Lifestyle modifications to prevent and control hypertension

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1. Methods and an overview of the Canadian recommendations

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Abstract

Objective: To provide updated, evidence-based recommendations for health care professionals on lifestyle changes to prevent and control hypertension in otherwise healthy adults (except pregnant women).

Options: For people at risk for hypertension, there are a number of lifestyle options that may avert the condition — maintaining a healthy body weight, moderating consumption of alcohol, exercising, reducing sodium intake, altering intake of calcium, magnesium and potassium, and reducing stress. Following these options will maintain or reduce the risk of hypertension. For people who already have hypertension, the options for controlling the condition are lifestyle modification, antihypertensive medications or a combination of these options; with no treatment, these people remain at risk for the complications of hypertension.

Outcomes: The health outcomes considered were changes in blood pressure and in morbidity and mortality rates. Because of insufficient evidence, no economic outcomes were considered.

Evidence: A MEDLINE search was conducted for the period January 1966 to September 1996 for each of the interventions studied. Reference lists were scanned, experts were polled, and the personal files of the authors were used to identify other studies. All relevant articles were reviewed, classified according to study design and graded according to level of evidence.

Values: A high value was placed on the avoidance of cardiovascular morbidity and premature death caused by untreated hypertension.

Benefits, harms and costs: Lifestyle modification by means of weight loss (or maintenance of healthy body weight), regular exercise and low alcohol consumption will reduce the blood pressure of appropriately selected normotensive and hypertensive people. Sodium restriction and stress management will reduce the blood pressure of appropriately selected hypertensive patients. The side effects of these therapies are few, and the indirect benefits are well known. There are certainly costs associated with lifestyle modification, but they were not measured in the studies reviewed. Supplementing the diet with potassium, calcium and magnesium has not been associated with a clinically important reduction in blood pressure in people consuming a healthy diet.

Recommendations: (1) It is recommended that health care professionals determine the body mass index (weight in kilograms/height in metres)^2 and alcohol consumption of all adult patients and assess sodium consumption and stress levels in all hypertensive patients. (2) To reduce blood pressure in the population at large, it is recommended that Canadians attain and maintain a healthy body mass index. For those who choose to drink, alcohol intake should be limited to 2 or fewer standard drinks per day (maximum of 14/week for men and 9/week for women). Adults should exercise regularly. (3) To reduce blood pressure in hypertensive patients, individualized therapy is recommended. This therapy should emphasize weight loss for overweight patients, abstinence from or moderation in alcohol intake, regular exercise, restriction of sodium intake and, in appropriate circumstances, individualized cognitive behaviour modification to reduce the negative effects of stress.

Validation: The recommendations were reviewed by all of the sponsoring organizations and by participants in a satellite symposium of the fourth International Conference on Preventive Cardiology. They are similar to those of the World Hypertension League and the Joint National Committee, with the exception of the recommendations on stress management, which are based on new information. They have not been clinically tested.

Sponsors: The Canadian Hypertension Society, the Canadian Coalition for High Blood Pressure Prevention and Control, the Laboratory Centre for Disease Control at Health Canada, and the Heart and Stroke Foundation of Canada.
Hypertension affects approximately 22% of Canadian adults. The incidence of hypertension increases with age, and most elderly Canadians have high blood pressure. Hypertension is an enormous public health issue, because it is a reversible risk factor for stroke, ischemic heart disease, congestive heart failure, renal failure and peripheral vascular disease. There is now general agreement that cardiovascular disease can be prevented by altering diet and lifestyle and by reducing risk factors such as hypertension.

The 1992 Victoria Declaration on Heart Health advised that a public health approach to the prevention and control of cardiovascular disease be adopted, one that promotes healthy dietary habits, a tobacco-free lifestyle, regular physical activity and a supportive psychosocial environment. Health care professionals play a vital role in the promotion and success of this approach, because they interact on a regular basis with a large proportion of the population and are well placed to counsel individual patients.

In 1989 the Canadian Coalition for High Blood Pressure Prevention and Control and the Canadian Hypertension Society developed consensus recommendations for lifestyle modification to prevent and treat hypertension. However, few Canadians with high blood pressure became aware of these recommendations, probably because the guidelines were poorly disseminated and because health care professionals adopted them to only a limited extent. Even when professionals were aware of the recommendations, there were limited resources for implementation, and patients failed to adhere to them.

An objective of the recommendations presented here is to increase awareness of the value of lifestyle modification in preventing and controlling hypertension. To meet this objective, the Canadian Hypertension Society, the Canadian Coalition for High Blood Pressure Prevention and Control, the Laboratory Centre for Disease Control at Health Canada, and the Heart and Stroke Foundation of Canada have revised and updated the previous recommendations using an evidence-based approach.

Lifestyle modification is a suitable primary therapy for patients with mild hypertension (i.e., blood pressure greater than 140/90 mm Hg) and is a suitable adjunct to pharmacologic therapy. Furthermore, lifestyle modification may prevent increases in blood pressure and the development of hypertension in people at risk, and such changes may be applicable to population-based interventions.

Methods

An Organizing Committee was formed with representatives from the Canadian Hypertension Society (E.B.), the Canadian Coalition for High Blood Pressure Prevention and Control (N.R.C.C.), the Laboratory Centre for Disease Control at Health Canada (G.T.) and the Heart and Stroke Foundation of Canada (E.W.). On the basis of extensive research into the factors that contribute to hypertension and those that prevent and control hypertension, the Organizing Committee chose to focus on weight loss, alcohol consumption, exercise, salt reduction, ion supplementation (calcium, magnesium and potassium) and stress reduction.

Treatment with antihypertensive medications is the primary alternative to lifestyle modification for treating hypertension. Canadian guidelines on the use of antihypertensive medications are currently being revised.

The major outcomes considered were changes in blood pressure and in morbidity and mortality rates. Economic outcomes were not considered because of insufficient research in that area.

Each of the 6 selected lifestyle areas was represented by a panel. The panel members and chairs were suggested by the executive committees of the Canadian Coalition for High Blood Pressure Prevention and Control and the Canadian Hypertension Society in September 1996. The Organizing Committee selected for panel membership health care professionals who would be affected by the guidelines as well as people with public health expertise and research interests in these areas.

The panels obtained evidence examining the association between each lifestyle modification and blood pressure in adults (except pregnant women) first by using MEDLINE searches, for which the specific dates and search terms are given in the individual reports. References cited in articles found through the literature search were also reviewed. Subject experts and the authors of some of the articles identified were asked to supply additional references, and panel members searched their personal files for relevant materials. The articles were classified according to study design and were reviewed individually. No other specific quality criteria were used to select or exclude articles. The evidence and recommendations were graded with a system used by the Canadian Hypertension Society (Tables 1 and 2).

The working panels met in January 1997 to critically evaluate the literature that had been gathered and to develop preliminary recommendations. The evidence and recommendations were presented to the members of the other panels, and the recommendations were revised after discussion. Major Canadian organizations (Table 3) with an interest in cardiovascular disease were asked to review the revised recommendations. Appropriate comments were incorporated after discussion by the members of the relevant panel, and a second revision was produced. These second revisions were circulated to the members of all panels for comment, and the recommendations and evidence were then presented at a satellite symposium of the fourth International Conference on Preventive Cardiology in July 1997 for national and international input. Any panel member who had a different opinion on some aspect of his or her panel’s recommendations was given the opportunity to voice concerns in the Interpretation section of the particular manuscript.

Results

The panels adopted the following recommendations. To prevent and treat hypertension, certain lifestyle and dietary habits of all adult patients should be routinely assessed (Table 4). The recommendations for preventing high blood pressure are given in Table 5, and the recommendations for treating high blood pressure are shown in Table 6. The evidence supporting the recommendations for each lifestyle modification was reviewed by the respective panel and is presented separately.
Interpretation

Health care professionals can help their patients by checking blood pressure at every opportunity and by counselling patients and their families about preventing hypertension. All patients would benefit from general advice on healthy lifestyle habits, in particular healthy body weight, moderate consumption of alcohol and regular exercise. Because the risk of cardiovascular disease rises with blood pressure throughout the normotensive blood pressure range, patients with normal blood pressure may also benefit from lifestyle modification. It has been estimated, for example, that lowering the median blood pressure of the population by 2 mm Hg could be more effective in reducing the rate of cardiovascular disease than medically treating individual patients who have diastolic blood pressure greater than 95 mm Hg. Several of the recommendations presented here, such as maintaining ideal body weight, exercising regularly and reducing stress, may delay the onset of or prevent cardiovascular disease, independent of their effects in reducing blood pressure. Reducing blood pressure and the risk of cardiovascular disease by changing lifestyle habits could decrease the cost of health care by decreasing the use of pharmacologic and invasive cardiovascular treatments.

Lifestyle advice can be tailored to individual patients. Some changes may be more appropriate for certain people. For example, weight loss is more likely to benefit people who are obese. Research has not addressed which lifestyle changes are preferred by patients, but such information would influence the way in which advice could be tailored to the individual. For some patients, a single change may be appropriate, whereas for others, several changes may be advisable.

Table 7 provides summaries of randomized controlled trials in which multiple, simultaneous lifestyle modifications were made to prevent hypertension in people with normal blood pressure. Table 8 summarizes those in which multiple, simultaneous interventions were used to treat hypertension. The strength of these trials is that they used meaningful endpoints, such as the development of hypertension or avoidance of antihypertensive medication. However, there were some limitations. To a large extent the subjects were highly selected (usually white, educated men who adhered to the interventions), and in some studies the endpoints were retrospective, the statistical analyses were unclear, the endpoint was determined arbitrarily by nonstudy physicians prescribing drug treatment, or the analysis was restricted to a subgroup of patients. De-

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**Table 2: Grading system for recommendations**

<table>
<thead>
<tr>
<th>Rank</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>The recommendation is based on one or more level I studies</td>
</tr>
<tr>
<td>B</td>
<td>The best evidence available was at level II</td>
</tr>
<tr>
<td>C</td>
<td>The best evidence available was at level III</td>
</tr>
<tr>
<td>D</td>
<td>The best evidence available was ranked lower than level III and included expert opinion</td>
</tr>
</tbody>
</table>

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**Table 3: Organizations that received draft recommendations for review**

<table>
<thead>
<tr>
<th>AMG Medical Inc.</th>
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</thead>
<tbody>
<tr>
<td>Atlantic Cardiovascular Health Association</td>
</tr>
<tr>
<td>Bayer Inc.</td>
</tr>
<tr>
<td>Bristol-Myers Squibb Pharmaceutical Group</td>
</tr>
<tr>
<td>Canadian Cardiology Society</td>
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<tr>
<td>Canadian Council of Cardiovascular Nurses</td>
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<tr>
<td>Canadian Council on Smoking and Health</td>
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<tr>
<td>Canadian Diabetes Association</td>
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<tr>
<td>Canadian Hypertension Society</td>
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<tr>
<td>Canadian Medical Association</td>
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<tr>
<td>Canadian Nurses Association</td>
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<tr>
<td>Canadian Pediatric Society</td>
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<tr>
<td>Canadian Pharmacists Association</td>
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<tr>
<td>Canadian Public Health Association</td>
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<tr>
<td>Canadian Stroke Society</td>
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<tr>
<td>College of Family Physicians of Canada</td>
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<tr>
<td>Dietitians of Canada</td>
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<tr>
<td>Health Canada, Disease Prevention Division</td>
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<tr>
<td>Health Canada, Laboratory Centre for Disease Control</td>
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<tr>
<td>Heart and Stroke Foundation of Canada</td>
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<tr>
<td>Hoechst Marion Roussel Canada</td>
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<tr>
<td>Merck Frosst Canada Inc.</td>
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<tr>
<td>Parke Davis Canada</td>
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<tr>
<td>Pfizer Canada Inc.</td>
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<tr>
<td>Searle Canada Inc.</td>
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<tr>
<td>Servier Canada Inc.</td>
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</tbody>
</table>

*Not all organizations commented on the draft recommendations.*

**Table 4: Recommendations to assess lifestyle and dietary habits**

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Determine the weight, height and body mass index of hypertensive patients</td>
<td>D</td>
</tr>
<tr>
<td>Determine the alcohol consumption of all adult patients</td>
<td>D</td>
</tr>
<tr>
<td>Determine the salt consumption of hypertensive patients</td>
<td>D</td>
</tr>
<tr>
<td>Consider the contribution of stress in hypertensive patients</td>
<td>D</td>
</tr>
</tbody>
</table>

**Table 5: Recommendations to prevent hypertension**

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>All adults should attain and maintain a healthy body mass index</td>
<td>B</td>
</tr>
<tr>
<td>Alcohol consumption should be in accordance with Canadian low-risk drinking guidelines (i.e., healthy adults should limit alcohol consumption to 2 drinks or fewer per day, and consumption should not exceed 14 standard drinks per week for men and 9 standard drinks per week for women)</td>
<td>B</td>
</tr>
<tr>
<td>All adults should be encouraged to participate in regular, moderately intense (40% to 60% of maximal oxygen consumption) physical activity for 50–60 minutes, 3 or 4 times per week</td>
<td>B</td>
</tr>
</tbody>
</table>
spite these limitations, the data consistently support multiple lifestyle changes to prevent hypertension or, in some patients, to replace antihypertensive therapy.

Two studies that were not published at the time the panels reviewed the literature and that therefore could not be included in these recommendations merit discussion. In the Dietary Approaches to Stop Hypertension (DASH) study, the blood pressure of 459 untreated hypertensive patients and people with normal blood pressure was significantly reduced after 8 weeks of a dietary intervention. The people in this study followed 1 of 3 diets: a diet rich in fruits and vegetables, a diet low in fat, or a diet low in fat and rich in fruit and vegetables. This approach requires further research. In the Trial of Nonpharmacologic Intervention in the Elderly (TONE), weight loss, sodium restriction, and combined weight loss and sodium restriction were effective replacements for pharmacologic therapy in elderly hypertensive patients. Unfortunately, at the time the panels were reviewing the literature the TONE data were available only in abstract form and therefore were not appropriate for evaluation.

**Dissemination and implementation strategies**

The public needs to be made aware of the risks of unhealthy lifestyles and the benefits of change. These recommendations should therefore be disseminated to health care professionals, patients and the general public.

Resources need to be developed at a local level to provide counselling to patients and to monitor the advice they are given. A philosophical switch is required whereby more resources would be invested in maintaining health and promoting lifestyle changes. Public policy promoting good health is a vital component of the comprehensive approach required to support and encourage lifestyle changes. Public policy can be implemented at all levels of government and can affect both workplaces and public places. Through legislation, public policy produces such benefits as smoke-free public spaces and green spaces in communities. One public policy that promotes good nutrition is the Health Check program of the Heart and Stroke Foundation of Canada. This program identifies and labels food products, providing easy-to-use guidance for the public in choosing foods that are part of a healthy diet. This is a simple way of creating a supportive environment in which healthier lifestyle choices are easier to make. There are, however, many other aspects of lifestyle where public policy could be supportive. Governments must be encouraged, by health care professionals and the public, to act in these areas.

The Organizing Committee plans to disseminate these recommendations through a series of publications and on the Web sites of the many organizations involved in their development. In addition, radio and television announcements or programs should be used to increase public awareness.

In 1998 plans for implementing these recommendations and other Canadian blood pressure guidelines were developed, and this process is continuing. The success of these recommendations will depend on the degree to which they are adopted and used by physicians, nurses, dietitians, pharmacists, psychologists, other health care professionals and health-related organizations, such as the Heart and Stroke Foundation of Canada. It will also depend on the provision of adequate resources by provincial and local health authorities.

**Future research implications**

There are some serious limitations to both the published research on the use of lifestyle management to reduce blood pressure and the methods used to assess this evidence. The most serious limitation of current research is the outcome measures that have been used. Most trials have focused on reducing blood pressure, whereas only a few have examined endpoints that are meaningful to patients, such as the prevention of hypertension and thus the avoidance of pharmacologic therapy, the replacement of pharmacologic therapy in those previously treated, the development of cardiovascular disease and early death. Data

| Table 6: Recommendations for lifestyle modification to treat high blood pressure |
|---------------------------------|-----|
| Recommendation | Grade |
| Weight loss should be encouraged for all overweight patients; even moderate weight loss (i.e., 4.5 kg in obese, hypertensive patients) can improve blood pressure | B |
| Alcohol consumption should be in accordance with Canadian low-risk drinking guidelines (i.e., healthy adults should limit alcohol consumption to 2 drinks or fewer per day, and consumption should not exceed 14 standard drinks per week for men and 9 standard drinks per week for women) | C |
| All adults should be encouraged to participate in regular, moderately intense (40% to 60% of maximal oxygen consumption) physical activity for 50–60 minutes, 3 or 4 times per week | B |
| Patients should be advised to choose foods low in salt, to avoid salty foods and to minimize the use of salt at the table and during cooking | D |
| In selected patients with hypertension in whom stress appears to be a significant factor, individualized cognitive behaviour interventions should be advised | B |
on the degree to which changes in lifestyle reduce morbidity and mortality rates is lacking. To date, there have been no randomized controlled trials of lifestyle modification to treat or prevent hypertension that have also examined rates of illness and death. The demonstration by such studies of significant effects on morbidity and mortality rates would be a major incentive to implement lifestyle measures to treat and prevent hypertension.

The second limitation of our work has to do with the levels of evidence used to develop the recommendations. A substantial proportion of current lifestyle research is epidemiologic. Current systems for grading the evidence of treatment effects have no evidence-based way to evaluate the validity of different study designs, and some even ignore epidemiologic studies altogether.

Although it is accepted that associations found in epidemiologic studies do not prove causality or the benefits of interventions, these studies may be more reliable than some clinical study designs (e.g., case series).

The inability to objectively grade epidemiologic evidence and evidence from other clinical study designs represents a glaring deficit. It should be possible to assess the reliability of epidemiologic studies in relation to well-designed randomized controlled trials (which repre-

### Table 7: Randomized controlled trials with simultaneous lifestyle interventions to prevent hypertension in people with normal blood pressure

<table>
<thead>
<tr>
<th>Study design</th>
<th>Subjects</th>
<th>Intervention</th>
<th>Duration</th>
<th>Endpoint</th>
<th>Study limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parallel10,11</td>
<td>385 men, 179 women; 30–54 yr; baseline BP 80–89 mm Hg</td>
<td>Reduced caloric and alcohol intake; increased activity</td>
<td>18 mo</td>
<td>RR of hypertension = 0.49 (95% CI 0.29–0.83)</td>
<td>Retrospective analysis; highly selected patients; analysis difficult to verify</td>
</tr>
<tr>
<td>Parallel12</td>
<td>174 men, 27 women; 30–44 yr; baseline BP 85–89 mm Hg or 80–84 mm Hg; weight 110%–139% of ideal; resting pulse &gt; 80 BPM</td>
<td>Reduced caloric, sodium and alcohol intake; increased activity</td>
<td>5 yr</td>
<td>RR of hypertension = 0.46, p = 0.027</td>
<td>Highly selected patients; diagnosis of hypertension not standardized</td>
</tr>
<tr>
<td>Parallel13</td>
<td>541 men, 292 women; 25–49 yr; baseline BP &gt; 78 and &lt; 89 mm Hg</td>
<td>1. Reduced caloric and sodium intake 2. Reduced sodium, increased potassium intake</td>
<td>3 yr</td>
<td>1. RR of hypertension = 0.812 2. RR of hypertension = 0.65</td>
<td>Definition of hypertension not clinically relevant; highly selected patients; ineffective weight loss regimen; incomplete statistical analysis</td>
</tr>
<tr>
<td>2 × 2 factorial14</td>
<td>1485 men, 765 women; 30–54 yr; baseline BP 83–89 mm Hg; body weight 110%–163% of ideal</td>
<td>1. Weight loss (reduced caloric intake, increased physical activity) 2. Reduced sodium intake 3. Both 1 and 2</td>
<td>4 yr</td>
<td>RR of hypertension = 0.812 1. Weight loss RR = 0.87, p = 0.06 2. Sodium restriction RR = 0.86, p = 0.04 3. Combined intervention RR = 0.85, p = 0.02</td>
<td>Highly selected patients</td>
</tr>
</tbody>
</table>

Note: BP = blood pressure; RR = relative risk; CI = confidence interval; BPM = beats per minute.

### Table 8: Randomized controlled trials substituting simultaneous lifestyle interventions for pharmacologic therapy in hypertensive patients

<table>
<thead>
<tr>
<th>Study design</th>
<th>Patients</th>
<th>Intervention</th>
<th>Duration</th>
<th>Endpoint</th>
<th>Study limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parallel15</td>
<td>120 men, 69 women; age &gt; 35 yr; baseline BP 120/78.2 mm Hg</td>
<td>Reduced sodium, caloric and alcohol intake; discontinued pharmacologic therapy</td>
<td>4 yr</td>
<td>39% of intervention group and 5% of control group did not require pharmacologic therapy (RR = 7.8, p &lt; 0.001)</td>
<td>Highly selected patients; small trial; alcohol intervention ineffective</td>
</tr>
<tr>
<td>Parallel16</td>
<td>Untreated Japanese patients; 53 men, 58 women; baseline BP 140–179/90–109 mm Hg</td>
<td>Reduced sodium, alcohol and sugar intake; increased milk intake; increased activity</td>
<td>18 mo</td>
<td>91% of intervention group and 76% of control group did not require pharmacologic therapy (RR = 1.2, p = 0.02)</td>
<td>Endpoint for initiating pharmacologic therapy was not standardized</td>
</tr>
</tbody>
</table>
sent level I evidence) and other study designs. There is a pressing need to develop an objective, evidence-based scheme for grading all types of evidence." This would improve our current guideline methodology.

Support for research

Pharmaceutical companies deserve recognition for their investment in this process to improve the health of Canadians, to find alternatives to drug therapy and to reduce drug use. The positive influence of the Canadian pharmaceutical industry on health, beyond the development of new pharmaceutical products, must be discussed, encouraged and recognized.

Conclusion

The development of the recommendations presented in this supplement was possible only through the cooperation of many organizations. Care was taken to ensure that these recommendations are compatible with an overall improvement in health and do not lead simply to a reduction in blood pressure. The panels found no evidence that harm would come to patients who followed these recommendations. The broad application of these recommendations is strongly encouraged because of the beneficial effect they could have on overall mortality rates, as well as on cardiovascular disease, osteoporosis, alcohol-related violence and certain cancers. However, as for all general recommendations, the physician must consider each patient individually and assess the risks and benefits of every therapy before providing advice.

We are grateful for the external reviews of the Canadian Council of Cardiovascular Nurses, the Canadian Nurses Association, the Canadian Pharmacists Association, the Canadian Public Health Association, the College of Family Physicians of Canada, the Heart and Stroke Foundation of Canada, Hoechst Marion Roussel and Merck Frosst Canada Inc.

The financial assistance of the Laboratory Centre for Disease Control at Health Canada and Astra Pharma Inc., Bayer Inc., Bristol–Myers Squibb Pharmaceutical Group, Knoll Pharma Inc., Merck Frosst Canada Inc., Searle Canada Inc. and Servier Canada Inc. is gratefully acknowledged, as is the secretarial assistance of Leslie Holmes.

Competing interests: None declared for Drs. Burgess, Choi, Taylor, Wilson, Cleroux, Fodor and Leiter. Drs. Campbell and Spence have received consultancy fees, speaker’s fees, educational grants and travel assistance from various pharmaceutical companies.

References


Reprint requests to: Heart and Stroke Foundation of Canada, 1402–222 Queen St, Ottawa ON K1P 5V9; fax 613 569-3278
2. Recommendations on obesity and weight loss

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Abstract

Objective: To provide updated, evidence-based recommendations concerning the effects of weight loss and maintenance of healthy weight on the prevention and control of hypertension in otherwise healthy adults (except pregnant women).

Options: The main options are to attain and maintain a healthy body weight (body mass index [BMI] 20–25 kg/m²) or not to do so. For those at risk for hypertension, weight loss and maintenance of healthy weight may prevent the condition. For those who have hypertension, weight loss and maintenance of healthy weight may reduce or obviate the need for antihypertensive medications.

Outcomes: The health outcome considered was change in blood pressure. Because of insufficient evidence, no economic outcomes were considered.

Evidence: A MEDLINE search was conducted for the years 1992–1996 with the terms hypertension and obesity in combination and antihypertensive therapy and obesity in combination. Other relevant evidence was obtained from the reference lists of the articles identified, from the personal files of the authors and through contacts with experts. The articles were reviewed, classified according to study design and graded according to level of evidence.

Values: A high value was placed on the avoidance of cardiovascular morbidity and premature death caused by untreated hypertension.

Benefits, harms and costs: Weight loss and the maintenance of healthy body weight reduces the blood pressure of both hypertensive and normotensive people. The indirect benefits of a healthy body weight are well known. The negative effects of weight loss are primarily the frustrations associated with attaining and maintaining a healthy weight. The costs associated with weight loss programs were not measured in the studies reviewed.

Recommendations: (1) It is recommended that health care professionals determine weight (in kilograms), height (in metres) and BMI for all adults. (2) To reduce blood pressure in the population at large, it is recommended that Canadians attain and maintain a healthy BMI (20–25). (3) All overweight hypertensive patients (BMI greater than 25) should be advised to reduce their weight.

Validation: These recommendations are similar to those of the World Hypertension League, the National High Blood Pressure Education Program Working Group on Primary Prevention of Hypertension, the Canadian Hypertension Society and the Canadian Coalition for High Blood Pressure Prevention and Control. They have not been clinically tested.

Sponsors: The Canadian Hypertension Society, the Canadian Coalition for High Blood Pressure Prevention and Control, the Laboratory Centre for Disease Control at Health Canada, and the Heart and Stroke Foundation of Canada.

Weight loss is often considered for the treatment of hypertension, yet the specifics of the responses to treatment are not well known. In 1989 a consensus conference sponsored by the Canadian Coalition for High Blood Pressure Prevention and Control and the Canadian Hypertension Society recommended that weight loss may help in both the prevention and the treatment of hypertension. Those recommendations were based on relatively sparse data. Since then, a number of new studies have been published on the relation between hypertension and obesity, as well as on the relation between weight loss and blood pressure.

In 1992 the Canadian Heart Health Survey highlighted the importance of obesity as a public health problem. At that time in Canada 35% of men and 27% of women were obese (body mass index [BMI] greater than 27). Guidelines for BMI (which is calculated by dividing body weight, in kilograms, by the square of height, in metres) have been published for Canada. A BMI above 27 is associated with increased health risks, including hypertension. The causes of the higher blood pressure associated with obesity are likely multiple and include insulin resistance or hyperinsulinemia,
overactivity of the sympathetic nervous system and alterations in the renin–angiotensin system.

The primary objective of these guidelines is to identify the evidence for and provide evidence-based recommendations to health care professionals about the relation between obesity and hypertension and the response of blood pressure to weight loss.

Methods

A complete description of the methods for these guidelines is given in part 1 of this supplement.6 The chair and members of the panel were selected by the Organizing Committee for the lifestyle modification recommendations to obtain a spectrum of health care professionals and scientists with expertise and interest in the areas of hypertension, obesity and heart health.

The initial MEDLINE search was performed using the terms hypertension and obesity in combination and antihypertensive therapy and obesity in combination for English-language studies published between 1992 and 1996. Another search was performed just before publication using the terms hypertension, obesity and controlled clinical trial in combination for the years 1996–1998. Additional articles and articles published before 1992 were identified by reviewing the reference lists of the identified articles, were found in the personal files of the panel members and were suggested by other experts. For the primary recommendations regarding the efficacy of weight loss, the results of randomized controlled trials were used. However, epidemiologic studies and nonrandomized intervention trials were also reviewed. The data from the randomized controlled trials were interpreted in the context of the more extensive data obtained from studies with these other designs, which were used for the secondary recommendations. The principles for grading the evidence and the recommendations were based on those previously used by the Canadian Hypertension Society7 and are summarized in part 1 of this supplement.6 An attempt was made to reach a consensus on all recommendations. The evidence and the recommendations were presented for comment to the other expert panels for this guidelines series, submitted for review to major Canadian organizations and presented at an international conference on preventive cardiology, to allow further national and international input. All revisions were reviewed and assessed by the panel before incorporation into the final document.

Results

Observational data

Extensive epidemiologic data support a positive association between body weight and blood pressure. Cross-sectional studies have demonstrated the association in both sexes, for people of different ages and in several ethnic groups. Among Canadian adults younger than 55 years of age, the prevalence of hypertension is at least 5-fold higher for those with a BMI greater than 30 than for those whose BMI is less than 20.4 The association is similar but less pronounced in older Canadians. In another study of one million North American subjects5 the odds ratio for hypertension (in comparisons of obese and non-obese subjects) was 2.42 for those aged 20–39 years and 1.54 for those aged 40–64 years. Similarly, when subjects in the Framingham study8 were grouped in BMI quintiles, both systolic and diastolic blood pressure increased progressively; the mean difference between the first and fifth quintile was 16 mm Hg for systolic blood pressure and 9 mm Hg for diastolic blood pressure. The Nurses Health Study9 reported that hypertension was 2 to 6 times more prevalent among heavier women (BMI of 29 or above) than among less heavy women (BMI less than 22). Other studies have indicated that the association is even stronger for people with relatively higher abdominal fat.10

Prospective cohort studies have also shown a correlation between weight gain and increases in blood pressure.11 In the Framingham study, for each 4.5 kg of weight gain there was an associated increase in systolic blood pressure of 4 mm Hg in both men and women.5

To facilitate the identification of a risk of having hypertension or cardiovascular disease due to obesity or of these conditions developing, a reliable objective assessment is required. BMI is simple to calculate, is based on readily available clinical data and provides a basis for both assessing cardiovascular prognosis and monitoring change. BMI has been used extensively to generate prognostic cardiovascular data and is superior to body weight alone for the assessment of obesity. Other measures of obesity may predict cardiovascular disease more accurately than BMI but require measurements not performed routinely in clinical practice. Therefore, BMI is currently the most useful clinical assessment on which to base recommendations for weight loss, to assess cardiovascular risk, to establish therapeutic goals and to monitor change.

Recommendation

• Body mass index should be routinely assessed for all adult patients (grade D recommendation).

Randomized controlled trials

Several randomized controlled trials of weight loss have demonstrated that a reduction in weight is associated with a reduction in blood pressure. Table I summarizes the data from randomized controlled trials of weight reduction in overweight hypertensive patients.12–20 In all of the trials except one,17 weight reduction was associated with a reduction in mean blood pressure (level II evidence), although the lack of an appropriate statistical analysis was a limitation for some of the studies18,19,20 (Table I). For overweight patients, the efficacy of weight loss in reducing blood pressure is similar to that of single antihypertensive drug therapy.16,17

The Treatment of Mild Hypertension Study provides level II evidence that the effect of antihypertensive medications on blood pressure is additive to that achieved by weight loss alone.21 However, patients randomly assigned to receive β-blocker therapy lost less weight than those receiving other drug therapies. The Trial of Antihypertensive In-
terventions and Management (TAIM) revealed an additive effect of chlorthalidone therapy and weight loss on blood pressure in obese subjects.

**Recommendations**

- Overweight hypertensive patients (BMI greater than 25) should be advised to reduce their weight (grade B recommendation).
- Overweight hypertensive patients receiving antihypertensive drugs should be advised to lose weight for additional antihypertensive effect (grade B recommendation).

Table 2 summarizes the data from randomized controlled trials of weight reduction in overweight normotensive people. In this population weight reduction is associated with a reduction in blood pressure, which indicates the potential utility of weight reduction in preventing hypertension (level II evidence). The phase I study of the Trials of Hypertension Prevention showed that weight reduction was more effective than other lifestyle strategies in preventing hypertension.

**Recommendation**

- Adults should be encouraged to attain and maintain a healthy body weight (BMI of 20–25) to prevent or forestall the development of hypertension (grade B recommendation).

**Nonrandomized trials and post hoc analysis of intervention trials**

Evidence from nonrandomized trials and from post hoc analysis of intervention trials constitutes level III evidence.

<table>
<thead>
<tr>
<th>Study design</th>
<th>Subjects</th>
<th>Intervention</th>
<th>Duration</th>
<th>Change in body weight, kg</th>
<th>Change in BP, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCT*</td>
<td>49 hypertensive patients</td>
<td>Dietitian counselling = 15 Diet sheet = 14 MD advice = 20</td>
<td>1 yr</td>
<td>-5.1</td>
<td>-11.9/6.9</td>
</tr>
<tr>
<td>RCT*</td>
<td>107 overweight, hypertensive patients</td>
<td>No antihypertensives, weight reduction program = 24 Antihypertensives, weight reduction program = 57 Antihypertensives, no dietary program = 26</td>
<td>6 mo</td>
<td>-8.8</td>
<td>75% achieved normal BP</td>
</tr>
<tr>
<td>RCT*</td>
<td>54 mildly hypertensive, obese people</td>
<td>Behaviourally oriented hypocaloric diet</td>
<td>6 mo</td>
<td>-3.3*</td>
<td>+5.0/1.5 NS</td>
</tr>
<tr>
<td>RCT*</td>
<td>56 overweight patients, &lt; 55 yr</td>
<td>Hypocaloric diet = 20 Metoprolol = 18 Placebo = 18</td>
<td>21 wk</td>
<td>-7.4‡</td>
<td>-13.3/9.8‡</td>
</tr>
<tr>
<td>RCT*</td>
<td>163 of 692 overweight, mildly hypertensive patients, 21–65 yr</td>
<td>Usual care and placebo = 79 Weight reduction program and placebo = 84</td>
<td>6 mo</td>
<td>-3.5</td>
<td>-10.5/8.2</td>
</tr>
<tr>
<td>RCT*</td>
<td>30 obese hypertensive patients</td>
<td>16 dexfenfluramine 14 placebo</td>
<td>3 mo</td>
<td>-6.0‡</td>
<td>-11.0/4.0</td>
</tr>
<tr>
<td>RCT*</td>
<td>61 mildly hypertensive, overweight men</td>
<td>31 hypocaloric diet 30 drug therapy, no diet restriction</td>
<td>1 yr</td>
<td>-8.8§</td>
<td>-4/3</td>
</tr>
<tr>
<td>RCT 2 × 2 factorial</td>
<td>585 obese patients, 60–80 yr</td>
<td>Behaviourally oriented weight loss program v. usual care Sodium restriction, group counselling v. usual care Weight loss and sodium restriction v. usual care</td>
<td>29 mo</td>
<td>-3.8§</td>
<td>RR of drug treatment or a cardiovascular event = 0.64‡</td>
</tr>
</tbody>
</table>

Note: RCT = randomized controlled trial, BP = blood pressure, NS = nonsignificant, RR = relative risk.

*Statistics not provided.

†p < 0.05 compared with group treated with antihypertensives and no dietary program.

‡p < 0.05 compared with placebo.

§p < 0.001.

¶p < 0.002.
The TAIM study examined the response of diastolic blood pressure to various combinations of dietary and pharmacologic interventions in 879 mildly hypertensive subjects (baseline weight 88.0 kg, mean BMI 30.4). Over 6 months, the mean weight loss was 4.7 kg, which was associated with an improvement in diastolic blood pressure in all groups. In patients receiving placebo who lost 4.5 kg or more, diastolic blood pressure declined by 11.6 mm Hg. For those who lost less than 4.5 kg, the decrease in diastolic blood pressure was not significantly different from the decrease in those receiving a placebo and eating their usual diet. Over the subsequent 4.5 years of follow-up, those following the weight-loss diet lost 2–3 kg, whereas those eating their usual diet lost no weight. Among those who were able to maintain their weight loss, the 5-year incidence of treatment failure (defined as not remaining on the initial drug therapy) was 23% lower than among those who did not maintain their weight loss.

**Recommendation**

- In overweight patients with high blood pressure it should be emphasized that even 4.5 kg of weight loss may reduce their blood pressure (grade C recommendation).

The recommendations on weight loss and hypertension are summarized in Table 3.

**Interpretation**

The attainment and maintenance of a healthy body weight can prevent hypertension and can be used as the primary treatment for mild hypertension or as an adjunct to pharmacologic therapy. Weight reduction may also reduce other cardiovascular risk factors, although there are no data from long-term randomized controlled trials demonstrating that weight reduction decreases morbidity and mortality rates. Although the long-term effectiveness of weight loss in the management of hypertension has been questioned, some studies using a multifactorial approach involving nutrition education, alcohol reduction and physical activity have demonstrated long-term weight and blood pressure reduction. However, knowledge that even modest weight loss (4.5 kg) can result in improvement in blood pressure and other metabolic abnormalities should help to emphasize the potential important role of weight reduction in blood pressure control.

Unfortunately, the overall results of lifestyle modification to reduce obesity are poor and lead to frustration and pessimism for both the patient and the health care provider. Most long-term trials of weight reduction have found that weight returns to baseline levels after several years, although a few people are able to maintain their reduced weight. Pharmacotherapy for obesity also has substantial problems. Two drugs (fenfluramine and dexfenfluramine) used to assist in weight reduction have recently been withdrawn from the market because they were associated with valvular heart lesions and pulmonary hypertension. Sympathomimetic appetite suppressants are still available but may be associated with increased blood pressure and have limited effectiveness in reducing weight.

Strategies for weight reduction in a hypertensive obese person are no different from those for any other obese person. According to the Canadian Task Force on the Treatment of Obesity, a comprehensive, multifactorial approach is preferred. The approach should employ instruction about diet, increased physical activity and behaviour modification techniques. Among those receiving antihypertensive medications, weight loss may be more difficult in those treated with β-blocking antihypertensive drugs. The selection of initial antihypertensive drug therapy for an obese person should follow the guidelines of the Canadian Hypertension Society (new guidelines will be available later in 1999).

**Validation**

These recommendations are similar to those of the World Hypertension League, the National High Blood Pressure Education Program Working Group on Primary Hypertension, and several other national and international organizations.
Prevention of Hypertension, the Canadian Hypertension Society and the Canadian Coalition for High Blood Pressure and Control.1,10

Future research

Several issues remain to be addressed:
- Strategies to promote and sustain long-term weight loss must be developed and tested.
- Long-term studies relating weight loss to cardiovascular morbidity and mortality rates must be undertaken.
- More information must be gathered on the predictors of blood pressure response to weight loss, including patient characteristics, metabolic data and relation between degree of weight loss and blood pressure response.
- Data on the prevalence of hypertension among Aboriginal populations in Canada and the response to treatment in these groups are needed.
- The utility of visceral fat as a predictor of blood pressure must be confirmed, and improved, simpler methods of quantifying visceral fat should be developed.
- More information must be gathered on the interactions between weight loss and specific antihypertensive drugs in controlling blood pressure.
- Trials comparing blood pressure responses to specific antihypertensive drugs in obese and non-obese individuals must be undertaken.

Conclusion

An increased awareness of the benefits of attaining and maintaining a healthy body weight for overall health and specifically for the prevention and treatment of hypertension is indicated. Future efforts must be geared to better understanding this phenomenon, disseminating and implementing this information and finding the most effective means of sustaining weight loss.

We are grateful for the external reviews of the Canadian Council of Cardiovascular Nurses, the Canadian Nurses Association, the Canadian Pharmacists Association, the Canadian Public Health Association, the College of Family Physicians of Canada, the Heart and Stroke Foundation of Canada, Hоечшт Marion Roussel and Merck Frosst Canada Inc.

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Competing interests: None declared for Ms. Abbott and Drs. Chockalingam, Leiter, Mendelson and Ogilvie. Dr. Campbell has received consultancy fees, speaker’s fees, educational grants and travel assistance from various pharmaceutical companies.

References


Table 3: Recommendations for weight loss to control and prevent hypertension

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height and weight should be measured and body mass index (BMI) calculated for all adults</td>
<td>D</td>
</tr>
<tr>
<td>A healthy BMI (20–25) is recommended for all adults to prevent hypertension</td>
<td>B</td>
</tr>
<tr>
<td>All overweight hypertensive people (BMI &gt; 25) should be advised to reduce their weight</td>
<td>B</td>
</tr>
<tr>
<td>Overweight patients (BMI &gt; 25) should be advised to lose a minimum of 4.5 kg to reduce blood pressure</td>
<td>C</td>
</tr>
<tr>
<td>A multifactorial approach to weight loss, incorporating diet instruction, increased physical activity and behaviour modification techniques, should be used</td>
<td>D</td>
</tr>
</tbody>
</table>


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3. Recommendations on alcohol consumption

Norman R.C. Campbell, MD; Mary Jane Ashley, MD, MSc; S. George Carruthers, MD; Yves Lacourcière MD; Donald W. McKay, PhD

Abstract

Objective: To provide updated, evidence-based recommendations concerning the effects of alcohol consumption on the prevention and control of hypertension in otherwise healthy adults (except pregnant women).

Options: There are 2 main options for those at risk for hypertension: avert the condition by limiting alcohol consumption or by using other nonpharmacologic methods, or maintain or increase the risk of hypertension by making no change in alcohol consumption. The options for those who already have hypertension include decreasing alcohol consumption or using another nonpharmacologic method to reduce hypertension; commencing, continuing or intensifying antihypertensive medication; or taking no action and remaining at increased risk of cardiovascular disease.

Outcomes: The health outcomes considered were changes in blood pressure and in morbidity and mortality rates. Because of insufficient evidence, no economic outcomes were considered.

Evidence: A MEDLINE search was conducted for the period 1966–1996 with the terms ethyl alcohol and hypertension. Other relevant evidence was obtained from the reference lists of articles identified, from the personal files of the authors and through contacts with experts. The articles were reviewed, classified according to study design, and graded according to the level of evidence.

Values: A high value was placed on the avoidance of cardiovascular morbidity and premature death caused by untreated hypertension.

Benefits, harms and costs: A reduction in alcohol consumption from more than 2 standard drinks per day reduces the blood pressure of both hypertensive and normotensive people. The lowest overall mortality rates in observational studies were associated with drinking habits that were within these guidelines. Side effects and costs were not measured in any of the studies.

Recommendations: (1) It is recommended that health care professionals determine how much alcohol their patients consume. (2) To reduce blood pressure in the population at large, it is recommended that alcohol consumption be in accordance with Canadian low-risk drinking guidelines (i.e., healthy adults who choose to drink should limit alcohol consumption to 2 or fewer standard drinks per day, with consumption not exceeding 14 standard drinks per week for men and 9 standard drinks per week for women). (3) Hypertensive patients should also be advised to limit alcohol consumption to the levels set out in the Canadian low-risk drinking guidelines.

Validation: These recommendations are similar to those of the World Hypertension League, the National High Blood Pressure Education Program Working Group on Primary Prevention of Hypertension and the previous recommendations of the Canadian Coalition for High Blood Pressure Prevention and Control and the Canadian Hypertension Society. They have not been clinically tested. The low-risk drinking guidelines are those of the Addiction Research Foundation of Ontario and the Canadian Centre on Substance Abuse.

Sponsors: The Canadian Hypertension Society, the Canadian Coalition for High Blood Pressure Prevention and Control, the Laboratory Centre for Disease Control at Health Canada, and the Heart and Stroke Foundation of Canada. The low-risk drinking guidelines have been endorsed by the College of Family Physicians of Canada and several provincial organizations.

Hypertension affects approximately 1 in 5 adult Canadians and leads to premature cardiovascular disease and death. The costs of treating hypertension are high and rising. Nonpharmacologic interventions and lifestyle changes that prevent hypertension and reduce blood pressure may play an important role in reducing morbidity and death related to cardiovascular disease and in moderating treatment costs. Consensus recommendations on lifestyle modifications for the treatment and control of hypertension were formulated by the Canadian Coalition for High Blood Pressure Prevention and Control and the Canadian Hypertension Society.
in 1989. Since then, many randomized controlled studies have been conducted examining the effect of lifestyle interventions on blood pressure.

The consumption of alcohol by adults in Canada is common. Overall, 75% of Canadians over the age of 15 years drink alcohol, and 61% of adult Canadians have 15 or more drinks per week. In a 1997 survey of Ontario adults, 25% of men and 10% of women either exceeded recommended weekly intake levels or had more than 2 drinks per day as often as once per week. These limits are considered to represent a low risk of health and other alcohol-related problems.

A number of epidemiologic and clinical studies have addressed the association between alcohol consumption and blood pressure. In light of the high prevalence of both hypertension and alcohol consumption among adults in Canada and the evidence that alcohol contributes to hypertension, the expert panel considered this an important and potentially reversible public health problem.

The primary objective of this guideline is to review the evidence examining the relation between alcohol consumption and blood pressure, including the effects of moderation or abstinence on reducing blood pressure in hypertensive men and non-pregnant women and on preventing hypertension in the general adult population, and to advise health care professionals and the public accordingly. The main health care strategy under examination consisted of the guidelines on low-risk drinking of the Addiction Research Foundation of Ontario and the Canadian Centre on Substance Abuse.

Methods

A complete description of the methods used in developing these recommendations is given in part 1 of this supplement.

The chair and members of the panel were selected by the Organizing Committee of the lifestyle modification recommendations to obtain a spectrum of health care professionals and scientists with expertise and interest in the areas of hypertension and alcohol consumption.

A MEDLINE search was performed using the terms ethyl alcohol and hypertension for English-language studies published between 1966 and 1996. Additional articles were identified by reviewing the reference lists of the identified articles, were found in the personal files of the panel members and were suggested by other experts. The principles for grading the evidence and the recommendations were based on those previously used by the Canadian Hypertension Society and are summarized in part 1 of this supplement. An attempt was made to reach consensus on all recommendations. The evidence and the recommendations were presented for comment to the other expert panels for this guidelines series, submitted for review to major Canadian organizations and presented at an international conference on preventive cardiology, to allow further national and international input. All revisions were reviewed and approved by the panel before incorporation into the final document. All randomized controlled trials examining the effect of alcohol on blood pressure that were identified through our search were included in the analysis.

To objectively assess the effect of alcohol on blood pressure as indicated by the results of randomized controlled trials, we examined data for blood pressure readings obtained with the patient in the sitting position (or the supine position if a reading for the sitting position was not available) after a 5-minute rest (or the closest time interval longer than 5 minutes if data were not available for 5 minutes). This method most closely approximates the current Canadian recommendations for determining blood pressure. We standardized the alcohol content of beverages as reported in the various studies such that one drink was considered to contain 13.6 g of ethanol (approximately the amount of ethanol in 1.5 fluid ounces of 40% spirits, 5 fluid ounces of 12% wine or 12 fluid ounces of 5% beer). Thus, the accuracy of the estimates of alcohol consumption in this review are limited by the methodology used to determine alcohol consumption, as well as by the design and reporting of the various studies.

Results

Effect of alcohol on blood pressure

We found extensive observational data on the association among alcohol consumption, blood pressure, cardiovascular complications, and specific and all-cause mortality rates. In addition, there were 14 randomized controlled trials examining the effect of changes in alcohol consumption on blood pressure. However, there were no randomized controlled trials designed primarily to determine the effect of a reduction in alcohol consumption on rates of hypertensive complications or death. Because of ethical considerations, it is impossible to study the effect of increasing alcohol consumption on blood pressure and cardiovascular complications in a randomized controlled study with extended follow-up. Therefore, most studies have taken place over a relatively short period and have been specifically designed to determine the effect of reducing alcohol consumption, usually from high levels.

The results of population cohort and cross-sectional studies have almost uniformly demonstrated a positive association between levels of alcohol consumption and blood pressure in both men and women. However, many of the studies found that, for people who consumed alcohol at low levels, blood pressure was no different from or was slightly lower than for those who abstained from alcohol use. Other studies have found that the association between alcohol consumption and blood pressure is linear, and some experts have suggested that reductions in blood pressure associated with low levels of alcohol consumption are related to methodologic problems. Studies in which no association was found between high alcohol consumption and increased blood pressure were, in general, smaller or subgroup analyses and may have lacked the power to detect an effect. High levels of alcohol consumption were a strong predictor of the development of high blood pressure in both men and women in most of the cohort studies.

The results of epidemiologic studies suggest that approximately 0% to 33% and 0% to 8% of high blood pressure in
men and women respectively is attributable to alcohol consumption. Building on the meta-analysis of English and associates, single and colleagues recently estimated age- and sex-specific etiologic fractions for hypertension attributable to alcohol consumption for Canadian men and women. They estimated that among men the etiologic fraction ranged from 4% (for those aged 60–64 years) to 9.2% (for those aged 20–24 years), whereas among women it ranged from 0.6% (for those aged 80–84 years) to 2.6% (for those aged 20–24 years). Although these studies do not prove cause and effect, the extent to which alcohol is associated with high blood pressure in a population is likely related in part to the quantity of alcohol consumed.

To facilitate both therapeutic and preventive interventions, we recommend that the alcohol consumption of all patients be assessed. This can be accomplished only by careful history-taking. The quantity, frequency and other characteristics of the use of wine, beer and distilled spirits should be ascertained. Standardized screening instruments are useful in assessing the likelihood of hazardous or problem drinking.

Recommendation

- It is recommended that health care professionals determine the alcohol consumption of their patients (grade D recommendation).

Alcohol consumption by normotensive adults

Table 1 summarizes the randomized controlled trials examining the effect of alcohol consumption on blood pressure in normotensive people.

<table>
<thead>
<tr>
<th>Study design</th>
<th>Subjects</th>
<th>Intervention</th>
<th>Duration</th>
<th>Estimated alcohol consumption, standard drinks/wk</th>
<th>Change in blood pressure, sitting or supine, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crossover²</td>
<td>45 men; normotensive</td>
<td>Low-alcohol beer</td>
<td>6 wk</td>
<td>20</td>
<td>16</td>
</tr>
<tr>
<td>2 × 2 factorial²</td>
<td>86 men; overweight</td>
<td>Low-alcohol beer; weight loss</td>
<td>18 wk</td>
<td>26</td>
<td>22</td>
</tr>
<tr>
<td>2 × 2 factorial²</td>
<td>72 men; sedentary</td>
<td>Exercise; low-alcohol beer</td>
<td>4 wk</td>
<td>28</td>
<td>25</td>
</tr>
<tr>
<td>Crossover²</td>
<td>8 men; normotensive</td>
<td>Alcohol v. no alcohol</td>
<td>4 d</td>
<td>34</td>
<td>34</td>
</tr>
<tr>
<td>Crossover²</td>
<td>10 men; normotensive</td>
<td>Alcohol v. no alcohol</td>
<td>7 d</td>
<td>28‡</td>
<td>28‡</td>
</tr>
<tr>
<td>Parallel²</td>
<td>641 men, 268 women; moderate to heavy drinkers§</td>
<td>Standardized advice, education</td>
<td>12 mo</td>
<td>Men 42, women 24</td>
<td>Men 6.7, women 3.5</td>
</tr>
<tr>
<td>Crossover²</td>
<td>5 men, 5 women; normotensive</td>
<td>Alcohol v. no alcohol</td>
<td>4 d</td>
<td>&gt; 21</td>
<td>41</td>
</tr>
</tbody>
</table>

Note: SBP = systolic blood pressure, DBP = diastolic blood pressure, NP = data not provided.

*One standard drink contains 13.6 g of ethanol. This is approximately the amount of ethanol in 1.5 fluid ounces of spirits (40%), 12 fluid ounces of beer (5%) or 5 fluid ounces of wine (12%). The accuracy of estimates of alcohol consumption are limited by study methodology, design and reporting of results. Reduction in consumption is relative to control group.

† Effects of alcohol alone.

The study population included people with hypertension.
the consent process or another aspect of entry into the clinical trial led to a reduction in alcohol consumption. This appears to have been the major factor in the lack of difference in blood pressure reduction for the largest randomized controlled study examining this issue.106 Some people have difficulty reducing alcohol consumption, so some hypertensive patients in the studies may not have adhered to the alcohol reduction protocol, even though they reported a reduction in alcohol consumption when questioned directly. This phenomenon could account for the lack of effectiveness of alcohol restriction in some studies.

We also examined evidence from uncontrolled clinical interventions, community-based programs, and case-control and twin studies34,112–119 (level III evidence), along with the data from the cross-sectional and cohort studies previously cited. Most of these studies showed a strong association between heavy alcohol consumption and high blood pressure and provided evidence consistent with the conclusion that a reduction in alcohol consumption may lead to a reduction in blood pressure.

Recommendation

• For hypertensive patients, it is recommended that alcohol consumption be in accordance with Canadian low-risk drinking guidelines (i.e., healthy adults who choose to drink should limit alcohol consumption to 2 or fewer standard drinks per day, with consumption not exceeding 14 standard drinks per week in men and 9 standard drinks per week in women) (grade C recommendation).

Multiple-intervention studies

A reduction in alcohol consumption was attempted in 3 randomized controlled trials120–122 that simultaneously examined several lifestyle interventions to reduce blood pressure or prevent hypertension. In two of these studies120,121 there were similar reductions in alcohol consumption in the intervention and control groups (i.e., no significant effect of the intervention on alcohol consumption), and there was no association between change in alcohol consumption and change in blood pressure. In the third study122 the intervention resulted in a reduction in alcohol intake, and there was an association between reduction in alcohol intake and reduction in systolic blood pressure. These studies do not provide strong evidence but are consistent with the conclusion that heavy alcohol consumption leads to increased blood pressure.

Interpretation

The results of randomized controlled trials involving normotensive adults indicate a causal association between alcohol consumption above recommended levels and increases in blood pressure. Prospective cohort studies have shown a strong association between heavy alcohol consumption and the development of hypertension in men and women. The association between blood pressure and alcohol consumption of fewer than 2 standard drinks per day is not clear, as observational studies have variously found increases, decreases and no change in blood pressure.

Table 2: Randomized controlled trials examining the effect of alcohol consumption on blood pressure in hypertensive patients

<table>
<thead>
<tr>
<th>Study design</th>
<th>Subjects</th>
<th>Intervention</th>
<th>Duration</th>
<th>Estimated alcohol consumption, standard drinks/wk</th>
<th>Change in blood pressure, sitting or supine, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parallel22,106</td>
<td>375 normotensive; 266 hypertensive</td>
<td>Alcohol reduction; cognitive behaviour program</td>
<td>2 yr</td>
<td>32</td>
<td>8.4</td>
</tr>
<tr>
<td>2 × 2 factorial23</td>
<td>59 hypertensive men</td>
<td>Low-alcohol beer; low salt</td>
<td>4 wk</td>
<td>32</td>
<td>28</td>
</tr>
<tr>
<td>Parallel23</td>
<td>41 hypertensive men</td>
<td>Advice to decrease alcohol intake</td>
<td>8 wk</td>
<td>44</td>
<td>22</td>
</tr>
<tr>
<td>Crossover23</td>
<td>44 hypertensive men</td>
<td>Low-alcohol beer</td>
<td>6 wk</td>
<td>26</td>
<td>23</td>
</tr>
<tr>
<td>Crossover23</td>
<td>54 hypertensive men</td>
<td>Advice to maintain or decrease alcohol consumption</td>
<td>3 wk</td>
<td>23</td>
<td>12</td>
</tr>
<tr>
<td>Parallel23</td>
<td>49 hypertensive men</td>
<td>Advice to abstain from or reduce alcohol consumption</td>
<td>2 wk</td>
<td>30</td>
<td>19</td>
</tr>
<tr>
<td>Parallel23</td>
<td>123 men, 6 women; all hypertensive</td>
<td>Training of physician</td>
<td>1 yr</td>
<td>49</td>
<td>8</td>
</tr>
</tbody>
</table>

Note: NS = nonsignificant. *Effects of alcohol alone.
Randomized controlled trials of reduction in alcohol intake indicate that this is an efficacious means of reducing blood pressure in normotensive people. The intervention data for patients with hypertension, while inconclusive, are consistent with such an effect. We deliberately chose to present data on blood pressure determined in the sitting or supine position after 5 minutes rest (or the closest approximation to this interval) to be consistent with current recommendations on how blood pressure should be determined. In some of the original studies,23–28,106 more dramatic reductions in blood pressure were reported because the method of determining blood pressure was not consistent with these recommendations.

Our recommendations concerning alcohol use and the prevention and treatment of hypertension are based on a number of considerations. First, it cannot be established whether the blood pressure of people who consume alcohol at low levels is lower or higher than that of nondrinkers. Current Canadian low-risk drinking guidelines take into account a wide range of outcomes associated with alcohol use, balancing both risks and benefits. For most consequences (e.g., diseases of the liver, pancreas and nervous system, hemorrhagic stroke, and various cancers) risk increases with alcohol consumption.107 For accidents and injuries, as well as for adverse effects on social well being, lower consumption is associated with lower risk. Conversely, for ischemic heart disease, alcohol appears to be protective over a wide range of consumption levels, from less than 4 drinks per week to 5 or 6 drinks per day, at least in older people.14,29,32–35 However, within this range, ischemic heart disease does not appear to diminish with increased intake, and most of the protective effect is associated with drinking very small amounts of alcohol. The association between alcohol consumption and ischemic stroke is less clear.114 The lowest risks of overall illness and death are associated with alcohol consumption within the limits recommended in the Canadian guidelines on low-risk drinking.15,44,46,70,118,129–131,135–141

It is widely recognized that some people should not use alcohol at all (e.g., those with a personal or family history of serious drinking problems, those with liver disease and those receiving medications that interact with alcohol). However, with regard to the prevention and treatment of hypertension, there were insufficient data to support a general recommendation about abstinence. Similarly, a general recommendation that hypertensive or normotensive people who currently abstain from drinking alcohol should begin consuming small amounts of alcohol to protect against ischemic heart disease and stroke was not supported by the panel. The panel agreed that a careful assessment of the patient’s characteristics and his or her risk factors and circumstances by the health care provider was appropriate as a basis for individual advice.

Validation

These recommendations are consistent with those of the World Hypertension League,14 the National High Blood Pressure Education Program Working Group on Primary Prevention of Hypertension1 and previous recommendations of the Canadian Coalition for High Blood Pressure Prevention and Control14,12 and the Canadian Hypertension Society.1 The low-risk drinking guidelines are those jointly recommended by the Addiction Research Foundation of Ontario and the Canadian Centre on Substance Abuse in a 1997 policy statement and endorsed by the Canadian College of Family Physicians and other agencies, including provincial associations. These guidelines build on an earlier set of guidelines produced by the same 2 organizations, which were endorsed by the Canadian Medical Association, the Royal College of Physicians and Surgeons of Canada and the Canadian Medical Society on Alcohol and Other Drugs.144

Future research

More research on the association between alcohol consumption and blood pressure is needed. Many of the randomized controlled trials we reviewed included few women; future research must be designed to ensure adequate representation of both sexes. Several studies have suggested that the pattern of drinking, for example continuous or binge drinking48,74,93,100 and infrequent or frequent low-level consumption, may be important.95 Future studies should closely examine the effect of timing of blood pressure determination in relation to most recent alcohol consumption and would benefit from the inclusion of ambulatory blood pressure monitoring over several days.

Further studies examining the additive effects of several lifestyle interventions, including reduction in alcohol consumption, are needed. In addition, studies should include measurements of the side effects of the interventions.

More care is required in study design to address the possibility of similar reductions in alcohol consumption in both the intervention and the control groups. The possibility of reporting bias indicates a need for objective measures of alcohol consumption (e.g., levels of γ-glutamyltransferase).

Finally, research into the public health impact in Canada of hypertension attributable to alcohol consumption, including the economic costs, should be a priority. The recent study by Single and colleagues104 provides a prototype for such research.

Conclusion

Assessment of alcohol intake should be an important part of routine medical assessments.

For those who consume large quantities of alcohol, a reduction in consumption will reduce blood pressure and have other beneficial health effects. Adherence to low-risk drinking guidelines is an important nonpharmacologic manoeuvre to prevent and control hypertension.

We are grateful for the external reviews of Drs. Eric Single, Gre-
gory Taylor, Elinor Wilson and Nady el-Guebaly, as well as those of the Canadian Council of Cardiovascular Nurses, the Canadian Nurses Association, the Canadian Public Health Association, the College of Family Physicians of Canada, the Heart and Stroke Foundation of Canada, Hooecht Marion Roussel and Merck Frosst Canada Inc. The financial assistance of the Laboratory Centre for Disease Control at Health Canada and Astra Pharma Inc., Bayer Inc., Bristol-Meyers Squibb Pharmaceutical Group, Knoll Pharma Inc., Merck Frosst Canada Inc., Searle Canada Inc. and Servier Canada Inc. is gratefully acknowledged, as is the secretarial assistance of Leslie Holmes.

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References

Alcohol consumption


4. Recommendations on physical exercise training

Jean Cléroux, PhD; Ross D. Feldman, MD, PhD; Robert J. Petrella, MD, PhD

Abstract

Objective: To provide updated, evidence-based recommendations for health care professionals concerning the effects of regular physical activity on the prevention and control of hypertension in otherwise healthy adults.

Options: People may engage in no, sporadic or regular physical activity that may be of low, moderate or vigorous intensity. For sedentary people with hypertension, the options are to undertake or maintain regular physical activity and to avoid or moderate medication use; to use another lifestyle modification technique; to commence or continue antihypertensive medication; or to take no action and remain at increased risk of cardiovascular disease.

Outcomes: The health outcomes considered were changes in blood pressure and in morbidity and mortality rates. Because of insufficient evidence, no economic outcomes were considered.

Evidence: A MEDLINE search was conducted for the period 1966–1997 with the terms exercise, exertion, physical activity, hypertension and blood pressure. Both reports of trials and review articles were obtained. Other relevant evidence was obtained from the reference lists of these articles, from the personal files of the authors and through contacts with experts. The articles were reviewed, classified according to study design and graded according to level of evidence.

Values: A high value was placed on avoidance of cardiovascular morbidity and premature death caused by untreated hypertension.

Benefits, harms and costs: Physical activity of moderate intensity involving rhythmic movements with the lower limbs for 50–60 minutes, 3 or 4 times per week, reduces blood pressure and appears to be more effective than vigorous exercise. Harm is uncommon and is generally restricted to the musculoskeletal injuries that may occur with any repetitive activity. Injury occurs more often with jogging than with walking, cycling or swimming. The costs include the costs of appropriate shoes, garments and equipment, but these were not specifically measured.

Recommendations: (1) People with mild hypertension should engage in 50–60 minutes of moderate rhythmic exercise of the lower limbs, such as brisk walking or cycling, 3 or 4 times per week to reduce blood pressure. (2) Exercise should be prescribed as an adjunctive therapy for people who require pharmacologic therapy for hypertension, especially those who are not receiving β-blockers. (3) People who do not have hypertension should participate in regular exercise as it will decrease blood pressure and reduce the risk of coronary artery disease, although there is no direct evidence that it will prevent hypertension.

Validation: These recommendations agree with those of the World Hypertension League, the American College of Sports Medicine, the report of the US Surgeon General on physical activity and health, and the US National Institutes of Health Consensus Development Panel on Physical Activity and Cardiovascular Health. These guidelines have not been clinically tested.

Sponsors: The Canadian Hypertension Society, the Canadian Coalition for High Blood Pressure Prevention and Control, the Laboratory Centre for Disease Control at Health Canada, and the Heart and Stroke Foundation of Canada.

The 1989 Canadian Consensus Conference on Non-pharmacologic Approaches to the Management of High Blood Pressure observed that the evidence available at that time on the relation between physical activity and hypertension was not strong. It was concluded that “although there is evidence that regular aerobic activity may result in the lowering of blood pressure in patients or clients with 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.” Since that time, evidence has become available to address those concerns. This report updates the previous recommendations.
There are 2 major types of exercise: rhythmic or dynamic and resistive. Rhythmic or dynamic exercise consists of repeated low-resistance movements (e.g., walking or cycling), whereas resistive exercise typically consists of a small number of high-resistance movements (e.g., weight lifting). Most research on hypertension and exercise examines dynamic exercise training.

In assessing exercise, both the frequency and the intensity of the exercise are important. Exercise training is generally categorized as being of low intensity (less than 45% of maximal oxygen uptake), moderate intensity (45% to 60% of maximal oxygen uptake), vigorous intensity (61% to 75% of maximal oxygen uptake) and strenuous intensity (greater than 75% of maximal oxygen uptake). Moderate-intensity exercise, for example, corresponds to an exercise that elicits 60% to 70% of maximal heart rate. In a typical 40-year-old Canadian (of either sex), this corresponds to a heart rate of 110 to 125 beats/min. This can typically be attained by cycling on a stationary bicycle at 75 to 100 W or walking briskly, at 5 to 6 km/h (3 to 4 mi/h). Vigorous-intensity exercise corresponds to activities that elicit 71% to 85% of maximal heart rate or 126 to 150 beats/min in a typical 40-year-old Canadian. Such levels can be achieved by cycling on a stationary bicycle at 120 to 150 W or by jogging at 6 to 8 km/h (4 to 5 mi/h).

The reader is referred to the compendium of physical activities by Ainsworth and coworkers for detailed information on the energy equivalents of several physical activities. Briefly, energy equivalents are measured in METs, which are units of metabolism. At rest, a person spends 1 MET of energy to maintain body functions. Moderate-intensity exercise corresponds to an energetic equivalent of approximately 4 METs for women and 6 METs for men. Vigorous-intensity exercise corresponds on average to 6 METs for women and 8 METs for men (for a synopsis table of these energy expenditures, see Table 2 in Pate and associates). In terms of caloric equivalents, three 55-minute exercise sessions per week (for a weekly total of 165 minutes) at a moderate intensity correspond to an energy expenditure of approximately 600 kcal (2500 kJ) per week for a 55-kg woman and 1200 kcal (5000 kJ) per week for a 70-kg man. Exercising for the same weekly duration at a vigorous intensity represents an energy expenditure of approximately 900 kcal (3800 kJ) per week for a 55-kg woman and 1500 kcal (6300 kJ) per week for a 70-kg man.

The primary objective of the guidelines presented here is to summarize the evidence of the relation between exercise and hypertension, as well as the response of blood pressure to exercise and to advise health care professionals and the public accordingly.

Methods

A complete description of the methods used in developing these guidelines is given in part 1 of this supplement.

The chair and the members of the panel were selected by the Organizing Committee for the lifestyle modification recommendations to obtain a spectrum of health care professionals and scientists with expertise and interest in the areas of hypertension, exercise and health.

A MEDLINE search was performed using the terms exercise, exertion, physical activity, hypertension and blood pressure for English-language studies published between January 1966 and July 1997 and was limited to exercise training trials in hypertensive humans. Additional articles were identified by reviewing the reference lists of the identified articles, were found in the personal files of the panel members and were suggested by other experts. The definition of hypertension in the selected papers varied according to the criteria adopted by the authors.

We included all primary evidence from randomized crossover or controlled trials, as well as nonrandomized controlled trials. We also examined trials that included a detraining control period or an observation control period of at least 4 weeks (during which blood pressure was monitored weekly) in which the exercise interventions lasted at least 4 weeks. The work of the panel was aided by several reports, including the 1996 US Surgeon General’s report on physical activity and health, the 1996 National Institutes of Health (NIH) consensus statement on physical activity and cardiovascular health, the 1994 position statement issued by the American College of Sports Medicine and the 1991 consensus statement of the World Hypertension League. Other non-trial materials reviewed by the panel included a meta-analysis and 5 reviews or book chapters, one of which included a simplified meta-analysis.

The principles for grading the evidence and developing recommendations were based on those previously used by the Canadian Hypertension Society and are summarized in part 1 of this supplement. An attempt was made to reach consensus on all recommendations. The evidence and the recommendations were presented for comment to the other expert panels, submitted for review to major Canadian organizations and presented at an international conference on preventive cardiology, to allow further national and international input. All revisions were reviewed and assessed by the panel before incorporation into the final document.

Results

Regular exercise and mild hypertension

There is consistent evidence that regular rhythmic physical exercise of the lower extremities decreases both systolic and diastolic blood pressure by 5–7 mm Hg independent of weight loss, alcohol intake or salt intake. Using our established criteria, we identified 26 studies comprising 35 groups or interventions that measured the effects of physical training on blood pressure in a total of 486 patients with mild to moderate hypertension who were not taking any medication (Table 1). Most of these studies measured blood pressure with the patient in a seated position; 5 of them also recorded ambulatory blood pressure. One additional open trial recorded only ambulatory blood pressure.

Study design and setting

Randomized studies of the effect of exercise training on blood pressure (level II studies, a total of 18 groups or
interventions) found mean reductions in blood pressure (by 5/7 mm Hg) comparable to those reported in non-randomized studies (level III and IV studies, a total of 17 groups or interventions) (for which the mean reduction in blood pressure was 7/9 mm Hg) (Table 1). This observation agrees with that of Fagard,⁴⁴ who found that the amplitude of the reduction in blood pressure with exercise training did not usually differ with study design.

### Table 1: Longitudinal studies on the effects of physical exercise training on clinic and/or ambulatory blood pressure (BP) in subjects with essential hypertension

<table>
<thead>
<tr>
<th>Study design</th>
<th>Level of evidence*</th>
<th>Subjects</th>
<th>Intervention group</th>
<th>Training program</th>
<th>Change in BP after training, mm Hg</th>
<th>RCT†, **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinic BP (seated unless specified otherwise)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RXT†</td>
<td>II 7 men, 6 women</td>
<td>44 Cycle</td>
<td>4</td>
<td>3</td>
<td>05 65</td>
<td>143 96</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 8 men, 1 woman</td>
<td>41 Cycle</td>
<td>10</td>
<td>3</td>
<td>45 13</td>
<td>50</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 8</td>
<td>46 Cycle</td>
<td>10</td>
<td>3</td>
<td>60 180 (50)</td>
<td>154 93</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 5 men, 1 woman</td>
<td>44 Comb</td>
<td>12</td>
<td>3</td>
<td>50 150 70</td>
<td>161 105</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 12 men</td>
<td>42 Comb</td>
<td>16</td>
<td>3</td>
<td>50 150 (56)</td>
<td>145 99</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 44 men</td>
<td>30 Walk-jog</td>
<td>16</td>
<td>3</td>
<td>60 180 (64)</td>
<td>146 94</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 4 men, 6 women</td>
<td>51 Cycle</td>
<td>10</td>
<td>3</td>
<td>80 180 (50)</td>
<td>156 103</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 21</td>
<td>46 Cycle</td>
<td>10</td>
<td>3</td>
<td>60 180 (50)</td>
<td>154 100</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 11</td>
<td>65 Walk-jog</td>
<td>37</td>
<td>1</td>
<td>51 158</td>
<td>164 94</td>
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<tr>
<td>RXT†</td>
<td>II 10 men</td>
<td>44 Comb</td>
<td>10</td>
<td>4</td>
<td>120</td>
<td>137 95</td>
</tr>
<tr>
<td>RXT†</td>
<td>b 72 Walk-jog</td>
<td>26</td>
<td>3</td>
<td>120</td>
<td>156 86</td>
<td>–8</td>
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<tr>
<td>RXT†</td>
<td>II 24 men, 15 women</td>
<td>44 Walk-jog</td>
<td>16</td>
<td>2.5</td>
<td>55 138</td>
<td>141 95</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 6</td>
<td>45 Walk-jog</td>
<td>12</td>
<td>3</td>
<td>50 135 45</td>
<td>140 93</td>
</tr>
<tr>
<td>RXT†</td>
<td>II 7</td>
<td>39 Walk-jog</td>
<td>12</td>
<td>3</td>
<td>50 135 71</td>
<td>91 4</td>
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<td>NRCT†</td>
<td>III 12 men</td>
<td>41 Walk-jog</td>
<td>12</td>
<td>2.5</td>
<td>88 (67)</td>
<td>148 97</td>
</tr>
<tr>
<td>NRCT†</td>
<td>III 10</td>
<td>56 Comb</td>
<td>12</td>
<td>2.5</td>
<td>105 (69)</td>
<td>168 100</td>
</tr>
<tr>
<td>NRCT†</td>
<td>III 12 men, 5 women</td>
<td>51 Comb</td>
<td>63</td>
<td>3</td>
<td>120 (55)</td>
<td>155 101</td>
</tr>
<tr>
<td>NRCT†</td>
<td>III 9 men, 5 women</td>
<td>61 Walk-jog</td>
<td>26</td>
<td>3.5</td>
<td>14 144 (58)</td>
<td>141 92</td>
</tr>
<tr>
<td>NRCT†</td>
<td>III 10</td>
<td>63 Walk-jog</td>
<td>52</td>
<td>3.6</td>
<td>180 (64)</td>
<td>143 92</td>
</tr>
<tr>
<td>NRCT†</td>
<td>II 10 women</td>
<td>49 Cycle</td>
<td>10</td>
<td>3</td>
<td>60 180 (50)</td>
<td>150 95</td>
</tr>
<tr>
<td>NRCT†</td>
<td>II 2 men, 15 women</td>
<td>48 Cycle</td>
<td>10</td>
<td>3</td>
<td>60 180 (50)</td>
<td>159 96</td>
</tr>
<tr>
<td>NRCT†</td>
<td>II 7 men, 5 women</td>
<td>47 Swim</td>
<td>10</td>
<td>3</td>
<td>45 15 (66)</td>
<td>150 96</td>
</tr>
<tr>
<td>DCP7</td>
<td>IV 60 men, 17 women</td>
<td>54 Comb</td>
<td>5</td>
<td>7</td>
<td>90 630 (50)</td>
<td>157 98</td>
</tr>
<tr>
<td>DCP7</td>
<td>IV 14 men, 2 women</td>
<td>35 Walk-jog</td>
<td>26</td>
<td>3.5</td>
<td>154 NA</td>
<td>148 52</td>
</tr>
<tr>
<td>OCP†</td>
<td>IV 27 women</td>
<td>55 Walk-jog</td>
<td>12</td>
<td>3</td>
<td>10 90 (59)</td>
<td>179 113</td>
</tr>
<tr>
<td>OCP†</td>
<td>IV 5 men, 7 women</td>
<td>46 Cycle</td>
<td>10</td>
<td>3</td>
<td>60 180 (50)</td>
<td>153 103</td>
</tr>
<tr>
<td>OCP†</td>
<td>IV 9</td>
<td>46 Cycle</td>
<td>20</td>
<td>3</td>
<td>60 180 (50)</td>
<td>157 104</td>
</tr>
<tr>
<td>OCP†</td>
<td>IV 8</td>
<td>46 Cycle</td>
<td>16</td>
<td>3</td>
<td>10 90 (70)</td>
<td>131 89</td>
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<td>OCP*</td>
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<td>DCP*</td>
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</table>

**Note:** NA = not available; RXT = randomized crossover trial; RCT = randomized controlled trial; NRCT = non-randomized controlled trial; DCP = the same subjects were studied after a detraining control period of at least 4 weeks; OT = open trial; SBP = systolic blood pressure; DBP = diastolic blood pressure.

*Level of evidence according to the Canadian Hypertension Society consensus conference.

†Mode of training: cycle = stationary cycle ergometer; comb = combination of cycle and walking/jogging.

‡Training intensity as percent maximal oxygen uptake; values in parentheses estimated from lactate threshold or from a fixed lactate level; values in square brackets estimated from target exercise heart rate according to the formula of Miller and associates.⁴³

§Significant changes reported by authors.

**Significant blood pressure group.

†‡†Subjects from the control regimen who then trained.
The results of exercise training did not seem to be affected by the setting. Indeed, several studies used home training programs and found comparable reductions in blood pressure to those in which subjects trained under staff supervision. Type of exercise

Very few studies have examined the effect of resistive training on blood pressure. A recent review of resistive training (consisting of 3 sessions of circuit weight training per week) identified 3 randomized controlled trials in hypertensive patients, of which only one study reported a significant decrease in diastolic blood pressure (and in that study the decrease was less than 3 mm Hg). We therefore conclude that resistive exercise is not very effective in reducing blood pressure. Most of the studies we identified examined the effect of regular dynamic exercise. The recommendations presented here are therefore based on this type of exercise. The training modes were stationary cycling, walking or jogging (and swimming in one study only).

Duration of program

Two studies found significant reductions in blood pressure after only 4 and 5 weeks of training respectively. In another study approximately 75% of the antihypertensive effect found after 20 weeks of exercise training occurred in the first 10 weeks. The antihypertensive effect of training persisted as long as the training program (over 1 year in the studies reviewed). In contrast, the antihypertensive effect was no longer seen after detraining periods of 10 weeks. The antihypertensive effect of training is therefore reversible.

Frequency of sessions

The antihypertensive effect of exercise training is observed when the patient follows a schedule consisting of 3 sessions per week and increases little, if at all, when the patient follows a daily routine. Approximately 75% of the antihypertensive effect that can be obtained by exercising 7 times per week is achieved by exercising only 3 times per week. In studies in which subjects trained at 50% of maximal oxygen uptake, the antihypertensive effects of 3 sessions per week were comparable to those of 7 sessions per week. Thus, daily exercise is not essential to obtain an antihypertensive effect.

Duration of sessions

In the studies examined, the exercise sessions were 30–90 minutes long. Of the 35 groups or interventions from the 26 studies, 31 used moderate- to vigorous-intensity training with cycling, walking or jogging, on a schedule of 2.5 to 4 sessions per week. In 17 of these groups or interventions, the subjects trained no longer than 45 minutes per session (mean 38 [range 30–45] minutes). In 11 (67%) of these 17 groups or interventions, there were significant decreases in systolic or diastolic blood pressure, or both. Of the 14 groups or interventions in which the intervention group trained for longer than 45 minutes per session (mean 57 [range 50–60] minutes), 13 (94%) had significant decreases in systolic or diastolic blood pressure, or both. Therefore, training for 50–60 minutes, at moderate to vigorous intensity, is more likely to produce a significant antihypertensive effect than training for 30–45 minutes.

Intensity of exercise

There is evidence that moderate-intensity exercise may be more effective than vigorous exercise in decreasing blood pressure in hypertensive patients. In 17 of the 31 groups or interventions, subjects trained at moderate intensity (mean 53% [range 45% to 60%] of maximal oxygen up-
take). Significant decreases in systolic or diastolic blood pressure, or both, were reported in 15 (88%) of these 17 groups or interventions. Of the 14 groups or interventions in which subjects trained at vigorous or strenuous intensities (mean 70% [range 64% to 80%] of maximal oxygen uptake), only 9 (64%) reported significant decreases in systolic or diastolic blood pressure, or both. In studies that examined the effects of 2 training intensities, blood pressure measured in a clinical setting decreased to a greater extent in the subjects who trained at a moderate intensity than in those who trained vigorously. One study found no difference in blood pressure changes between moderate- and vigorous-intensity exercise programs. Another study reported a significant decrease in mean daytime ambulatory blood pressure after training at 50% of maximal oxygen uptake but not after training at 70% of maximal oxygen uptake. The evidence reviewed therefore indicates that training at a moderate intensity produces more significant antihypertensive effects than training at a vigorous intensity.

A corollary to this observation is that the effect of physical activity in reducing blood pressure does not depend on increasing maximal oxygen uptake, which typically occurs with training above 60% of maximal oxygen uptake. Several studies that used training intensities below 55% of maximal oxygen uptake found significant reductions in clinical and ambulatory blood pressure without any significant changes in maximal oxygen uptake.

Effect of age, sex and race

Age does not seem to have any bearing on the antihypertensive effects of exercise. In the studies reviewed, similar reductions in blood pressure were observed in younger (e.g., 30-35-year-old subjects) and older (e.g., 60-70-year-old subjects) age groups (reductions of 11/7 and 10/11 mm Hg respectively). This finding agrees with the results of Fagard and Tipton, who reported that training-induced changes in systolic and diastolic blood pressure did not correlate with age.

For the studies listed in Table 1 that specified the numbers of men and women, there were in total 125 (43%) women and 168 (57%) men. For the studies in which the subjects were exclusively or predominantly women, the decrease in blood pressure after training was comparable to that reported for studies of men.

Race does not appear to influence the effect of regular exercise in reducing blood pressure; comparable antihypertensive effects have been seen among black American, Japanese and white subjects.

Recommendations

- For people with mild hypertension, dynamic exercise (including walking, cycling, noncompetitive swimming and other equivalent leisure activities) should be prescribed to reduce blood pressure (grade B recommendation).
- Moderate-intensity dynamic exercise, in sessions of 50-60 minutes, 3 or 4 times per week, is preferable to vigorous-intensity exercise, as moderate-intensity exercise appears to be more effective in reducing blood pressure (grade B recommendation).
not require pharmacologic therapy was greater than the proportion of the control group that did not.52

Recommendation

- Exercise should be prescribed as adjunctive therapy for people who require pharmacologic therapy for hypertension, especially for those who are not receiving β-blockers (grade B recommendation).

Regular exercise in normotensive individuals

To evaluate the relation between blood pressure and physical exercise training in normotensive individuals, this panel relied mainly on a recent meta-analysis.53 (The major longitudinal cohort studies that address this topic54,55 were used for background reference only.) That review of 35 human clinical training studies involving a total of 800 intervention subjects and 276 control subjects found that physical training reduced blood pressure in normotensive people.

In patients with established atherosclerotic disease, numerous randomized controlled trials have indicated that exercise training reduces total, cardiovascular and re-infarc-
tion mortality rates (level I evidence) (reviewed by Oldridge and associates,56 O'Connor and collaborators57 and Berlin and Colditz58). In a recent randomized controlled trial, Hambrecht and coworkers59 reported significantly lower rates of progression of coronary atherosclerotic lesions in an exercise group than in a sedentary control group of patients with coronary artery disease (level I evidence).

Mild-intensity exercise training also has beneficial effects on the lipid profile. Here again, the metabolic changes associated with training are related more to the training volume than to the improvement in maximal oxygen uptake.60

Recommendations

- For people who do not have hypertension or coronary artery disease, exercise training is beneficial because it reduces blood pressure (grade B recommendation) and may reduce the risk of coronary artery disease (grade D recommendation).
- People who do not have hypertension but who do have established atherosclerotic disease should become physically active to reduce the risk of death from cardiovascular disease, from re-infarction and from other causes (grade A recommendation).

The recommendations presented in this paper are summarized in Table 2.

Adverse effects

A few studies have provided some insight into adverse events related to activities commonly undertaken to increase physical activity. Most running-related injuries involve the leg and the foot, are self-correcting in a relatively short time and are related to distance running.61,62 The injury rate appears to increase sharply among people taking more than 4 aerobic classes per week.63 Exertion may provoke asthmatic attacks, which usually occur after exercise in susceptible individuals. People with underlying cardiovascular disease may experience angina or acute myocardial infarction during vigorous activity. The US Surgeon General’s report concluded that, “compared with sedentary people who suddenly begin exercising vigorously, persons who exercise regularly have a lower risk of exercise-related sudden death, although even this group has a transient elevation of risk during and immediately after vigorous exercise. Nonetheless, the net effect of regular physical activity is to decrease the risk of cardiac death.”

Interpretation

There is now excellent evidence that mild hypertension can be treated with moderate physical activity. The antihypertensive effect of exercise does not depend on an increase in maximal aerobic capacity, but does correlate with the initial level of activity. The lower the initial level of activity, the greater the expected reduction in blood pressure associated with a given increase in physical activity. This statement on physical activity and blood pressure agrees with the more general US recommendations on physical activity and public health.4

The present recommendations differ from those published in 19901 mainly because of evidence from a relatively large number of well-controlled studies published since the late 1980s. In addition, our review of the literature identified several studies published before 1990 that were not included in the original review. Thus, the main concerns expressed in the earlier recommendations have been addressed by well-controlled studies on physical exercise and hypertension.

Validation

The present recommendations agree with earlier guidelines indicating that exercise training can reduce blood pressure in hypertensive patients, specifically those of the World Hypertension League,9 the American College of Sports Medicine,4 the US Surgeon General6 and the NIH Consensus Development Panel on Physical Activity and Cardiovascular Health.7 In accordance with those guidelines, we believe that the evidence indicates that moderate exercise is at least as effective as vigorous exercise for the treatment of hypertension.

Future research

The panel on exercise training and hypertension suggests that additional research is required in a number of areas.

Future research should determine if there are differ-
ences in the additive effects of (1) specific exercise and drug combinations (between and within classes of medication) and (2) combinations of exercise and other non-pharmacologic treatments (e.g., weight loss and stress reduction).

More research is needed to determine if exercise-induced reductions in blood pressure lead to a reduction in various endpoints of cardiovascular disease.

Finally, prospective trials should be conducted to determine if regular exercise in previously sedentary people can prevent hypertension.

### Conclusion

Regular dynamic physical exercise, which need not be vigorous and which can be gradually incorporated into everyday activities, can reduce blood pressure by 5 to 10 mm Hg in people with hypertension. Brisk walking is a typical moderate-intensity exercise that can lead to such reductions when practised for 3 hours per week. In addition to reducing hypertension, physical activity improves other cardiovascular risk factors.

We are grateful for the external reviews of the Canadian Council of Cardiovascular Nurses, the Canadian Nurses Association, the Canadian Pharmacists Association, the Canadian Public Health Association, the College of Family Physicians of Canada, the Heart and Stroke Foundation of Canada, Hoehct Marion Roussel and Merck Frosst Canada Inc.

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Competing interests: None declared.

### References

5. Recommendations on dietary salt

J. George Fodor, MD, PhD; Beverly Whitmore, RD; Frans Leenen, MD, PhD; Pierre Larochelle, MD

Abstract

Objective: To provide updated, evidence-based recommendations concerning the effects of dietary salt intake on the prevention and control of hypertension in adults (except pregnant women). The guidelines are intended for use in clinical practice and public education campaigns.

Options: Restriction of dietary salt intake may be an alternative to antihypertensive medications or may supplement such medications. Other options include other nonpharmacologic treatments for hypertension and no treatment.

Outcomes: The health outcomes considered were changes in blood pressure and in morbidity and mortality rates. Because of insufficient evidence, no economic outcomes were considered.

Evidence: A MEDLINE search was conducted for the period 1966–1996 using the terms hypertension, blood pressure, vascular resistance, sodium chloride, sodium, diet, sodium or sodium chloride dietary, sodium restricted/reducing diet, clinical trials, controlled clinical trial, randomized controlled trial and random allocation. Both trials and review articles were obtained, and other relevant evidence was obtained from the reference lists of the articles identified, from the personal files of the authors and through contacts with experts. The articles were reviewed, classified according to study design and graded according to level of evidence. In addition, a systematic review of all published randomized controlled trials relating to dietary salt intake and hypertension was conducted.

Values: A high value was placed on the avoidance of cardiovascular morbidity and premature death caused by untreated hypertension.

Benefits, harms and costs: For normotensive people, a marked change in sodium intake is required to achieve a modest reduction in blood pressure (there is a decrease of 1 mm Hg in systolic blood pressure for every 100 mmol decrease in daily sodium intake). For hypertensive patients, the effects of dietary salt restriction are most pronounced if age is greater than 44 years. A decrease of 6.3 mm Hg in systolic blood pressure and 2.2 mm Hg in diastolic blood pressure per 100 mmol decrease in daily sodium intake was observed in people of this age group. For hypertensive patients 44 years of age and younger, the decreases were 2.4 mm Hg for systolic blood pressure and negligible for diastolic blood pressure. A diet in which salt is moderately restricted appears not to be associated with health risks.

Recommendations: (1) Restriction of salt intake for the normotensive population is not recommended at present, because of insufficient evidence demonstrating that this would lead to a reduced incidence of hypertension. (2) To avoid excessive intake of salt, people should be counselled to choose foods low in salt (e.g., fresh fruits and vegetables), to avoid foods high in salt (e.g., pre-prepared foods), to refrain from adding salt at the table and minimize the amount of salt used in cooking, and to increase awareness of the salt content of food choices in restaurants. (3) For hypertensive patients, particularly those over the age of 44 years, it is recommended that the intake of dietary sodium be moderately restricted, to a target range of 90–130 mmol per day (which corresponds to 3–7 g of salt per day). (4) The salt consumption of hypertensive patients should be determined by interview.

Validation: These recommendations were reviewed by all of the sponsoring organizations and by participants in a satellite symposium of the fourth International Conference on Preventive Cardiology. They have not been clinically tested.

Sponsors: The Canadian Hypertension Society, the Canadian Coalition for High Blood Pressure Prevention and Control, the Laboratory Centre for Disease Control at Health Canada, and the Heart and Stroke Foundation of Canada.

Hypertension is an important part of the etiology and pathogenesis of myocardial infarction, cerebrovascular accidents, congestive heart failure and renal failure. In Canada approximately one-fifth of the adult population has high blood pressure. Epidemiologic, clinical and experimental studies suggest that ingestion of a diet habitually high in salt plays a role in the etiology and pathogenesis of hypertension.
the largest quantities of salt now consumed in North America originate from industrially processed food. Only 20% to 30% of total dietary sodium consumption is discretionary — or consumer-controlled — through the addition of salt to food after its preparation. The rest is derived from naturally occurring sources or commercial processes.

The question of whether restriction of dietary salt can prevent primary hypertension and whether a low-salt diet is an efficacious intervention in the treatment of hypertension is still controversial. The most recent Canadian consensus statement on the role of salt in controlling hypertension was published in 1990. That document recommended moderate salt reduction in normotensive people and salt restriction in those with high blood pressure.

Over the past 20 years, more than 60 randomized controlled trials have been published studying the effects of salt intake in normotensive and hypertensive subjects. Despite the vast literature on this issue, there is still little agreement as to the efficacy, safety and acceptability of this dietary intervention.8-11

The purpose of this report is to review the current evidence concerning the effect of dietary salt intake on blood pressure. On the basis of a critical assessment of these data, recommendations for health care professionals are offered as guidelines for patient counselling and for the formulation of relevant nutritional policies.

Methods

A complete description of the methods used in developing these guidelines is given in part 1 of this supplement.12

A panel of 5 health care professionals with expertise in the area of hypertension and diet was selected by the Organizing Committee for the lifestyle modification recommendations to address the issue of interaction between salt intake and hypertension. The panel established definitions of modestly restricted salt intake, normal salt intake and excessive salt intake (Table 1).

A published well-conducted meta-analysis formed the foundation of the evidence. Each article cited in the meta-analysis was reviewed by the panel. In addition, a MEDLINE search was conducted for the period January 1966 to September 1996 using the following terms in combination: hypertension, blood pressure, vascular resistance, sodium chloride, sodium, diet, sodium or sodium chloride dietary, sodium restricted/reducing diet, clinical trials, controlled clinical trial, randomized controlled trial and random allocation. In addition, each member of the panel searched his or her own reprint files for any additional articles that might have been missed in the literature search. To ensure consistency, the inclusion criteria used were identical with those of the published meta-analysis.6

Specifically, to assess the effect of sodium restriction on blood pressure, all randomized trials that included an intervention involving dietary salt and that measured diastolic and systolic blood pressure as main outcomes were used. Abstracts and unpublished studies were excluded. Other studies that investigated the effect of salt restriction on left ventricular hypertrophy and papers dealing with the interaction of salt restriction and pharmacologic treatment were used. Review articles and papers reporting on trials dealing with the interaction effects of salt intake, drugs and weight loss were also reviewed by members of the panel. The articles were classified according to study design and subjects (hypertensive or normotensive people). The principles for grading the evidence and the recommendations were based on those previously used by the Canadian Hypertension Society and are summarized in part 1 of this supplement.12

A statistical analysis was performed to determine the effect of dietary interventions by correlating changes in urinary excretion of sodium with changes in blood pressure. The methods used to calculate this effect are described in the previously published meta-analysis.6

An attempt was made to reach consensus on all recommendations. The evidence and the recommendations were presented to the other expert panels for this guidelines series, submitted for review to major Canadian organizations and presented at an international conference on preventive cardiology, to allow for further national and international input. All revisions were reviewed and assessed by the panel before incorporation into the final document.

Results

Twenty-nine studies of normotensive subjects14-42 and 30 studies of hypertensive subjects42,43,44,45-69 were identified and reviewed. Studies of normotensive subjects tended to be short-term studies with less than 1 month of intervention. Only 3 long-term studies of normotensive subjects (lasting more than 1 year) were identified.22,42,43 The subjects in the studies of normotensive people were generally younger (mean age 26 years) than those in the studies of hypertensive people (mean age 47 years). Trials with hypertensive subjects had longer intervention periods, and 5 long-term studies were identified.43,45,46,64,69

Apart from the studies identified from the meta-analysis,6 19 additional studies were evaluated.4,5,22,40,41,42,49,57,70-80

Salt and normotensive adults

Only a few studies have investigated the feasibility of preventing hypertension by nonpharmacologic interventions.22,42,71,81 In a recent trial of more than 2000 subjects,62 restricting salt intake was much less effective than weight loss in preventing hypertension.

Recommendation

- Restriction of salt intake for normotensive people is not recommended at present because of insufficient evidence to indicate that this would lead to reduced incidence of hypertension (grade B recommendation).

| Table 1: Daily intake of sodium for moderately restricted, normal and excessive sodium diets |
|---------------------------------|---------------------------------|---------------------------------|
| Moderately restricted sodium intake | Normal sodium intake | Excessive sodium intake |
| 90–130 mmol Na⁺ | 131–175 mmol Na⁺ | > 175 mmol Na⁺ |
| 1.0–1.5 tsp NaCl | 1.6–3 tsp NaCl | > 3 tsp NaCl |
| 5.0–7.5 g NaCl | 7.6–10 g NaCl | > 10 g NaCl |
| 2.0–3.0 g Na⁺ | 3.1–6.0 g Na⁺ | > 6 g Na⁺ |
The meta-analysis\(^6\) revealed that the effect of restricting salt in free-living normotensive subjects (people who had been counselled about salt intake but who were not enrolled in tightly controlled trials) was not statistically significant. After adjustment for error in the measurement of urinary excretion of sodium, the decrease in blood pressure for normotensive subjects after a reduction in sodium intake of 100 mmol/day was 1.0 mm Hg for systolic blood pressure and 0.1 mm Hg for diastolic blood pressure.\(^7\)

Although these results suggest that salt restriction yields only modest effects in terms of reducing blood pressure, this panel recognizes that excessive intake of salt in the North American diet should be avoided. A downward shift in the entire distribution of systolic blood pressure by 1 mm Hg is likely to reduce the annual mortality rate from stroke by 3%, the mortality rate from coronary artery disease by 2% and the annual all-cause mortality rate by 1.5%.\(^7\)

**Recommendation**

- To avoid excessive intake of salt people should be counselled to choose foods low in salt (e.g., fresh fruits and vegetables), to avoid foods high in salt (e.g., pre-prepared foods), to refrain from adding salt at the table and minimize the amount of salt used in cooking, and to increase their awareness of the salt content of food choices in restaurants (grade D recommendation).

### Salt and hypertensive adults

For people with high blood pressure, salt restriction seems to have significant value in reducing blood pressure. Midgley and colleagues\(^6\) found that in trials with hypertensive subjects, the adjusted decrease in blood pressure associated with a reduction in daily sodium intake of 100 mmol was 3.7 mm Hg for systolic blood pressure and 0.9 mm Hg for diastolic blood pressure. This effect was more pronounced in people older than 44 years of age.

In a subgroup analysis using only trials in which the mean age was 44 years or older, the decrease was much greater: for a reduction in daily sodium intake of 100 mmol the reduction in systolic blood pressure was 6.3 mm Hg and the reduction in diastolic blood pressure was 2.2 mm Hg. For younger hypertensive patients, the decrease was 2.4 mm Hg for systolic blood pressure and negligible for diastolic blood pressure.\(^6\)

Although reducing salt intake may have beneficial effects on blood pressure in hypertensive patients, a cohort study of 3000 people with mild or moderate hypertension reported that those with a daily sodium consumption of less than 89 mmol had a 4-fold greater likelihood of myocardial infarction than did those with higher sodium intake.\(^6\) Thus, the safety of restricting dietary sodium to less than 89 mmol/day in hypertensive patients has not been established.

### Recommendation

- In hypertensive patients, particularly those over the age of 44 years, it is recommended that the intake of dietary sodium be moderately restricted to a target range of 90–130 mmol per day (grade B recommendation).

To determine whether salt consumption plays a role in a patient's elevated blood pressure, an estimate of daily salt consumption must be obtained. Although 24-hour urine collection yields a good estimate of daily salt consumption, accurate collection of such a specimen takes considerable effort on the part of several people, including the physician requesting the test; this makes the test less appealing to medical personnel. In addition, it has been found that repeated 24-hour urine collections are needed to obtain an accurate estimate.

In contrast, an interview assessment of diet can, with a little encouragement, be easily incorporated into a physician's interview with the patient. The mere fact that the physician wants to discuss salt consumption flags it as an important issue for the patient. Several studies have indicated the effectiveness and accuracy of self-reporting and interview assessments in determining a patient's diet. A simple questionnaire suitable for determining the probable level of salt intake is shown in Appendix 1.

### Recommendation

- It is recommended that the salt consumption of hypertensive patients be determined by interview (grade D recommendation).

Salt reduction has been suggested as a possible adjunct to pharmacologic treatment to enhance blood pressure control. Several studies have investigated this issue and found that, for hypertensive patients who are receiving antihypertensive medication, salt restriction provides additional benefits in terms of blood pressure control.

One of the larger studies of this type was conducted by Erteman and associates,\(^49\) who found that an additional 3 mm Hg decrease in diastolic blood pressure could be achieved through salt restriction among patients taking diuretics and \(\beta\)-blockers. Similar results were reported in another study involving 356 patients, in which a low-salt diet provided an additional 4 mm Hg decrease in systolic blood pressure and an additional 2 mm Hg decrease in diastolic blood pressure.\(^73\) Carney and collaborators\(^77\) reported a similar trend for patients using diuretic drugs.

Hypertensive patients receiving angiotensin-converting enzyme inhibitors also appear to benefit from salt restriction. Both MacGregor and colleagues\(^23\) and Hollenberg and coworkers\(^25\) reported that patients receiving captopril had a further decrease in blood pressure when salt intake was moderately reduced. Kristinsson and associates\(^26\) stud-
ied 27 patients and reported an additional reduction of 4 mm Hg in systolic blood pressure and 3 mm Hg in diastolic blood pressure when a low-salt diet was used in conjunction with captopril treatment.

The effect of calcium channel blockers in reducing blood pressure does not appear to be enhanced by restriction of dietary salt. In fact, most studies found that the reduction in blood pressure caused by calcium channel blockers was greater when patients were on a high-salt diet than when they were on a low-salt diet.77-79

Salt restriction appears to provide additional benefits for hypertensive patients taking medication, except those taking calcium channel blockers. The additive effect of salt restriction leads to a further decrease of 3-4 mm Hg in blood pressure.80

Although this report focuses on the relation between salt intake and blood pressure, the effects of sodium restriction on left ventricular hypertrophy should not be neglected. A positive correlation between sodium intake and changes in left ventricle geometry has been confirmed in normotensive and hypertensive subjects.84-86 Schmieder and collaborators and Liebson and coworkers87 found that urinary excretion of sodium was the strongest predictor of left ventricular mass in patients with primary hypertension. Given that hypertrophy is an important independent risk factor for future cardiovascular events, this issue requires further research.

Interpretation

The role of sodium in the etiology and pathogenesis of hypertension remains controversial. Since the 1989 Canadian Consensus Conference on Non-pharmacologic Approaches to the Management of High Blood Pressure,7 many new studies have addressed the effects of a reduction in dietary sodium on blood pressure. Our panel had at its disposal a recent meta-analysis of 56 randomized trials and assessed additional evidence. The panel ascertained that there is still no evidence that salt restriction prevents hypertension in the general population. It reconfirmed that this manoeuvre is an efficacious strategy for treating hypertensive or hypertensive patients, particularly those over 44 years of age. Thus, in these patients, counselling and assistance in embarking on a low-salt diet should be an integral part of the overall therapeutic regimen. The panel has recommended a desirable daily target range for salt intake for hypertensive people.

Future research

There is a need for further research in several areas. Studies of normotensive people 40 years of age and over are needed to further assess the influence of sodium on blood pressure. This research should help in formulating nutritional policies for the Canadian population.

Strategies must be developed for educating patients about successful implementation of a salt-restricted diet. Such strategies might include innovative approaches to counselling, development of better teaching aids and development of alternative diets.

In cooperation with the food industry, researchers must find palatable solutions to achieve a gradual reduction of the salt content in food products and to increase the availability of low-salt alternatives. In addition, research on public health policy should include identifying a consumer-friendly way to highlight salt content on food labels and formulating public education campaigns about healthy food choices.

Conclusion

The Canadian population should recognize the benefits of reducing excessive salt intake as a move toward a healthier lifestyle. Health care professionals should be aware that reducing excess salt intake is an efficacious measure in hypertensive patients, particularly those over 44 years of age. Thus, in these patients, counselling and assistance in embarking on a low-salt diet should be an integral part of the overall therapeutic regimen. The panel has recommended a desirable daily target range for salt intake for hypertensive people.

During the development of these recommendations, invaluable assistance was provided by Dr. Alexander Logan and Mr. Rodrick Chew. We express our sincere thanks. We are also grateful for the external reviews of the Canadian Council of Cardiovascular Nurses, the Canadian Nurses Association, the Canadian Pharmacists Association, the Canadian Public Health Association, the College of Family Physicians of Canada, the Heart and Stroke Foundation of Canada, Hoechst Marion Roussel and Merck Frosst Canada Inc.

The financial support of the Laboratory Centre for Disease Control at Health Canada and of Astra Pharma Inc., Bayer Inc., Bristol-Myers Squibb Pharmaceutical Group, Knoll Pharma Inc., Merck Frosst Canada Inc., Searle Canada Inc. and Servier Canada Inc. is gratefully acknowledged.

Competing interests: None declared for Dr. Fodor and Ms. Whitmore. Drs. Leenen and Larochele receive honoraria, speaker’s fees, educational grants and travel assistance from various pharmaceutical companies.

References


Dietary salt
Fodor et al.

Appendix 1: Interview to evaluate sodium consumption in hypertensive people

Statement to assess sodium consumption*

I use prepared foods such as frozen dinners, packaged or canned goods, or processed meats or cheeses

I eat salty snack foods such as potato chips, salted nuts, cheese snacks or pretzels

I eat restaurant meals

I salt my food at the table

*Possible responses are “usually” (more than once per week), “sometimes” (approximately once per week) or “rarely” (less than once per week). Use processed foods sparingly (e.g., canned vegetables; processed meats such as bologna, bacon, construed beef, ham, smoked meat and sausage; processed cheese foods; salted snack foods such as potato chips and popcorn; pickles; sauerkraut; and prepared sauces such as ketchup and soy sauce).

Reprint requests to: Heart and Stroke Foundation of Canada, 1402-222 Queen St., Ottawa ON K1P 5V9; fax 613 569-3278

Appendix 2: Guidelines for a low-sodium diet (90–130 mmol sodium)

Eat fresh foods such as fruits and vegetables as often as possible (frozen vegetables without sauces are acceptable).

Use processed foods sparingly (e.g., canned vegetables; processed meats such as bologna, bacon, cornered beef, ham, smoked meat and sausage; processed cheese food; salted snack foods such as potato chips and popcorn; pickles; sauerkraut; and prepared sauces such as ketchup and soy sauce).

Many meals are high in sodium. The type of meal chosen and the number of meals eaten outside the home will affect sodium intake. People may be able to determine the sodium content of the meal by taste; thrice a meal may be an indicator of a high-salt meal. Chinese food, pizza and battered deep-fried chicken are particularly high in sodium.
6. Recommendations on potassium, magnesium and calcium

Ellen Burgess, MD; Richard Lewanczuk, MD, PhD; Peter Bolli, MD; Arun Chockalingam, PhD; Heather Cutler, RD; Gregory Taylor, BSc, MD; Pavel Hamet, MD, PhD

Abstract

Objective: To provide updated, evidence-based recommendations on the consumption, through diet, and supplementation of the cations potassium, magnesium and calcium for the prevention and treatment of hypertension in otherwise healthy adults (except pregnant women).

Options: Dietary supplementation with cations has been suggested as an alternative or adjunctive therapy to antihypertensive medications. Other options include other nonpharmacologic treatments for hypertension.

Outcomes: The health outcomes considered were changes in blood pressure and in morbidity and mortality rates. Because of insufficient evidence, no economic outcomes were considered.

Evidence: A MEDLINE search was conducted for the period 1966–1996 with the terms hypertension and potassium, magnesium and calcium. Reports of trials, meta-analyses and review articles were obtained. Other relevant evidence was obtained from the reference lists of articles identified, from the personal files of the authors and through contacts with experts. The articles were reviewed, classified according to study design, and graded according to the level of evidence.

Values: A high value was placed on the avoidance of cardiovascular morbidity and premature death caused by untreated hypertension.

Benefits, harms and costs: The weight of the evidence from randomized controlled trials indicates that increasing intake of or supplementing the diet with potassium, magnesium or calcium is not associated with prevention of hypertension, nor is it effective in reducing high blood pressure. Potassium supplementation may be effective in reducing blood pressure in patients with hypokalemia during diuretic therapy.

Recommendations: For the prevention of hypertension, the following recommendations are made: (1) The daily dietary intake of potassium should be 60 mmol or more, because this level of intake has been associated with a reduced risk of stroke-related mortality. (2) For normotensive people obtaining on average 60 mmol of potassium daily through dietary intake, potassium supplementation is not recommended as a means of preventing an increase in blood pressure. (3) For normotensive people, magnesium supplementation is not recommended as a means of preventing an increase in blood pressure. (4) For normotensive people, calcium supplementation above the recommended daily dietary intake is not recommended as a means of preventing an increase in blood pressure. For the treatment of hypertension, the following recommendations are made: (5) Potassium supplementation above the recommended daily dietary intake of 60 mmol is not recommended as a treatment for hypertension. (6) Magnesium supplementation is not recommended as a treatment for hypertension. (7) Calcium supplementation above the recommended daily dietary intake is not recommended as a treatment for hypertension.

Validation: These guidelines are consistent with the results of meta-analyses and recommendations made by other organizations. They have not been clinically tested.

Sponsors: The Canadian Hypertension Society, the Canadian Coalition for High Blood Pressure Prevention and Control, the Laboratory Centre for Disease Control at Health Canada, and the Heart and Stroke Foundation of Canada.

In 1931 recommendations for hypertensive patients regarding dietary intake of and supplementation with potassium, calcium and sodium were published in CMAJ. Since then, the methodology for conducting and reporting clinical and other research, for reviewing the literature and for formulating recommendations has changed considerably. Over the years, there has been a belief that a diet high in sodium or low in potassium, magnesium or calcium predisposes people to high blood pressure and that correcting such diets would bring blood pressure to normal levels. An extreme diet of rice and fruit has been used as an in-hospital or urgent treatment for hypertension because it makes these dietary corrections. Although

Special supplement

Dr. Burgess (chair) is with the Division of Nephrology, Faculty of Medicine, University of Calgary, Calgary, Alta.; Dr. Lewanczuk is with the Faculty of Medicine, University of Alberta, Edmonton, Alta.; Dr. Bolli is with the Faculty of Medicine, University of Manitoba, Winnipeg, Man.; Dr. Chockalingam is with the Adult Health Division, Health Canada, Ottawa, Ont.; Ms. Cutler is with the Department of Clinical Nutrition, London Health Sciences Centre, London, Ont.; Dr. Taylor is with the Bureau of Cardio-Respiratory Diseases and Diabetes, Laboratory Centre for Disease Control, Health Canada, Ottawa, Ont.; and Dr. Hamet is with the Faculté de médecine, Université de Montréal, Montréal, Que.

This article has been peer reviewed.
such a diet reduces both blood pressure and body weight, it is not practical as a long-term therapy. However, patients were often counselled to make these dietary adjustments.

The primary objective of this guideline is to review contemporary clinical research on the relation between cations (potassium, magnesium and calcium) and blood pressure and to advise health care professionals and the public accordingly about the prevention and treatment of hypertension in otherwise healthy adults (except pregnant women) in the ambulatory care setting.

Methods

A complete description of the methods used in developing these recommendations is given in part 1 of this supplement. The chair and members of the panel were selected by the Organizing Committee for the lifestyle modification recommendations to obtain a spectrum of health care professionals and scientists with expertise and interest in the dietary aspects of hypertension prevention and control.

A MEDLINE search of the English and French literature was performed for the period 1966–1996 with the term hypertension and the terms potassium, magnesium and calcium used in sequential searches. Secondary searches were done using the references found in review articles and meta-analyses. Additional articles were identified by reviewing the reference lists of the identified articles, were found in the personal files of the panel members and were suggested by other experts. The principles for grading the evidence and the recommendations were based on those previously used by the Canadian Hypertension Society and are summarized in part 1 of this supplement.

An attempt was made to reach consensus on all recommendations. The evidence and the recommendations were presented for comment to the other expert panels for this guidelines series, submitted for review to major Canadian organizations and presented at an international conference on preventive cardiology, to allow further national and international input. All revisions were reviewed and assessed by the panel before incorporation into the final document.

Results

Potassium supplementation

Epidemiologic studies relating dietary intake of potassium and blood pressure were reviewed to provide a basis for understanding the intervention studies, even though such epidemiologic studies should not influence the drafting of recommendations. In those studies, dietary intake of potassium was estimated using different methods, including dietary recall or diet records, food frequency questionnaires and urine collection. Urine collection was done as 24-hour collections, overnight collections or spot collections; the results were expressed as total potassium content or as a ratio of total potassium content to urine creatinine content or urine sodium content.

Khaw and Barrett-Connor reported the results of a 12-year prospective cohort from the Rancho Bernardo project and demonstrated that the relative risk of stroke-associated death in the lowest tertile of potassium intake, compared with the top 2 tertiles combined, was 2.6 (difference not significant) for men and 4.8 (p = 0.01) for women. A 10-mmol increase in daily potassium intake was associated with a 40% reduction in risk of death from stroke (p < 0.001). This effect was apparently independent of other dietary variables and known cardiovascular risk factors. Some cross-sectional studies of other populations, including normotensive and hypertensive people, have demonstrated an association between estimated dietary potassium intake and blood pressure, although other studies have not (Table 1). Although the results of some epidemiologic studies are consistent with a link between diets high in potassium and the prevention of hypertension or hypertension-associated death, or both, these data cannot form the basis of recommendations when the results of intervention trials are available.

In evaluating intervention studies of potassium supplementation, we found no trials that examined the effect of supplementation on morbidity and mortality rates. There have been several level II intervention trials assessing the effect of increased potassium intake on blood pressure in hypertensive subjects; most of these used supplements rather than increasing dietary intake. In the pre-eminent trial of potassium supplementation, Grimm and associates conducted a randomized controlled trial in which 287 hypertensive men were randomly assigned to receive 96 mmol of potassium chloride per day or placebo for 2.2 years. Because antihypertensive medication had to be reinstated (the primary outcome variable) for 79 men in each group, long-term potassium supplementation appeared to be ineffective for treating hypertension. Other large trials have shown no reduction in blood pressure, even with doses as high as 96 mmol of potassium chloride per day; the results of smaller trials have been mixed (Table 2). Therefore, it appears that potassium supplementation is not effective in reducing blood pressure.

Studies with more than one change in diet or supplementation have been attempted; the design of such studies makes it difficult, if not impossible, to assess the role of potassium supplementation. Chalmers and colleagues conducted a trial in which 212 untreated hypertensive patients were randomly assigned to 1 of 4 diet interventions: a control diet, a high-potassium diet (urinary potassium excretion of 97 mmol/day and urinary sodium excretion similar to that of subjects on the control diet), a low-sodium diet (urinary sodium excretion 86 mmol/day) and a low-sodium/high-potassium diet (urinary sodium excretion approximately 73 mmol/day and urinary potassium excretion 87 mmol/day). The reduction in blood pressure with either the low-sodium diet or the high-potassium diet was significant relative to the control diet. However, the combination diet did not result in any greater reduction in blood pressure than a single dietary change. Another study, conducted over 6 months, used potassium supplementation in combination with calcium or magnesium supplementation. There was no statistically
significant effect of either treatment relative to placebo. In a separate trial of 100 patients with mild to moderate hypertension, multiple changes in mineral intake (obtained by administering a low-sodium, high-potassium and high-magnesium salt substitute) were made over 24 weeks and resulted in only a modest reduction in systolic blood pressure. In the short-term DASH (Dietary Approaches to Stop Hypertension) trial participants were randomly assigned for 6 months to a standard diet, a diet enhanced with fruits and vegetables, or a diet enhanced with fruits, vegetables and low-fat dairy products. Overall, for normotensive and hypertensive participants, the only significant reduction was in systolic blood pressure (level II evidence). For the 133 hypertensive subjects, the fruit-and-vegetable diet led to a change of $-7.2/-2.8$ mm Hg more than the control diet, and the combination diet led to a change of $-11.4/-5.5$ mm Hg more than the control diet (level III evidence). In the normotensive subjects, only the combination diet reduced blood pressure more than the control diet (level III evidence). Because of the multiple dietary changes, the confounding changes in other nutrients as the diets were changed and the inclusion of both normotensive and hypertensive subjects, it is difficult to understand the results of this short-term study.

There may be some distinct patient populations for whom potassium supplementation is useful, such as patients with diuretic-induced hypokalemia and patients of African descent. In a study by Kaplan and collaborators, patients with hypokalemia from treatment with diuretic drugs experienced significant reductions in blood pressure when given 60 mmol of potassium daily for 6 weeks. However, in a substudy of the large hypertension studies conducted by the Medical Research Council in the United Kingdom, patients randomly assigned to receive diuretic therapy underwent a secondary randomization to compare two diuretics and to assess the utility of potassium supplementation. In this setting, the use of potassium supplements over 3 years was not associated with any additional reduction of blood pressure.

There has been much discussion in the literature suggesting that black hypertensive patients may benefit from potassium supplementation. Obel reported significant reductions in blood pressure ($-39/-17$ mm Hg) in a placebo-controlled study conducted in Africa in which 48 patients were ran-

### Table 1: Epidemiology studies of the relation between dietary potassium (K) and blood pressure or hypertension

<table>
<thead>
<tr>
<th>Method of assessing dietary K</th>
<th>Subjects</th>
<th>Results</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet recall with dietitian$^a$</td>
<td>859 men and women</td>
<td>10 mmol increase in dietary K associated with a stroke mortality RR of 0.56–0.65</td>
<td>12-yr prospective study; BP measured</td>
</tr>
<tr>
<td>Overnight urine K/Cr ratio$^a$</td>
<td>574 normotensive and hypertensive</td>
<td>$r = -0.23$, $p &lt; 0.001$</td>
<td>BP measured</td>
</tr>
<tr>
<td>24-hr urine collection$^a$</td>
<td>662</td>
<td>Urinary K NS</td>
<td>Followed 1–4 yr; BP measured</td>
</tr>
<tr>
<td>Duplicate diet, 24-hr urine collection$^a$</td>
<td>Blacks: 148 men, 208 women Whites: 342 men, 328</td>
<td>NS when other variables controlled</td>
<td>BP measured</td>
</tr>
<tr>
<td>Total body content, exchangeable serum K level$^a$</td>
<td>91 hypertensive, 121 normotensive</td>
<td>Serum K, total body K, exchangeable K related to SBP and DBP</td>
<td>Diet was not assessed, patients drug-free; BP measured</td>
</tr>
<tr>
<td>Urine K/Cr ratio$^a$</td>
<td>98 vegetarian, 98 nonvegetarian</td>
<td>Urinary K &gt; 80 had lower BP</td>
<td>2% of vegetarians and 26% of nonvegetarians had hypertension; BP measured</td>
</tr>
<tr>
<td>Diet recall with dietitian$^a$</td>
<td>309 men, 376 women</td>
<td>Diet K related to SBP</td>
<td>SBP was age-adjusted; BP measured</td>
</tr>
<tr>
<td>Overnight urine Na/K ratio$^a$</td>
<td>919</td>
<td>Urinary Na/K related to DBP, $r = 0.15$, $p &lt; 0.05$</td>
<td>Recruited in stroke cities; BP measured</td>
</tr>
<tr>
<td>Morning spot urine for K and Cr$^a$</td>
<td>120</td>
<td>Urinary K/Cr related to SBP, $r = -0.10$, $p &lt; 0.05$</td>
<td>Traditional Japanese lifestyle; BP measured</td>
</tr>
<tr>
<td>Diet recall$^a$</td>
<td>667</td>
<td>Urinary Na/K and BP higher in blacks</td>
<td>NHANES-1 subset with no hypertension; BP measured</td>
</tr>
<tr>
<td>Diet recall$^a$</td>
<td>8000</td>
<td>Inconclusive</td>
<td>Inter correlation between cations prevented assessment individually; BP measured</td>
</tr>
<tr>
<td>24-hr urine collection$^a$</td>
<td>3754 men, 3600 women</td>
<td>$r = -0.04$ to 0.06</td>
<td>Very weak correlations; BP measured</td>
</tr>
<tr>
<td>24-hr urine collection$^a$</td>
<td>1079</td>
<td>Dietary K related to BP (pooled adjusted), $r = -0.0446$</td>
<td>In only 3 centres; BP measured</td>
</tr>
<tr>
<td>Diet recall with dietitian$^a$</td>
<td>584 men, 718 women</td>
<td>Dietary Na/K ratio related to SBP and DBP</td>
<td>In both men and women; BP measured</td>
</tr>
<tr>
<td>FFQ$^a$</td>
<td>58 218 women</td>
<td>No significant relation</td>
<td>4-yr follow-up; BP self-reported</td>
</tr>
<tr>
<td>FFQ$^a$</td>
<td>30 681 men</td>
<td>No significant relation</td>
<td>BP self-reported</td>
</tr>
<tr>
<td>24-hr urine collection$^a$</td>
<td>201 men</td>
<td>Urinary K/Cr related to SBP, $r = -0.294$</td>
<td>BP measured</td>
</tr>
<tr>
<td>FFQ$^a$</td>
<td>41 541 women</td>
<td>No significant relation</td>
<td>BP self-reported</td>
</tr>
</tbody>
</table>

Note: RR = relative risk, Cr = creatinine, Na = sodium, NS = nonsignificant, BP = blood pressure, SBP = systolic blood pressure, DBP = diastolic blood pressure, NHANES = National Health and Nutrition Examination Surveys database, FFQ = food frequency questionnaire.
domly assigned to receive 64 mmol of potassium daily for 16 weeks. Brancati and coworkers\textsuperscript{50} conducted a study of black Americans with normal blood pressure, who were given a potassium-poor diet for 3 weeks and then randomly assigned to receive 80 mmol of potassium (as a supplement) or placebo for 3 weeks. In this contrived setting, potassium supplementation was associated with a reduction in blood pressure, but it is unclear how this study relates to clinical practice and hence it has not been included in our analysis of the evidence. Supporting studies are required before specific recommendations can be made for this distinct population.

Two meta-analyses\textsuperscript{51,52} both suggested a small beneficial effect that may be related to the dietary intake of sodium. The more recent meta-analysis,\textsuperscript{52} which covered treatment studies of potassium supplementation, suggested that supplementation be considered for patients with high sodium intake but stopped short of recommending supplementation for prevention and treatment of hypertension in everyone. This meta-analysis did not include the level II study by Grimm and associates\textsuperscript{22} on the basis that sodium restriction was part of the intervention in that trial; however, 3 other studies that used combined sodium restriction and

<table>
<thead>
<tr>
<th>Study design</th>
<th>Subjects</th>
<th>Daily K dose, mmol</th>
<th>Duration</th>
<th>Dietary changes, mmol</th>
<th>Change in BP, mm Hg*</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level II</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{22}</td>
<td>287 men</td>
<td>96</td>
<td>2.2 yr</td>
<td>–18/8 h</td>
<td>+24/8 h</td>
<td>Not given</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{23}</td>
<td>1185</td>
<td>16.8–33.6</td>
<td>35 mo</td>
<td>No significant reduction</td>
<td>–8.66–7.91 NS</td>
<td>79 men per group restarted drugs</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{24}</td>
<td>258 of 787 Diet 103</td>
<td>6 mo</td>
<td>–35</td>
<td>+13</td>
<td>+0.2–0.6 NS</td>
<td>Dose of K low; diuretic treatment</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{48}</td>
<td>298</td>
<td>64</td>
<td>12 wk</td>
<td>+1</td>
<td>+39</td>
<td>NS</td>
</tr>
<tr>
<td>Single blind\textsuperscript{47}</td>
<td>47</td>
<td>&gt; 30</td>
<td>1 yr</td>
<td>+45% (25 mmol)</td>
<td>NS</td>
<td>Subgroup of TAIM</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{28}</td>
<td>37</td>
<td>60</td>
<td>32 wk</td>
<td>–12</td>
<td>+20</td>
<td>–12.1–13.1</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{49}</td>
<td>48</td>
<td>64</td>
<td>112 d</td>
<td>+40</td>
<td>–39–17</td>
<td>Inadequate power</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{25}</td>
<td>37</td>
<td>48</td>
<td>105 d</td>
<td>–6</td>
<td>+30</td>
<td>–14–10.5</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{26}</td>
<td>101</td>
<td>120</td>
<td>8 wk</td>
<td>+35</td>
<td>+62</td>
<td>–71–3 NS</td>
</tr>
<tr>
<td>Single-blind crossover\textsuperscript{31}</td>
<td>32</td>
<td>65</td>
<td>6 wk</td>
<td>+12</td>
<td>+57</td>
<td>–2.5–0.6 NS</td>
</tr>
<tr>
<td>Double-blind crossover\textsuperscript{32}</td>
<td>40</td>
<td>72</td>
<td>6 wk</td>
<td>+12</td>
<td>+57</td>
<td>–2.5–0.6 NS</td>
</tr>
<tr>
<td>Double-blind crossover\textsuperscript{33}</td>
<td>16</td>
<td>60</td>
<td>6 wk</td>
<td>+1</td>
<td>+46</td>
<td>–5.6–5.8</td>
</tr>
<tr>
<td>Double-blind crossover\textsuperscript{34}</td>
<td>12</td>
<td>140</td>
<td>2–6 wk</td>
<td>+5</td>
<td>+123</td>
<td>–1.9–1.0 NS</td>
</tr>
<tr>
<td>Double-blind crossover\textsuperscript{35}</td>
<td>20</td>
<td>64</td>
<td>4 wk</td>
<td>+7</td>
<td>+50</td>
<td>–2.00 NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{36}</td>
<td>24</td>
<td>64</td>
<td>4 wk</td>
<td>+29</td>
<td>+56</td>
<td>–7.0–4.0</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{37}</td>
<td>18</td>
<td>60</td>
<td>4 wk</td>
<td>+13</td>
<td>+39</td>
<td>–10–6</td>
</tr>
<tr>
<td>Double-blind crossover\textsuperscript{38}</td>
<td>20</td>
<td>64</td>
<td>2 wk</td>
<td>+9</td>
<td>+52</td>
<td>–1.1–2.5</td>
</tr>
<tr>
<td>Placebo-controlled, double-blind crossover\textsuperscript{39}</td>
<td>12</td>
<td>120</td>
<td>8 d</td>
<td>–13</td>
<td>+104.9</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind, randomized\textsuperscript{40}</td>
<td>22</td>
<td>70</td>
<td>4 d</td>
<td>–29</td>
<td>+109</td>
<td>–8.6–4.0</td>
</tr>
<tr>
<td>Level IV</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Open crossover\textsuperscript{41}</td>
<td>20</td>
<td>100</td>
<td>10 d</td>
<td>+25</td>
<td>+82</td>
<td>–11.1–5.2</td>
</tr>
<tr>
<td>Open, single-blind\textsuperscript{42}</td>
<td>16</td>
<td>100</td>
<td>8 wk</td>
<td>+6</td>
<td>+87</td>
<td>–17–10 NS</td>
</tr>
<tr>
<td>Level V</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Open, single-blind\textsuperscript{43}</td>
<td>10</td>
<td>96</td>
<td>12 d</td>
<td>–6</td>
<td>+63</td>
<td>–9–2 NS</td>
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<tr>
<td>Combination therapies</td>
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</tr>
<tr>
<td>Level II</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RCT\textsuperscript{44}</td>
<td>212</td>
<td>Diet &gt; 100</td>
<td>12 wk</td>
<td>+22</td>
<td>–3.9–3.1</td>
<td>No additional reduction with low Na diet</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{45}</td>
<td>95</td>
<td>60</td>
<td>6 mo</td>
<td>NS</td>
<td>NS</td>
<td>Combined with Mg and/or Ca supplements</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT\textsuperscript{46}</td>
<td>100</td>
<td>Ad lib use of salt substitute with K and Mg</td>
<td>6 mo</td>
<td>–32</td>
<td>+22</td>
<td>–7.6–3.3</td>
</tr>
<tr>
<td>RCT\textsuperscript{47}</td>
<td>459</td>
<td>Diet</td>
<td>8 wk</td>
<td>–9</td>
<td>+32</td>
<td>–2.8–0.3</td>
</tr>
</tbody>
</table>

Note: RCT = randomized controlled trial, TAIM = Trial of Antihypertensive Interventions and Management.

*Statistically significant unless otherwise specified.
potassium supplementation were included in the meta-analysis, including that of Chalmers and colleagues,\textsuperscript{46} which demonstrated that a combination of low sodium and high potassium did not reduce blood pressure any more than either intervention alone. The analysis of confounding variables revealed that high urinary sodium excretion, higher pretreatment diastolic blood pressure and small sample size were directly related to treatment effect; it also revealed that race and study duration may be important factors.

Prevention of an increase in blood pressure or the development of hypertension through potassium supplementation has been the focus of 2 large prevention studies.\textsuperscript{53,54} The Hypertension Prevention Trial\textsuperscript{53} randomly assigned 841 men and women to 1 of 4 diets: a low-calorie diet, a low-sodium diet, a combination of these 2 diets, or a low-sodium/high-potassium diet. Patients were assessed at 6 months and again at 3 years. Counselling of the patients assigned to the low-sodium/high-potassium diet resulted in a decrease in sodium intake by approximately 36 mmol/day but no change in potassium intake (as judged by urinary excretion) after 6 months; hence, there was an increase in the urinary sodium-to-potassium ratio. After 3 years, there was no greater reduction in blood pressure in the low-sodium/high-potassium group than in the low-sodium group. The relative importance of the negative result of this trial is unclear; because there was no evidence of an increase in dietary potassium intake over the long term, it is not possible to truly assess the long-term effect of a high-potassium diet on blood pressure. However, these results suggest that people are not able to maintain these dietary changes, which would limit the effectiveness of prescribing high-potassium diets over the long term. The Trial of Hypertension Prevention\textsuperscript{14} was a large, short-term, level II trial of 318 patients that demonstrated no effect on blood pressure of a daily intake of 60 mmol of potassium chloride over 6 months. In addition, 4 nonrandomized trials\textsuperscript{57-60} have shown inconsistent results, and 2 randomized trials\textsuperscript{53,54} of normotensive patients demonstrated no reduction in blood pressure (Table 3). In the DASH trial\textsuperscript{49} the diet enhanced with fruits and vegetables did not affect blood pressure in the subgroup of normotensive participants (level III evidence). Despite the suggestive epidemiologic data, intervention trials have failed to demonstrate that potassium supplementation prevents an increase in blood pressure or the development of hypertension.

In these treatment and prevention trials, potassium supplementation was given in addition to a dietary intake that averaged approximately 60 mmol of potassium per day and was shown to be ineffective. This baseline amount of dietary potassium can be obtained by following Canada’s Food Guide to Healthy Eating,\textsuperscript{42} with a focus on fruits and vegetables. Evidence does not support potassium supplementation for normotensive people to prevent an increase in blood pressure, nor for hypertensive patients to reduce blood pressure.

**Recommendations**

**Prevention**

- The daily dietary intake of potassium should be 60 mmol or more, because this level of intake has been associated with a reduced risk of stroke-related death (grade D recommendation).
- For normotensive people obtaining on average 60 mmol of potassium daily through dietary intake, potassium supplementation is not recommended as a means of preventing an increase in blood pressure (grade B recommendation).

**Treatment**

- Potassium supplementation above the recommended daily dietary intake of 60 mmol is not recommended as a treatment for hypertension (grade B recommendation).

| Table 3: Prevention studies of potassium supplementation in normotensive subjects |
|---------------------------------|---------|----------------|-----------------|-----------------|-----------------|----------------|
| Study design                     | Subjects | Daily K dose, mmol | Duration | Dietary changes | Change in BP, mm Hg | Comments |
| Level II                         |         |                    |         |                 |                 |            |
| Randomized\textsuperscript{1}    | 195     | Diet > 100         | 3 yr    | −36             | 0                | No significant reduction in urinary K after 6 mo |
| Double-blind placebo-controlled RCT\textsuperscript{4} | 318     | 60                 | 6 mo    | −9              | +95              | Urinary Na 150 mmol/d |
| Double-blind placebo-controlled RCT\textsuperscript{5} | 44      | 80                 | 28 d    | NS              | NS               | Crossover design |
| Double-blind placebo-controlled RCT\textsuperscript{5} | 24      | 75                 | 14 d    | NS              | NS               | Crossover design |
| Level III                        |         |                    |         |                 |                 |            |
| Randomized\textsuperscript{9}    | 326 of 459 | Diet     | 8 wk    | +9              | +95              | −4.4/−0.1 NS |
| Single-blind open\textsuperscript{2} | 20      | 96                 | 7 d     | −3              | +23              | +0.4/+0.8 NS |
| Single-blind open\textsuperscript{2} | 64      | 66                 | 4 wk    | 0               | +97              | −4.2/−4.6* |
| Single-blind crossover\textsuperscript{2} | 23      | 100               | 2 wk    | −55             | +44              | −1.7/−4.5† |
| Level IV                         |         |                    |         |                 |                 |            |
| Open crossover\textsuperscript{9} | 20      | 120                | 2 wk    | −55             | +44              | −1.7/−4.5† |

\textsuperscript{a}Significant reduction of BP.

\textsuperscript{†}Significant only for reduction of DBP.
Magnesium supplementation

The relation between dietary magnesium and blood pressure has been evaluated in epidemiologic studies (Table 4). Dietary magnesium intake can be estimated using food diaries, food records or food frequency questionnaires. Urinary excretion is not a reliable indicator of ingestion because only a portion of ingested magnesium is absorbed and that amount may not be proportionately excreted through the kidneys.

The Nurses’ Health Study18,21 is a prospective cohort study of major diseases in a large group of nurses (mostly white) in the United States. The dietary intake of several nutrients was assessed using a food frequency questionnaire. Blood pressure was self-reported. The initial 4-year follow-up report18 stated that a diet high in magnesium was associated with a reduced risk of hypertension (relative risk 0.77). The food frequency questionnaire was “refined” for the second 4-year stage, and no protective effect was seen.21 The findings of the second stage were consistent with the conclusions of the Physicians’ Health Study,19 in which only dietary fibre intake was associated with a reduction in the relative risk of hypertension. A cross-sectional study from Belgium62 demonstrated a correlation between magnesium intake and measured blood pressure in women, but not in men. Joffres and colleagues63 assessed dietary nutrients and measured blood pressure in 615 men in the Honolulu Heart Study. They found a high intercorrelation among many nutrients and blood pressure but could not separate the effects of magnesium from the effects of other nutrients. Therefore, high dietary magnesium intake does not appear to be associated with prevention of hypertension.

Magnesium supplementation for hypertensive people has been tested in intervention trials, but there is no clear evidence of benefit (Table 5).64–75 Ten level II studies, in which the daily intake of magnesium was 12.5–40 mmol for up to 6 months, showed no effect, but one study64 demonstrated a modest reduction (by 3.4 mm Hg) in diastolic blood pressure. Sacks and associates47 gave magnesium in combination with potassium and calcium to 96 patients, but saw no significant effect at 6 months. In the DASH trial49 there was an increase in urinary excretion of magnesium in participants on the combination diet (low-fat dairy products and fruit and vegetables) consistent with an increase in

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**Table 4: Epidemiology studies of the relation between dietary magnesium (Mg) and blood pressure or hypertension**

<table>
<thead>
<tr>
<th>Method of assessing dietary Mg</th>
<th>Subjects</th>
<th>Results</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Questionnaire and interview62</td>
<td>8 058</td>
<td>Correlation in women only</td>
<td>Cross-sectional study; BP measured</td>
</tr>
<tr>
<td>FFQ18</td>
<td>58 218 women</td>
<td>Intake &lt; 200 mg/d associated with RR of 0.77 for hypertension over 4 yr</td>
<td>Inverse relation between dietary Mg and BP in normotensive subjects only; findings refuted in follow-up study; BP self-reported</td>
</tr>
<tr>
<td>FFQ19</td>
<td>30 681 men</td>
<td>No RR with low Mg diet</td>
<td>Inverse relation between dietary Mg and BP in normotensive subjects only; BP self-reported</td>
</tr>
<tr>
<td>FFQ20</td>
<td>41 541 women</td>
<td>No RR with low Mg diet</td>
<td>Inverse relation between dietary Mg and BP in normotensive subjects only; BP self-reported</td>
</tr>
</tbody>
</table>

**Table 5: Treatment studies of magnesium supplementation in hypertensive subjects**

<table>
<thead>
<tr>
<th>Study design</th>
<th>Subjects</th>
<th>Daily Mg supplement, mmol</th>
<th>Effect on BP, mm Hg</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level II</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>91 women</td>
<td>15</td>
<td>6 mo</td>
<td>–2.7</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>96</td>
<td>24</td>
<td>1 mo</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT64</td>
<td>41</td>
<td>12.5</td>
<td>6 mo</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>25</td>
<td>10</td>
<td>8 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>13</td>
<td>40</td>
<td>3 mo</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>37</td>
<td>20</td>
<td>2 mo</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>71</td>
<td>15</td>
<td>6 mo</td>
<td>NS</td>
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<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>14</td>
<td>15</td>
<td>6 mo</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT64</td>
<td>17</td>
<td>15–40</td>
<td>9 wk</td>
<td>–7.9</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>39</td>
<td>15</td>
<td>2 mo</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT64</td>
<td>21</td>
<td>15.8</td>
<td>3 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Level III</td>
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</tr>
<tr>
<td>Nonrandomized</td>
<td>21</td>
<td>15</td>
<td>1 mo</td>
<td>Yes</td>
</tr>
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<td>Level V</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Open65</td>
<td>20</td>
<td>15</td>
<td>6 mo</td>
<td>–12</td>
</tr>
</tbody>
</table>

*Statistically significant.
dietary intake of this nutrient. It is not clear if the effect of the combination diet in reducing blood pressure was related to increased magnesium intake for either the hypertensive or normotensive participants.

In the Trial of Hypertension Prevention, 430 patients were randomly assigned to receive placebo or 15 mmol magnesium per day for 6 months. No beneficial effect was demonstrated.

In summary, the epidemiologic studies have not reliably or consistently shown a relation between magnesium intake and blood pressure or prevention of hypertension. No beneficial effect of supplementation with magnesium has been demonstrated for either the treatment or the prevention of hypertension.

**Recommendations**

**Prevention**
- For normotensive people, magnesium supplementation is not recommended as a means of preventing an increase in blood pressure (grade B recommendation).

**Treatment**
- Magnesium supplementation is not recommended as a treatment for hypertension (grade B recommendation).

**Calcium supplementation**

Epidemiologic studies have examined the relation between dietary calcium and blood pressure or hypertension. Dietary intake of calcium is estimated in the same way as intake of magnesium, because gastrointestinal absorption of calcium is also incomplete. Reports describing an association between dietary calcium intake and blood pressure have delivered inconsistent messages (Table 6). The initial follow-up report from the Nurses’ Health Study demonstrated that a diet high in calcium was associated with a reduced risk of hypertension over 4 years. However, this conclusion was refuted in the second follow-up report. This inconsistency is thought to be related to the refinement of the food frequency questionnaire, which affected estimated dietary intake of calcium, as well as that of magnesium. Reports based on the National Health and Nutrition Examination Surveys (NHANES-1) database have also brought forth discrepancies in analyses and conclusions. McCarron and collaborators reported an inverse correlation between dietary calcium intake and blood pressure, but other analyses yielded different conclusions. A report by Sempo and coworkers using the NHANES-1 and NHANES-2 databases demonstrated a lack of association between dietary calcium and blood pressure. Some authors have found correlations only within subgroups of their main study populations, which weakens the support for an association. One meta-analysis reported a weak inverse relation between dietary calcium intake and blood pressure. Hamet under-

### Table 6: Epidemiologic studies of the relation between dietary calcium (Ca) and blood pressure or hypertension

<table>
<thead>
<tr>
<th>Method of assessing dietary Ca</th>
<th>Subjects</th>
<th>Effect on BP</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>FFQa</td>
<td>58 218 women</td>
<td>RR 0.78 for higher Ca intake*</td>
<td>Initial 4-yr follow-up of Nurses Health Study; refuted on follow-up study; BP self-reported</td>
</tr>
<tr>
<td>FFQb</td>
<td>30 681 men</td>
<td>NS</td>
<td>Lowered risk only in lean men; BP self-reported</td>
</tr>
<tr>
<td>FFQc</td>
<td>41 541 women</td>
<td>NS</td>
<td>4-yr follow-up of nonhypertensive subjects; BP self-reported</td>
</tr>
<tr>
<td>Diet recall</td>
<td>10 372</td>
<td>Risk higher if Ca intake low</td>
<td>High Na diet associated with low BP; BP measured</td>
</tr>
<tr>
<td>Diet recall</td>
<td>10 361</td>
<td>NS</td>
<td>4-yr follow-up NHANES-1; BP measured</td>
</tr>
<tr>
<td>Diet recall</td>
<td>5 840 men, 5 490 women</td>
<td>NS</td>
<td>NHANES-1 and 2; BP measured</td>
</tr>
<tr>
<td>Diet recall</td>
<td>8 000</td>
<td>Inconclusive</td>
<td>Intercorrelation between cations limited analysis; BP measured</td>
</tr>
<tr>
<td>Diet recall</td>
<td>615 men</td>
<td>NS</td>
<td>BP measured</td>
</tr>
<tr>
<td>7-day diet record</td>
<td>387 men</td>
<td>NS</td>
<td>BP measured</td>
</tr>
<tr>
<td>Diet record, dietitian</td>
<td>210 men</td>
<td>NS</td>
<td>BP measured</td>
</tr>
<tr>
<td>Diet record, blood levels</td>
<td>4 167 men, 3 891 women</td>
<td>Serum Ca related to SBP and DBP*</td>
<td>BP measured</td>
</tr>
<tr>
<td>Diet recall, dietitian</td>
<td>7 011 men</td>
<td>NS</td>
<td>Related in group with low alcohol use only; BP measured</td>
</tr>
<tr>
<td>24-h urine collection</td>
<td>10,477</td>
<td>Inverse relation</td>
<td>BP measured</td>
</tr>
<tr>
<td>Diet recall</td>
<td>7 073</td>
<td>NS, except in subgroup</td>
<td>NHANES 5–12-yr follow-up; BP measured</td>
</tr>
<tr>
<td>FFQd</td>
<td>6 517 non-black women, &gt; 64 yr</td>
<td>SBP*</td>
<td>1 g Ca = –1.5/+0.5 effect on BP; BP measured</td>
</tr>
<tr>
<td>FFQe</td>
<td>5 049</td>
<td>Variable effect depending on diet source of Ca</td>
<td>BP measured</td>
</tr>
<tr>
<td>Diet record</td>
<td>182 normotensives</td>
<td>r = –0.2*</td>
<td>BP measured</td>
</tr>
<tr>
<td>Diet recall, dietitian</td>
<td>167 women</td>
<td>NS</td>
<td>10-yr follow-up; BP measured</td>
</tr>
</tbody>
</table>

*Statistically significant.
took an extensive review of the issue but was unable to definitively support a link between calcium intake and hypertension.

In examining the potential effect of calcium supplementation on blood pressure, we reviewed 11 randomized trials and 5 non-randomized trials,47,91-103 (Table 7). In most studies, there were no significant changes in blood pressure. McCarron and Morris47 reported a trial that included 48 hypertensive patients randomly assigned to receive placebo or 1 g of calcium daily for 3 weeks. The effects on blood pressure were inconsistent, and the trial was judged to be a “negative” study. Sacks and associates47 reported no significant effect on blood pressure for 94 patients randomly assigned to receive placebo or combination therapy with either calcium and magnesium or calcium and potassium for 6 months. There have been suggestions that certain patient groups may benefit from calcium supplementation, but there is no trial evidence to support such suggestions. Two meta-analyses of randomized trials106,107 reported a modest reduction in blood pressure with calcium supplementation, but neither recommended calcium supplementation. Hamet90 reached a similar conclusion. The recent DASH trial47 reported that blood pressure declined to a significantly greater extent in the hypertensive subjects who were on a diet that included low-fat dairy products and extra fruit and vegetables than in those who were on either the control diet or the diet high in fruits and vegetables (level III evidence). Presumably this was because of the increase in calcium intake from the dairy products, although there was no significant change in urinary calcium excretion.

Calcium supplementation has not been shown to prevent an increase in blood pressure or hypertension. In the Trial of Hypertension Prevention, 445 normotensive patients were randomly assigned to receive either placebo or 1 g of calcium daily for 6 months. There was no difference in blood pressure between the groups. Three other small studies95,99,100 included normotensive subjects (in addition to hypertensive patients) and failed to demonstrate any reduction in blood pressure with calcium supplementation. In the DASH study,49 however, the normotensive subgroup experienced a decrease in blood pressure when eating the diet that included low-fat dairy products and extra fruit and vegetables (level III evidence). Presumably, this effect was related to the increase in dietary calcium, although again there was no change in urinary calcium excretion.

The recommended daily intake for calcium can be obtained by following Canada’s Food Guide to Healthy Eating,10 which recommends 2 to 4 servings of milk or milk products daily. Low-fat dairy products, as used in the DASH study, are preferred, to limit an increase in dietary fat. The evidence does not support the use of calcium supplementation as a means of preventing an increase in blood pressure in normotensive people or as a treatment for hypertension.

**Recommendations**

**Prevention**

- For normotensive people, calcium supplementation above the recommended daily dietary intake is not recommended as a means of preventing an increase in blood pressure (grade B recommendation).

**Treatment**

- Calcium supplementation above the recommended daily dietary intake is not recommended as a treatment for hypertension (grade B recommendation).

**Table 7: Treatment studies of calcium supplementation in hypertensive subjects**

<table>
<thead>
<tr>
<th>Study design</th>
<th>Subjects</th>
<th>Daily Ca dose</th>
<th>Duration</th>
<th>Effect on BP, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Level II</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>48 hypertensive, 32 normotensive</td>
<td>1 g</td>
<td>3 wk</td>
<td>Standing SBP –5.6*, supine SBP –3.8; standing DBP –2.3</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT47</td>
<td>90</td>
<td>1 g</td>
<td>12 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>47 hypertensive, 48 normotensive</td>
<td>10 mmol, 20 mmol</td>
<td>8 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>23</td>
<td>1 g</td>
<td>8 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>18</td>
<td>40 mmol, 1 mmol</td>
<td>3 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>8</td>
<td>1 g</td>
<td>3 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>17 hypertensive, 29 normotensive</td>
<td>1.5 g</td>
<td>8 wk</td>
<td>NS (overall)</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>26</td>
<td>800 mg, 400 mg</td>
<td>8 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>15</td>
<td>400 mg and 1400 mg each</td>
<td>8 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>19</td>
<td>1200 mg</td>
<td>6 mo</td>
<td>NS</td>
</tr>
<tr>
<td>Double-blind placebo-controlled RCT47</td>
<td>94</td>
<td>500 mg†</td>
<td>6 mo</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Level III</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single-blind placebo-controlled crossover RCT47</td>
<td>103</td>
<td>1 g</td>
<td>12 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Open crossover</td>
<td>6+5</td>
<td>400 mg, 400 mg by diet</td>
<td>6 wk for each diet</td>
<td>DBP –8 on low Ca*</td>
</tr>
<tr>
<td>Double-blind placebo-controlled crossover RCT47</td>
<td>18</td>
<td>1 g</td>
<td>15 wk</td>
<td>Standing SBP –8.6*</td>
</tr>
<tr>
<td>Crossover</td>
<td>13 men</td>
<td>400 mg v. 1500 mg</td>
<td>4 wk</td>
<td>NS</td>
</tr>
<tr>
<td>Single-blind placebo-controlled crossover RCT47</td>
<td>8 hypertensive, 8 normotensive</td>
<td>800 mg</td>
<td>8 d</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Statistically significant.
†Combined with K or Mg.
The recommendations on potassium, magnesium and calcium are summarized in Tables 8–10.

**Interpretation**

In the epidemiologic literature, several cross-sectional and prospective cohort studies have not supported a definitive link between the dietary intake of potassium, magnesium, or calcium and blood pressure or hypertension. Initial supportive evidence has been superseded by subsequent negative results in 2 large prospective studies after the method used to estimate dietary cation intake was revised. The method of assessing the blood pressure of participants in these reports was variable: some studies used direct measurements, and others relied on patients to recall their blood pressure as measured in a physician’s office. These variations in methodology for determining both dependent and independent variables undermine the quality of the evidence and the conclusions that can be drawn from it.

The intervention studies are similarly variable. With the “levels-of-evidence” methodology, long-term, randomized controlled trials are the basis for developing recommendations. Since there are few such studies, the recommendations may reflect the results of only one, albeit methodologically strong, study. Short-term studies that use dietary or multiple manipulations are more likely to reflect a real-life strategy for enhancing cation intake, but because they are shorter in length they are not usually used as a basis for recommendations. To date, no long-term studies have evaluated the effect of the increased intake of any of these cations on morbidity and mortality rates.

The recommendations developed from our evaluation of the literature using levels of evidence may differ from those developed through meta-analysis. However, the meta-analyses of potassium supplementation did not support its use in the treatment of hypertensive patients nor to prevent hypertension in those at risk for this condition; this is consistent with our recommendation against potassium supplementation. However, potassium supplementation may be effective in distinct groups of patients, such as those with diuretic-induced hypokalemia, those of African ancestry and those who have low dietary potassium intake. Two meta-analyses and an extensive review of the literature on calcium supplementation and hypertension did not recommend calcium supplementation because the effect was very small and not clinically meaningful.

A question has been raised concerning the definition of “nonpharmacologic management”: Is nutrient supplementation in capsule form a truly nonpharmacologic method, or does it represent a change in diet? For the purposes of this review, studies that used any oral means of augmenting cation intake were included. Some studies changed the intake of more than one cation, which makes it difficult, if not impossible, to determine the effect of a change in a single cation. Dietary change also presents challenges: What additional amounts of which cations were delivered by the dietary change, and which contributed to the blood pressure responses? Changes in the diet to increase the intake of potassium, magnesium or calcium, or any combination of these, may result in changes to other components of the diet, such as sodium, fat or fibre (from fruits, vegetables and cereal grains); these changes may also have a beneficial effect on body weight and on blood cholesterol and antioxidant levels, other factors thought to influence blood pressure in hypertensive patients and morbidity and death from cardiovascular disease.

For the purpose of formulating these recommendations, we assumed that people consume a diet consistent with the recommendations in Canada’s Food Guide to Healthy Eating. Such a diet would provide approximately 60 mmol of potassium and 1 g of calcium per day. Baseline cation intake may be an important factor determining the response to supplementation. As suggested in some of the work on potassium supplementation, the response to supplementation will probably depend on whether the subject is fol-

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**Table 8: Recommendations for potassium intake and supplementation**

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevention</td>
<td></td>
</tr>
<tr>
<td>A dietary intake of 60 mmol potassium per day or more over the long-term is recommended because this level of intake has been associated with a reduced risk of stroke-related death</td>
<td>D</td>
</tr>
<tr>
<td>Potassium supplementation is not recommended for normotensive people to prevent an increase in blood pressure over the short-term when given in addition to an average dietary intake of 60 mmol per day</td>
<td>B</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
</tr>
<tr>
<td>Potassium supplementation is not recommended as a treatment for hypertension when given in addition to an average dietary intake of 60 mmol per day</td>
<td>B</td>
</tr>
</tbody>
</table>

**Table 9: Recommendations for magnesium supplementation**

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevention</td>
<td></td>
</tr>
<tr>
<td>Magnesium supplementation is not recommended for normotensive people to prevent an increase in blood pressure</td>
<td>B</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
</tr>
<tr>
<td>Magnesium supplementation is not recommended as a treatment for hypertension</td>
<td>B</td>
</tr>
</tbody>
</table>

**Table 10: Recommendations for calcium supplementation**

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevention</td>
<td></td>
</tr>
<tr>
<td>Calcium supplemenation is not recommended for normotensive people to prevent an increase in blood pressure when given in addition to recommended daily dietary intake</td>
<td>B</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
</tr>
<tr>
<td>Calcium supplemenation is not recommended as a treatment for hypertension when given in addition to recommended daily dietary intake</td>
<td>B</td>
</tr>
</tbody>
</table>
lowering a deficient diet at baseline; hence a dietary history may be needed for all subjects. Overall, patients should be encouraged to follow a healthy diet.

We are grateful for the external reviews of the Canadian Council of Cardiovascular Nurses, the Canadian Nurses Association, the Canadian Pharmacists Association, the Canadian Public Health Association, the College of Family Physicians of Canada, the Heart and Stroke Foundation of Canada, Hoechst Marion Roussel and Merck Frosst Canada Inc.

The financial assistance of the Laboratory Centre for Disease Control at Health Canada and of Astra Pharma Inc., Bayer Inc., Bristol–Myers Squibb Pharmaceutical Group, Knoll Pharma Inc., Merck Frosst Canada Inc., Searsle Canada Inc., and Servier Canada Inc. is gratefully acknowledged.

Competing interests: None declared for Drs. Burgess, Taylor, Bolli, Hamet and Chockalingam. Dr. Lewanczuk receives speaker's fees, educational grants and travel assistance from various pharmaceutical companies for work related to hypertension.

References

Potassium, magnesium and calcium


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7. Recommendations on stress management

J. David Spence, MD; Peter A. Barnett, PhD; Wolfgang Linden, PhD; Vivian Ramsden, BScN; Paul Taenzer, PhD

Abstract

Objective: To provide updated, evidence-based recommendations for health care professionals concerning the effects of stress management on the prevention and control of hypertension in otherwise healthy adults (except pregnant women).

Options: Alternatives to stress management include other nonpharmacologic interventions and medical therapy; these options are not mutually exclusive.

Outcomes: The health outcome considered was reduction of blood pressure. There is little evidence to date that stress management prevents death or vascular events. Because of insufficient evidence, no economic outcomes were considered.

Evidence: A systematic search of the literature (which yielded, among other sources, 3 meta-analyses) was conducted for the period 1966–1997 with the terms essential hypertension, treatment, psychological, behavioural, cognitive, relaxation, meditation, biofeedback and stress management. Other relevant evidence was obtained from the reference lists of the articles identified, from the personal files of the authors and through contacts with experts. The articles were reviewed, classified according to study design and graded according to level of evidence.

Values: A high value was placed on the avoidance of cardiovascular morbidity and premature death caused by uncontrolled hypertension.

Benefits, harms and costs: The magnitude of the reduction in blood pressure obtained with multicomponent, individualized cognitive behavioural intervention for stress management was comparable in some studies to that obtained with weight loss or drugs; single-component interventions such as biofeedback or relaxation were less effective. The adverse effects of stress-management techniques are minimal, but the cost for effective interventions is substantial, similar initially to drug costs; continuing costs are probably minimal.

Recommendations: (1) In patients with hypertension, the contribution of stress should be considered. (2) For hypertensive patients in whom stress appears to be an important issue, stress management should be considered as an intervention. Individualized cognitive behavioural interventions are more likely to be effective than single-component interventions.

Validation: These recommendations were reviewed by all of the sponsoring organizations and by participants in a satellite symposium of the fourth International Conference on Preventive Cardiology. They have not been clinically tested.

Sponsors: The Canadian Hypertension Society, the Canadian Coalition for High Blood Pressure Prevention and Control, the Laboratory Centre for Disease Control at Health Canada, and the Heart and Stroke Foundation of Canada.

The association between psychosocial stress and atherosclerotic events, such as myocardial infarction, has received considerable attention over the past 2 decades.1–4 The only study to evaluate the relation between stress and stroke found a significantly higher incidence of stroke among men reporting a higher level of stress.5 Significant correlations have also been found between clinical symptoms of coronary artery disease and the type A behaviour pattern,6 as well as high levels of life stress7 and job strain.8 In addition, associations have been observed between type A behaviour and coronary artery atherosclerosis, as assessed by angiography.9–10 Finally, one study found an association between type A behaviour and carotid artery atherosclerosis, as measured by ultrasonography.11

These findings suggest a link between psychosocial factors and atherosclerosis; however, the specific nature of the association is not known. One hypothesis is that cardiovascular reactivity, or the response of the cardiovascular system to stress, may mediate this relation. For example, Manuck and Krantz12 hypothesized that repeated physiologic arousal involving acute changes in hemodynamic and cardiac functioning in response to psychological challenge could trigger atherogenic processes. Hemodynamic forces, such as turbulence and shear stress
(which may be highly influenced by cardiovascular reactivity), may cause or exacerbate existing endothelial damage and promote the development of atherosclerotic lesions. Some support for this hypothesis has been obtained in vivo. Monkeys that exhibited high cardiovascular reactivity to stress (threat with a capture glove) had twice as much coronary atherosclerosis as monkeys with low reactivity to stress.

Recently, it has been shown that the rise in blood pressure during mental stress induced by a frustrating cognitive task is a stronger predictor of progression of carotid atherosclerosis than any of the Framingham risk factors.

The previous Canadian consensus conference on stress management and hypertension found little evidence that interventions designed to reduce the effect of stress on blood pressure were effective and insufficient evidence to recommend stress management as an intervention for hypertensive patients. Since the publication of that report, there has been considerable progress in this area.

**Methods**

A complete description of the methods used in developing these guidelines is given in part 1 of this supplement.

The chair and members of the panel were selected by the Organizing Committee for the lifestyle modification recommendations to obtain a spectrum of health care professionals and scientists with expertise and interest in the areas of psychology, nursing and medicine.

MEDLINE and Psychinfo searches were conducted for the period 1966–1997 with the search terms essential hypertension, treatment, psychological, behavioral, cognitive, relaxation, meditation, biofeedback and stress management. Additional articles were identified by reviewing the reference lists of the identified articles, were found in the personal files of the panel members and were suggested by other experts. Trials, reviews and meta-analyses were considered. The principles for grading the evidence and the recommendations were based on those previously used by the Canadian Hypertension Society and are summarized in part 1 of this supplement. An attempt was made to reach consensus on all recommendations. The evidence and recommendations were presented to the other expert panels for this guidelines series, submitted for review to major Canadian organizations and presented at an international conference on preventive cardiology, to allow for further national and international input. All revisions were reviewed and assessed by the panel before incorporation into the final document.

**Results**

Strategies used in individualized cognitive behavioural stress therapy include increasing awareness of stressors and stress responses, re-evaluating negative life events, communications skills training (e.g., marital communication and assertiveness training), development of problem-solving skills, management of negative emotions (e.g., anger and anxiety) and techniques for decreasing sympathetic arousal (e.g., relaxation exercises).

There is no evidence that stress management prevents hypertension, but there is some evidence that stress management can reduce blood pressure in hypertensive patients. Although the evidence indicated that single-component interventions such as transcendental meditation and relaxation therapy could be efficacious in some centres, meta-analysis showed only small effects or no reduction in blood pressure. In one meta-analysis the change in blood pressure with such interventions was \(-1.5\) to \(+2.9/-0.8\) to \(+1.2\) mm Hg, whereas the change was \(-9/-6\) mm Hg in a second meta-analysis.

A third meta-analysis showed a similar pattern, although the differences between individualized cognitive stress management and other paired or single-component interventions was not as marked (Table 1).

In contrast, multicomponent individualized cognitive behavioural interventions reduce blood pressure to a greater degree and over a longer period of time. Linden and Chambers performed a meta-analysis and found that blood pressure was reduced by \(9.7/7.2\) mm Hg with multicomponent relaxation techniques. With individualized cognitive stress management, blood pressure was reduced on average by \(15.2/9.2\) mm Hg. The key to this approach is tailoring the intervention to the patient's needs.

Table 1 presents the results of overviews and meta-analyses. There was some overlap (approximately two-thirds) in the studies that were included in the meta-analyses, as determined from an examination of the bibliographies of the original papers. Table 2 presents the results of the 6 randomized controlled trials among the 9 studies we found that were not included in the meta-analyses. There was no grade I evidence of effects on morbidity or mortality rates. The study of Alexander and associates came closest to providing such evidence, since it compared mortality rates in a treatment group and a control group. They reported that 73 volunteers in a program in which they were randomly assigned to participate in transcendental meditation, a “mindfulness” intervention or a control intervention had significantly higher 3-year survival than did 478 nonparticipants. However, there is a significant risk of confounding when people volunteer for such a trial, and nonparticipants cannot legitimately be compared with volunteers. Survival was \(77\%\) in the control group, \(87.5\%\) among those who participated in the mindfulness intervention and \(100\%\) among those who did transcendental meditation. These differences were not statistically significant.

One small study showed a significant reduction in requirement for antihypertensive medication with a multicomponent cognitive behavioural intervention. After 12 months blood pressure was controlled without medication in 55% of the treatment group but only 30% of the control patients.

**Recommendations**

- In patients with hypertension, the contribution of stress should be considered (grade D recommendation).
• For hypertensive patients in whom stress appears to be an important issue, stress management should be considered as an intervention. Individualized cognitive behavioural interventions are more likely to be effective than single-component interventions (grade B recommendation).

Interpretation

Knowledge about the effectiveness of stress management is the area that has probably changed the most since the previous guidelines were published. The previous consensus conference was not able to make any recommendations regarding stress management, because evidence to support such recommendations was lacking at that time. There is now sufficient evidence to say that individualized cognitive behavioural stress therapy reduces blood pressure for up to a year. However, at best grade B recommendations can be made because there is as yet no evidence that such therapy reduces important endpoints such as death rate and incidence of myocardial infarction or stroke.

Research in this area has been hampered by methodologic problems. In comparison with the sophisticated dou-

---

### Table 1: Reviews and meta-analyses of studies of stress management and hypertension

<table>
<thead>
<tr>
<th>No. of studies</th>
<th>Subjects</th>
<th>Intervention</th>
<th>Baseline BP, mm Hg</th>
<th>Change in BP, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>26 RCTs††</td>
<td>1264 patients: 723 in therapy, 541 controls</td>
<td>Biofeedback</td>
<td>NA</td>
<td>Systolic: +2.9, Diastolic: +1.2</td>
</tr>
</tbody>
</table>

### Table 2: Individual studies not included in the reviews

<table>
<thead>
<tr>
<th>Study design</th>
<th>Subjects</th>
<th>Baseline BP, mm Hg</th>
<th>Intervention</th>
<th>Results*</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCT‡‡</td>
<td>NA</td>
<td>NA</td>
<td>Large stress-management groups†</td>
<td>No effect (ineffective intervention) on SBP</td>
</tr>
<tr>
<td>RCT‡‡</td>
<td>28 in trial of individual psychotherapy</td>
<td>ABP</td>
<td>Treatment</td>
<td>SBP –8; DBP –5.4</td>
</tr>
<tr>
<td>RCT‡‡</td>
<td>127 black-Americans, 55–85 yr</td>
<td>SBP ≤ 189; DBP 90–109</td>
<td>Transcendental meditation</td>
<td>SBP –10.4 (1.6); DBP –5.7 (1.2)</td>
</tr>
<tr>
<td>RCT‡‡</td>
<td>53 lonely women: 35 therapy, 18 control</td>
<td>Peer support</td>
<td>No support</td>
<td>SBP –6.9; DBP –3.6</td>
</tr>
<tr>
<td>RCT‡‡</td>
<td>73 volunteers, 478 non-participants all elderly (age 81 or older) nursing home residents</td>
<td>Transcendental meditation</td>
<td>3-yr survival 100%, NS</td>
<td></td>
</tr>
<tr>
<td>RCT‡‡</td>
<td>39 patients</td>
<td>DBP &gt; 95 with drug therapy</td>
<td>Multicomponent cognitive behavioural intervention</td>
<td>Reduced medication in treatment v. control group: at 12 mo, 55% of treatment and 30% of control group medication-free</td>
</tr>
</tbody>
</table>

Note: BP = blood pressure, RCT = randomized controlled trial, NA = not available, EMG = electromyographic, temp = temperature.
ble-blind randomized controlled trials for new medical treatments to reduce blood pressure, the quality of evidence on stress and hypertension is in many cases of a lower order. Some studies are not randomized, and it is virtually impossible to achieve a “double-blind” control in studies in which interventions are cognitive. The panel had difficulty finding convincing evidence. In addition, it appears likely that some patients respond to stress management, whereas others do not; therefore, a key issue is identification of those patients who are more likely to respond.

Future research

The strength of the recommendations that can be made at this time is limited by several methodologic issues. This area of research would be improved by attention to the following issues.

Studies should incorporate sufficient pre-intervention visits with the patient (a minimum of 3) to accurately determine baseline blood pressure levels. The role of 24-hour ambulatory monitoring needs further exploration with respect to prediction of cardiovascular events.

Follow-up to assess the efficacy of the intervention should last for at least a year, and even longer follow-up is desirable.

During the course of a stress-management intervention study, the patient should be receiving no drug therapy, or the drug therapy should not change. If drug therapy is to be changed during the course of a study, it is necessary to specify strict protocols for such changes.

Cognitive interventions should be described in sufficient detail to permit replication.

Control groups should receive credible sham interventions. Baseline characteristics should be carefully described. Additional research is needed to identify which subjects are more likely to respond to stress management. The characteristics of “responders” probably include perceived level of stress, life events, emotional arousability, psychophysiological reactivity, family history, white coat phenomenon, expectancies and motivation.

Studies are needed to determine the relation between provocation of blood pressure increases during stressful laboratory tasks, ambulatory blood pressure recordings and life events (which would be an indication of the “ecological” validity or generalizability of the study to the “real world”).

Hardware and software are needed for measuring blood pressure and heart rate from one beat to the next and providing power spectral analysis of heart rate variability to determine the contribution of sympathetic and parasympathetic activity.

Studies of women and various racial and ethnic groups are needed.

Long-term studies of clinically important outcomes such as death and myocardial infarction are ultimately required to show the effectiveness of stress management interventions.

Conclusions

Individualized multicomponent cognitive behavioural interventions are effective in reducing blood pressure; single-component interventions such as biofeedback, relaxation therapy and transcendental meditation are less likely to be effective.

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References


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