients.

### *Future study*

We recommend the following.

- That research be conducted to explore possible links between psychologic and social factors and blood pressure, particularly to determine whether such factors play a role in the maintenance of long-term hypertension.

- That long-term, large-scale, multidisciplinary clinical trials be performed to evaluate the efficacy of relaxation/stress management in the lowering of blood pressure.

### *The evidence*

It has been suggested that chronic stress leads to a persistent elevation in blood pressure even after the stress has passed. Studies in animals have provided evidence to support this hypothesis.<sup>97</sup> Cross-sectional studies in humans have suggested a link between increases in blood pressure and crowding<sup>98</sup> and vocational responsibility.<sup>99</sup> A longitudinal study of blood pressure showed a similar link with unemployment.<sup>100</sup> There was no evidence that any form of relaxation training or stress management protects against the development of hypertension.

Several distinct stress management and relaxation interventions have been used in attempts to lower blood pressure: progressive muscle relaxation, relaxation response training, autogenic training, and blood pressure, thermal and electromyographic biofeedback. Blanchard and associates<sup>101</sup> have provided detailed descriptions of these interventions and reviewed controlled trials in which they have been evaluated. Other intervention programs have used more multifaceted and comprehensive intervention strategies.<sup>102</sup> The results obtained with most of these interventions have been disappointing.<sup>101</sup> The best results have been with progressive relaxation training and the comprehensive approach taken by Patel and Marmot.<sup>102</sup> However, even these positive results are sometimes difficult to interpret with confidence. For instance, in an evaluation of progressive relaxation training, Southam and coworkers<sup>103</sup> found that after 8 weeks progressive relaxation training lowered both systolic and diastolic blood pressure (by 7.8 and 4.6 mm Hg, respectively) at the work site. Diastolic blood pressure was lowered (by 12.6 mm Hg) in the clinic. At a 15-month follow-up evaluation<sup>104</sup> the mean diastolic blood pressure of the relaxation subjects was still significantly lower at the clinic (by 13.8 mm Hg) and at the worksite (by 7.4 mm Hg). However, at this point there was no significant difference between treated and control subjects.

A recent multicentre randomized study found that the same treatment and research protocol as that used above led to inconsistent findings across settings.<sup>105</sup>

Given that controlled trials have resulted in inconsistent findings and that positive findings are



sometimes difficult to interpret with confidence the proposition that stress management and relaxation training are clinically useful for reducing blood pressure remains controversial.

## **Combination of pharmacologic and nonpharmacologic measures in the treatment of high blood pressure**

### *Health professionals*

Although some nonpharmacologic approaches can reduce blood pressure in hypertensive people, their effectiveness varies. Furthermore, the magnitude of the reduction achieved with each intervention is small compared with that observed with pharmacologic therapy. Hence, exclusive reliance on nonpharmacologic measures as initial therapy is recommended only for hypertensive patients with a diastolic blood pressure of less than 100 mm Hg and no evidence of complications of hypertension (e.g., target organ damage).

There is good evidence that some nonpharmacologic approaches (e.g., weight loss and sodium reduction) will add to the effectiveness of antihypertensive medication.

In some patients or clients who are well controlled on medication one can introduce nonpharmacologic measures and then judiciously attempt to taper the antihypertensive medications. Furthermore, there is evidence that a combination of nonpharmacologic measures may allow discontinuation of antihypertensive medication in some patients or clients; however, such patients or clients should continue to be monitored carefully.

Many of the nutrients previously discussed in isolation are, in fact, linked when one makes dietary recommendations. For example, a high-potassium diet will tend to be low in sodium and high in fibre. Similarly, restriction of energy intake will often reduce sodium intake.

### *Future study*

We recommend the following.

- That the interactions of pharmacologic and nonpharmacologic measures in the treatment of hypertension be explored further.
- That further research be conducted on how to improve compliance with nonpharmacologic measures.
- That the effect of nonpharmacologic measures on compliance with pharmacologic agents be studied.

### *The evidence*

Studies assessing the combination of nonphar-

macologic management of high blood pressure with antihypertensive drugs go back a number of years. Parijs and collaborators,<sup>106</sup> in a nonrandomized crossover study in 1973, showed that diuretics had a greater effect than salt restriction on reducing blood pressure, but a combination of the two was still more effective than either alone. Ram and colleagues<sup>107</sup> also found a greater reduction in blood pressure when moderate sodium restriction was combined with diuretics than when diuretics were given with a high-sodium diet. Both studies were short and had small numbers of subjects.

Erwteman and associates,<sup>108</sup> in another short-term randomized crossover trial, demonstrated that salt restriction increased the antihypertensive effect of  $\beta$ -blockers and of  $\beta$ -blockers combined with diuretics. They also confirmed that salt restriction enhanced the reduction in blood pressure produced by diuretics.

A nonrandomized controlled Japanese study, by Fujita and coworkers,<sup>109</sup> showed that the antihypertensive effect of an angiotensin-converting enzyme inhibitor, captopril, was greater when sodium was restricted, at least in the short term. The effect of sodium restriction on calcium channel blockers, however, is less clear, and there is some evidence that they may be more effective in the presence of increased sodium intake.<sup>110</sup>

In the classic 1978 study by Reisin and collaborators<sup>20</sup> weight reduction had significantly decreased blood pressure at 6 months' follow up in patients with high blood pressure who were inadequately controlled with antihypertensive medication compared with a similar group that did not participate in a weight reduction program.

Randomized<sup>20,111-113</sup> and nonrandomized<sup>114</sup> controlled studies have demonstrated that nutritional interventions such as weight reduction, sodium restriction and reduction of alcohol intake can reduce or even eliminate the amount of medication required to control high blood pressure. The most convincing of these studies, by Stamler and colleagues,<sup>111</sup> demonstrated that 39% of a group of previously treated hypertensive patients were able to stay off medication for as long as 4 years, compared with 5% of a group without nutritional treatment.

## **PRIORITIES**

### **Health professionals and their patients or clients**

Health professionals should first provide their patients or clients with information on the benefits and risks of appropriate nonpharmacologic measures for the prevention and treatment of high blood pressure.

We suggest that the measures considered in this report receive the following priority.

- Reduction of alcohol intake is probably the most potent of such approaches, although implementation may be difficult.

- Weight loss appears to be the next most efficacious measure, and, though the effect of physical activity on hypertension is less clear, physical activity is a useful component of weight reduction programs.

- A reduction in dietary salt intake and an increase in dietary potassium intake can be of value, particularly in patients or clients taking certain antihypertensive medications.

The specific mix and sequence of these approaches should be decided by the patients or clients in partnership with the health professional.

In addition to these recommendations, as em-

phasized in previous consensus conferences, smoking cessation and the adoption of measures to reduce total blood cholesterol must be encouraged.

This conference was supported by Astra Pharma Inc.; Boehringer Ingelheim (Canada) Ltd.; Boehringer Mannheim Canada; Ciba-Geigy Canada; Dairy Bureau of Canada; G.D. Searle & Co. of Canada Limited; Glaxo Canada Inc.; Hoffmann-La Roche Limited; I.C.I. Pharma Canada; Manufacturers Life Insurance Company; Merck Frosst Canada Inc.; Miles Canada Inc.; Nordic Laboratories Inc.; North American Life Insurance Company; Parke-Davis, a Division of Warner-Lambert Canada Inc.; Pfizer Canada Inc.; Produits pharmaceutiques Key. Division cardiovasculaire de Schering Canada Inc; Rhône-Poulenc Pharma Inc.; Rorer Canada Inc.; Sandoz Canada; Smith Kline & French Canada Ltd.; Squibb Pharmaceutical Products; and Syntex Inc. Canada.

## CONFERENCE CONTRIBUTORS

**Organizing Committee:** Dr. Arun Chockalingam (chmn), Drs. Tim Dean, George Fodor, Pavel Hamet, Brian Haynes, Pierre Larochelle, Yves Lacourciere, Frans Leenen, John McKenzie, Brian O'Connor and Andrés Petrasovits, Ms. Gloria Sacks-Silver, Mr. Erskine Simons, Dr. Sylvie Stachenko and Dr. Thomas Wilson

**Scientific Program Committee:** Drs. Pavel Hamet and George Fodor (cochmn), Drs. Arun Chockalingam, Brian Haynes, Pierre Larochelle, Brian O'Connor and Sylvie Stachenko

**Local Organizing Committee:** Dr. Tim Dean (chmn), Drs. Arun Chockalingam, George Fodor and Pavel Hamet and Mr. John Sutherland

**Public Relations Committee:** Dr. Brian O'Connor (chmn), Drs. Arun Chockalingam, Pavel Hamet and Pierre Larochelle, Ms. Gloria Sacks-Silver and Dr. Thomas Wilson

**Secretariat:** Mr. John Sutherland, A.B. Thompson Associates Ltd., Halifax

## SPEAKERS

*Epidemiology of Cardiovascular Diseases. Previous Consensus Conference:* **Frans Leenen**, MD, PhD, FRCPC, University of Ottawa Heart Institute, Ottawa

*Cardiovascular Disease — World-wide View:* **Lennart Hansson**, MD, Department of Medicine, Goteborgs Universitet, Goteborg, Sweden

*High Blood Pressure in the Context of Cardiovascular Diseases — Canadian Perspectives:* **Veronique Dery**, MD, Département de Santé, Cité de la Santé de Laval, Laval, PQ

*Healthy Weights:* **Heather Nielson**, PhD, Department of Nutrition, Department of National Health and Welfare, Ottawa

*Obesity and High Blood Pressure:* **Robert W. Jeffrey**, PhD, Division of Epidemiology, School of Public Health, University of Minnesota, Minneapolis, MN; **Brian Haynes**, MD, PhD, FRCPC, Department of Clinical Epidemiology and Biostatistics, Faculty of Health Sciences, McMaster University, Hamilton, Ont.

*Salt and High Blood Pressure:* **Paul Elliott**, MB, BS, MSc, MRCP, London School of Hygiene and Tropical Medicine, London, England; **Alexander G. Logan**, MD, MSc, FRCPC, Department of Nephrology, Mount Sinai Hospital, Toronto

*Alcohol and High Blood Pressure:* **Lawrence J. Beilin**, MB, BS, MD, FRCP, Department of Medicine, University of Western Australia, Perth

*Physical Exercise and High Blood Pressure:* **Herbert Langford**, MD, FACP, Department of Medicine, University of Mississippi Medical Center, Jackson, MI; **Patrick O'Sullivan**, MA, BPHE, Cardiovascular Disease Prevention and Rehabilitation Centre, Sudbury Memorial Hospital, Sudbury, Ont.

*Calcium and High Blood Pressure:* **Jeffrey Cutler**, MD, National Institutes of Health, Bethesda, MD; **Pavel Hamet**, MD, PhD, FRCPC, Clinical Research Institute of Montreal, Montreal

*Potassium and High Blood Pressure:* **Lawrence J. Beilin**, MB, BS, MD, FRCP, Department of Medicine, University of Western Australia, Perth; **J. George Fodor**, MD, PhD, FRCPC, Department of Community Medicine, Faculty of Medicine, Memorial University of Newfoundland, St. John's

*Relaxation/Stress Management and High Blood Pressure:* **Nemat Borhani**, MD, MPH, FACP, Department of Community Health, University of California, Davis, CA; **Anthony Bellissimo**, MD, MASc, PhD, Department of Psychiatry, Faculty of Health Sciences, McMaster University, Hamilton, Ont.

*Combination of Pharmacological and Non-Pharmacological Management in Treating and Controlling High Blood Pressure:* **Lennart Hansson**, MD, Department of Medicine, Goteborgs Universitet, Goteborg, Sweden

## REFERENCES

- Canadian Coalition for High Blood Pressure Prevention and Control: Recommendations for obtaining accurate blood pressure measurement: a working group report. In *Know Your Blood Pressure by Heart, Professional Guide*, Heart and Stroke Foundation of Canada, Ottawa, 1987: 3-6
- Health Services and Promotion Branch, Department of National Health and Welfare: *Promoting Healthy Weights: a Discussion Paper* (cat no H39-131/1988E), Dept of Supply and Services, Ottawa, 1988
- Chiang BN, Perlman LV, Epstein RH: Overweight and hypertension: a review. *Circulation* 1969; 39: 403-421
- National Institutes of Health Consensus, Development Panel on the Health Implication of Obesity: The health implication of obesity. *Ann Intern Med* 1985; 103: 1073-1077
- Blackburn H, Prineas R: Diet and hypertension: anthropology, epidemiology and public health implications. In Paoletti R (ed): *Progress in Biochemical Pharmacology*, Karger, Basel, 1983: 31-79
- Harlan WR, Hull AR, Schmouder RL et al: Blood pressure and nutrition in adults: the National Health and Nutrition Examination Survey. *Am J Epidemiol* 1984; 120: 17-28
- Epstein FH, Francis T, Hayner NS et al: Prevalence of chronic disease and distribution of selected physiologic variables in a total community, Tecumseh, Michigan. *Am J Epidemiol* 1965; 81: 307-322
- Intersalt Cooperative Research Group. An international study of electrolyte excretion and blood pressure: results for 24 hour urinary sodium and potassium excretion. *Br Med J* 1988; 297: 319-328
- Van Itallie TB: Health implication of overweight and obesity in the United States. *Ann Intern Med* 1985; 103: 983-988
- Gillum RF: The association of body fat distribution with hypertension, hypertensive heart disease, coronary heart disease, diabetes and cardiovascular risk factors in men and women aged 18-79 years. *J Chronic Dis* 1987; 40: 421-428
- Borkan GA, Sparrow D, Wisniewski C et al: Body weight and coronary disease risk: patterns of risk factors change associated with long-term weight change. *Am J Epidemiol* 1986; 124: 410-419
- Selby JV, Freidman GD, Quesenberry CP: Precursors of essential hypertension: the role of body fat distribution pattern. *Am J Epidemiol* 1989; 129: 43-53
- Paffenbarger RS, Wing AL, Hyde RT et al: Physical activity and incidence of hypertension in college alumni. *Am J Epidemiol* 1983; 117: 245-257
- Oberman A: Hypertension prevention trial: three-year effects of dietary changes on blood pressure. Presented at the American Heart Association, November 1988, Washington, DC
- Fortmann SP, Haskell WL, Wood PD et al: Effects of weight loss on clinic and ambulatory blood pressure in normotensive men. *Am J Cardiol* 1988; 62: 89-93
- MacMahon S, Cutler J, Brittain E et al: Obesity and hypertension: epidemiological and clinical issues. *Eur Heart J* 1987; 8 (suppl B): 57-70
- Jacob RG, Wing R, Shapiro AP: The behavioral treatment of hypertension: long-term effects. *Behav Ther* 1987; 18: 325-352
- Heyden S, Tyroler HA, Hames CG et al: Diet treatment of obese hypertensives. *Clin Sci Mol Med* 1973; 45: 209-212
- Ramsay LE, Ramsay MH, Hettiarachchi J et al: Weight reduction in a blood pressure clinic. *Br Med J* 1978; 2: 244-245
- Reisin E, Abel R, Modan M et al: Effect of weight loss without salt restriction on the reduction of blood pressure in overweight hypertensive patients. *N Engl J Med* 1978; 298: 1-6
- Haynes RB, Harper AC, Costley SR et al: Failure of weight reduction to reduce mildly elevated blood pressure: a randomized trial. *J Hypertens* 1984; 2: 535-539
- MacMahon S, MacDonald WGJ, Bernstein L et al: Comparison of weight reduction with metoprolol in treatment of hypertension in young overweight patients. *Lancet* 1985; 1: 1233-1236
- Croft PR, Brigg D, Smith S et al: How useful is weight reduction in the management of hypertension? *J Coll Gen Pract* 1986; 36: 445-458
- Heyden S, Borhani NO, Tyroler HA et al: The relationship of weight change to changes in blood pressure, serum uric acid, cholesterol and glucose in the treatment of hypertension. *J Chronic Dis* 1985; 38: 281-288
- Simpson FO: Salt and hypertension: a sceptical review of the evidence. *Clin Sci* 1979; 57: 463s-480s
- MacGregor GA: Sodium is more important than calcium in essential hypertension. *Hypertension* 1985; 7: 628-637
- Denton D: *The Hunger for Salt: an Anthropological, Physiological and Medical Analysis*, Springer-Verlag, Berlin, 1982: 542-629
- Laragh JH, Pecker MS: Dietary sodium and essential hypertension: some myths, hopes and truths. *Ann Intern Med* 1983; 98: 735-743
- Staessen J, Bulpitt CJ, Fagard R et al: Salt intake and blood pressure in the general population: a controlled intervention trial in two towns. *J Hypertens* 1988; 6: 965-973
- Rose G: Clinical trials and medical practice. In Mathias CJ, Sever PS (eds): *Concepts in Hypertension. Festschrift for Sir Stanley Peart*, Springer-Verlag, New York, 1988: 39-44
- Grobbbee DE, Hofman A: Does sodium restriction lower blood pressure? *Br Med J* 1986; 293: 27-29
- Logan AG: Sodium manipulation in the management of hypertension: the view against its general use. *Can J Physiol Pharmacol* 1986; 64: 793-802
- Australian National Health and Medical Research Council, Dietary Salt Study Management Committee. Fall in blood pressure with modest reduction in dietary salt intake in mild hypertension. *Lancet* 1989; 1: 399-402
- Beilin LJ, Puddey IB, Vandongen R: Alcohol-related hypertension. In Strasser T, Ganten D (eds): *Mild Hypertension: From Drug Trials to Practice*, Raven, New York, 1987: 147-156
- Klatsky AL, Friedman GD, Siegelau AB et al: Alcohol consumption and blood pressure: Kaiser-Permanente multiphasic health examination data. *N Engl J Med* 1977; 296: 1194-1200
- Arkwright PD, Beilin LJ, Rouse I et al: Effects of alcohol use and other aspects of lifestyle on blood pressure levels and prevalence of hypertension in a working population. *Circulation* 1982; 66: 60-66
- Cairns V, Keil U, Kleinbaum D et al: Alcohol consumption as a risk factor for high blood pressure: Munich blood pressure study. *Hypertension* 1984; 6: 124-131
- MacMahon SW, Blacket RB, MacDonald GJ et al: Obesity, alcohol consumption and blood pressure in Australian men and women: the National Heart Foundation of Australia Risk Factor Prevalence Study. *J Hypertens* 1984; 2: 85-91
- Mitchell PI, Morgan MJ, Boadle DJ et al: Role of alcohol in the aetiology of hypertension. *Med J Aust* 1980; 2: 198-200
- Salonen JT, Tuomilehto J, Tanskanen A: Relation of blood pressure to reported intake of salt, saturated fats and alcohol in healthy middle-aged population. *J Epidemiol Community Health* 1983; 37: 32-37
- Criqui MH, Wallace RB, Mishkel M et al: Alcohol consumption and blood pressure: the Lipid Research Clinics Prevalence Study. *Hypertension* 1981; 3: 557-565
- Milon H, Froment A, Gaspard J et al: Alcohol consumption and blood pressure in a French epidemiologic study. *Eur Heart J* 1982; 3 (suppl C): 59-64
- Ueshima H, Shimamoto T, Iida M et al: Alcohol intake and

- hypertension among urban and rural Japanese populations. *J Chronic Dis* 1984; 37: 585-592
44. Dyer AR, Stamler J, Paul O et al: Alcohol, cardiovascular risk factors and mortality: the Chicago experience. *Circulation* 64 (3 part 2): 20-27
  45. Gordon T, Kannel WB: Drinking and its relation to smoking, BP, blood lipids and uric acid. *Arch Intern Med* 1983; 143: 1366-1374
  46. Saunders JB, Beevers DG, Paton A: Alcohol induced hypertension. *Lancet* 1981; 2: 653-656
  47. Potter JF, Beevers DG: Pressor effect of alcohol in hypertension. *Lancet* 1984; 1: 119-122
  48. Puddey IB, Beilin LJ, Vandongen R et al: A randomized control trial of the effect of alcohol consumption on blood pressure. *Clin Exp Pharmacol Physiol* 1985; 12: 257-261
  49. Puddey IB, Beilin LJ, Vandongen R et al: Evidence for a direct effect of alcohol consumption on blood pressure in normotensive men. *Hypertension* 1985; 7: 707-713
  50. Puddey IB, Vandongen R, Beilin LJ: Regular alcohol use raises blood pressure in treated hypertensive subjects: a randomised controlled trial. *Lancet* 1987; 1: 647-651
  51. Mathews JD: Alcohol and hypertension (E). *Aust NZ J Med* 1979; 9: 124-128
  52. Kozararevic DJ, McGee D, Vojvodic N et al: Frequency of alcohol consumption and morbidity and mortality. *Lancet* 1980; 1: 613-616
  53. Ueshima H, Ohsaka MT, Tatara K et al: Alcohol consumption, blood pressure and stroke mortality in Japan. *J Hypertens* 1984; 2 (suppl 3): 191-195
  54. "Binge" drinking and stroke [E]. *Lancet* 1983; 2: 660-661
  55. Taylor JR, Combs-Orme T, Andersson D et al: Alcohol, hypertension and stroke. *Alcoholism (NY)* 1984; 8: 283-286
  56. Powell KE, Thompson PD, Casperson CJ et al: Physical activity and the incidence of coronary heart disease. *Ann Rev Public Health* 1987; 8: 253-287
  57. Stephens T, Jacobs DR, White CC: A descriptive epidemiology of leisure-time physical activity. *Public Health Rep* 1985; 100: 147-158
  58. Martin JE, Dubbert PM: The role of exercise in preventing and moderating blood pressure elevation. In Blafox MD, Langford HG (eds): *Non-Pharmacologic Therapy of Hypertension*, Karger, New York, 1987: 120-142
  59. Blair SN, Goodyear NW, Gibbons LW et al: Physical fitness and incidence of hypertension in healthy normotensive men and women. *JAMA* 1984; 252: 487-490
  60. Abbott RD, Levy D, Kannel WB et al: Cardiovascular risk factors and graded treadmill exercise endurance in healthy adults: the Framingham Offspring Study. *Am J Cardiol* 1989; 63: 342-346
  61. Blanchard EB, Martin JE, Dubbert PM: *Non-Drug Treatment for Essential Hypertension*, Pergamon, Elmsford NY, 1988: 107-119
  62. Martin JE, Dubbert PM: Exercise in hypertension. *Ann Behav Med* 1985; 7: 13-18
  63. Hunter GR, McCarthy JP: Pressor response associated with high intensity anaerobic training. *Physician Sportsmed* 1983; 11: 151-162
  64. McCarron DA, Morris CD, Cole C: Dietary calcium and human hypertension. *Science* 1982; 217: 267-269
  65. Kesteloot H, Joossens JV: Relationship of dietary sodium, potassium, calcium and magnesium with blood pressure: Belgian Interuniversity Research on Nutrition and Health. *Hypertension* 1988; 12: 594-599
  66. Reed D, McGee D, Yano K et al: Diet, blood pressure and multicollinearity. *Hypertension* 1985; 7: 405-410
  67. Garcia-Palmieri MG, Costas R, Cruz-Vidal M et al: Milk consumption, calcium intake and decreased hypertension in Puerto Rico: Puerto Rico Heart Health Program Study. *Hypertension* 1984; 6: 322-328
  68. Beilin LJ: Diet and hypertension: critical concepts and controversies. *J Hypertens* 1987; 5: s447-s457
  69. Belizan JM, Villar J, Pineda O et al: Reduction of blood pressure with calcium supplementation in young adults. *JAMA* 1983; 249: 1161-1165
  70. Belizan JM, Villar J, Zalazar A et al: Preliminary evidence of the effect of calcium supplementation on blood pressure in normal pregnant women. *Am J Obstet Gynecol* 1983; 146: 175-180
  71. Sunderrajan S, Bauer JH: Oral calcium supplementation does not alter blood pressure or vascular response in normotensive men [abstr]. *Circulation* 1984; 70: II-130
  72. Johnson NE, Smith EL, Freudenheim JL: Effects on blood pressure of calcium supplementation of women. *Am J Clin Nutr* 1985; 42: 12-17
  73. McCarron DA, Morris CD: Blood pressure response to oral calcium in persons with mild to moderate hypertension. *Ann Intern Med* 1985; 103: 825-831
  74. Meese RB, Gonzalez DG, Casparian JM et al: Failure of calcium supplements to relieve hypertension [abstr]. *Clin Res* 1986; 34: 218A
  75. Grobbee DE, Hofman A: Effect of calcium supplementation on diastolic blood pressure in young people with mild hypertension. *Lancet* 1986; 2: 703-706
  76. Strazzullo P, Siani A, Guglielmi S et al: Controlled trial of long-term oral calcium supplementation in essential hypertension. *Hypertension* 1986; 8: 1084-1088
  77. Bloomfield RL, Young LD, Zurek G et al: Effects of oral calcium carbonate on blood pressure in subjects with mildly elevated arterial pressure. *J Hypertens* 1986; 4 (suppl 5): s351-s354
  78. Zoccali C, Mallamaci F, Delfino D et al: Long-term oral calcium supplementation in essential hypertension: a double-blind, randomized, crossover study. *Ibid*: s676-678
  79. Nowson C, Morgan T: Effect of calcium carbonate on blood pressure. *Ibid*: s673-s675
  80. Cappuccio FP, Markandu ND, Singer DRJ et al: Does oral calcium supplementation lower high blood pressure? A double blind study. *J Hypertens* 1987; 5: 67-71
  81. Lasaridis AN, Kaisis CN, Zananiri KI et al: Oral calcium supplementation promotes renal sodium excretion in essential hypertension. *J Hypertens* 1987; 5 (suppl 5): s307-s309
  82. Lyle RM, Melby CL, Hyner GC et al: Blood pressure and metabolic effects of calcium supplementation in normotensive white and black men. *JAMA* 1987; 257: 1772-1776
  83. Thomsen K, Nilas L, Christiansen C: Dietary calcium intake and blood pressure in normotensive subjects. *Acta Med Scand* 1987; 222: 51-56
  84. Bierenbaum ML, Wolf E, Bisgeier G et al: Dietary calcium: a method of lowering blood pressure. *Am J Hypertens* 1988; 1 (suppl): 149s-152s
  85. Siani A, Strazzullo P, Guglielmi S et al: Controlled trial of low calcium versus high calcium intake in mild hypertension. *J Hypertens* 1988; 6: 253-256
  86. Bulpitt CJ, Broughton PM, Markowe HLJ et al: The relationship between both sodium and potassium intake and blood pressure in London civil servants. *J Chronic Dis* 1986; 39: 211-219
  87. Khaw KT, Barrett-Connor E: Dietary potassium and blood pressure in a population. *Am J Clin Nutr* 1984; 39: 963-968
  88. Louis WJ, Tobei R, Spector S: Effects of sodium intake on inherited hypertension in the rat. *Lancet* 1986; 2: 1283-1286
  89. Suzuki H, Kondo K, Saruta T: Effect of potassium chloride on the blood pressure in two-kidney, one clip Goldblatt hypertensive rats. *Hypertension* 1981; 3: 566
  90. MacGregor GA, Smith SJ, Markandu ND et al: Moderate potassium supplementation in essential hypertension. *Lancet* 1982; 2: 567-570
  91. Matlou SM, Isles CG, Higgs A et al: Potassium supplementation in blacks with mild to moderate essential hypertension. *J Hypertens* 1986; 4: 61-64
  92. Khaw KT, Thom S: Randomized double-blind cross-over

- trial of potassium on blood pressure in normal subjects. *Lancet* 1982; 2: 1127-1139
93. Iimura O, Kijima T, Kikuchi K et al: Studies on the hypotensive effect of high potassium intake in patients with essential hypertension. *Clin Sci Mol Med* 1981; 61 (suppl): 77s-80s
  94. Chalmers J, Morgan T, Doyle A et al: Australian National Health and Medical Research Council dietary salt study in mild hypertension. *J Hypertens* 1986; 4 (suppl 6): s629-s637
  95. Tobian L: High potassium diets markedly protect against stroke deaths and kidney disease in hypertensive rats, a possible legacy from prehistoric times. *Can J Physiol Pharmacol* 1986; 64: 840-848
  96. Khaw KT, Barrett-Connor E: Dietary potassium and stroke associated mortality. *N Engl J Med* 1987; 316: 235-240
  97. Caudill MA, Friedman R, Bensen H: Relaxation therapy in the control of blood pressure. In Blaurock MD, Langford HG (eds): *Non-Pharmacologic Therapy of Hypertension*, Karger, New York, 1987: 106-119
  98. D'Atri DA, Ostfeld D: Crowding: its effects on the elevation of blood pressure in a prison setting. *Prev Med* 1975; 4: 550-566
  99. Cobb S, Rose RM: Hypertension, peptic ulcer and diabetes in air traffic controllers. *JAMA* 1973; 224: 489-492
  100. Kasl SV, Cobb S: Blood pressure changes in men undergoing job loss: a preliminary report. *Psychosom Med* 1970; 32: 19-38
  101. Blanchard EB, Martin JE, Dubbert PM: *Non-Drug Treatments for Essential Hypertension*, Pergamon, Elmsford, NY, 1988: 41-54, 83-106
  102. Patel C, Marmot M: Can general practitioners use training in relaxation and management of stress to reduce mild hypertension? *Br Med J* 1988; 296: 21-24
  103. Southam MA, Agras WS, Taylor CB et al: Relaxation training: blood pressure lowering during the working day. *Arch Gen Psychiatry* 1982; 39: 715-717
  104. Agras WS, Southam MA, Taylor CB: Long-term persistence of relaxation-induced blood pressure lowering during the working day. *J Consult Clin Psychol* 1987; 51: 792-794
  105. Blanchard EB, Khramelashvili VV, McCoy GC et al: The USA-USSR collaborative cross-cultural comparison of autogenic training and thermal biofeedback in the treatment of mild hypertension. *Health Psychol* 1988; 7 (suppl): 175-192
  106. Parijs J, Joossens JV, Van Der Linden L et al: Moderate sodium restriction and diuretics in the treatment of hypertension. *Am Heart J* 1973; 85: 22-34
  107. Ram CVS, Garrett BN, Kaplan NM: Moderate sodium restriction and various diuretics in the treatment of hypertension: effects of potassium wastage and blood pressure control. *Arch Intern Med* 1981; 141: 1015-1019
  108. Erwtaman TM, Nagelkerke N, Lubsen J et al: Beta blockade, diuretics and salt restriction for the management of mild hypertension: a randomized double blind trial. *Br Med J* 1984; 289: 406-409
  109. Fujita T, Yamashita N, Yamashita K: Effects of indomethacin on antihypertensive action of captopril in hypertensive patients. *Clin Exp Hypertens* 1981; 3: 939-952
  110. Luft FC, Weinberger MH: Review of salt restriction and response to antihypertensive drugs: satellite symposium on calcium antagonists. *Hypertension* 1988; 11 (2 part 2): I 229-I 232
  111. Stamler R, Stamler J, Grimm R et al: Nutritional therapy for high blood pressure: final report of a four-year randomized controlled trial — the Hypertension Control Program. *JAMA* 1987; 257: 1484-1491
  112. Langford HG, Blaurock MD, Oberman A et al: Dietary therapy slows the return of hypertension after stopping prolonged medication. *JAMA* 1985; 253: 657-664
  113. Beard TC, Cooke HM, Gray WR et al: Randomized controlled trial of a no-added-sodium diet for mild hypertension. *Lancet* 1982; 2: 456-458
  114. Weinberger MH, Cohen SJ, Miller JZ et al: Dietary sodium restriction as adjunctive treatment of hypertension. *JAMA* 1988; 259: 2561-2565