
PROMOTING NONPHARMACOLOGIC INTERVENTIONS TO TREAT ELEVATED BLOOD PRESSURE

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PROMOTING NONPHARMACOLOGIC INTERVENTIONS TO TREAT ELEVATED BLOOD PRESSURE

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In this paper, the rationale for the nonpharmacologic treatment of hypertension will be presented. A decision-making matrix for implementing these strategies, the practical issues concerning implementation, the specific therapeutic techniques, and a comprehensive treatment programme which addresses the entire cardiovascular risk profile will also be presented.

Since a variety of health professionals are involved in nonpharmacologic treatment modalities, the term "clinician" is used generically to designate those providing care.

Definition and prevalence

The incidence of hypertension is an epidemic of world wide proportions. In the United States alone it has been estimated that as many as 58 million Americans have elevated blood pressure and, as a result, are at increased risk of morbidity and premature mortality. (1) Prevalence increases with age (2) and is more prominent in the black race who also suffer more morbidity and mortality secondary to hypertension. (3) Regional differences in blood pressure have also been observed. Blacks in the southeastern United States experience an increased prevalence rate and severity of hypertension as well as increased rate of stroke death compared to blacks in other parts of the country. (1) This association of increased prevalence with race and regional differences has also been found in Brazil (4) and parts of Africa. (5) Therefore this increased prevalence rate may have implications in other parts of the world. (6)

Reducing elevated blood pressure protects against major cardiovascular complications such as stroke, congestive heart

failure, renal damage, dissecting aneurysm, progression of hypertension to a more severe state and coronary artery disease. Present evidence indicates that risk increases continuously with increasing levels of systolic and diastolic blood pressure. (7)

While there is consensus to treat hypertension, the precise levels at which treatment should begin, as well as specific therapeutic strategies to be prescribed, are less well agreed upon. The recommendations for treatment of high blood pressure by expert panels worldwide illustrates these differences. The 1987 Report of the British Hypertension Society Working Party (8) and the 1988 Canadian Hypertension Society Consensus Conference (9) recommend treatment for diastolic pressures of ≥ 100 mm Hg for three to four months; the World Health Organization and International Society of Hypertension (1985) recommend treatment for diastolic pressures of ≥ 95 mm Hg; and the 1988 Fourth US Joint National Committee on Detection, Evaluation and Treatment of Hypertension (1), recommends initiating treatment for blood pressures ≥ 94 mm Hg and for blood pressures between 90 and 94 mm Hg when there are associated risk factors. Kaplan analyzed the differences, and concluded that, "As a compromise - and likely as the consensus - a diastolic pressure of 95 mm Hg seems to be the level wherein therapy has been shown to provide protection. That level may then be used in the operational definition of hypertension." (10)

For the purpose of consistency throughout the remainder of this paper, hypertension will be called mild, moderate or severe based on the criteria identified by the 1988 Joint National Committee.(1)

Fourth Joint National Committee Classification of BP^a

Range (mm Hg)		Category ^b
Diastolic	< 85	Normal BP
	85-89	High normal BP
	90-104	Mild hypertension
	105-114	Moderate hypertension
	≥ 115	Severe hypertension
Systolic (when DBP < 90)	< 140	Normal BP
	140-159	Borderline isolated systolic hypertension
	≥ 160	Isolated systolic hypertension

^a Classification based on the average of two or more readings on two or more occasions.

^b A classification of borderline isolated systolic hypertension (systolic BP, 140 to 159 mm Hg) or isolated systolic hypertension (systolic BP ≥ 160 mm Hg) takes precedence over a classification of high normal BP (diastolic BP, 85 to 89 mm Hg) when both occur in the same person. A classification of high normal BP (diastolic BP, 85 to 89 mm Hg) takes precedence over a classification of normal BP (systolic BP < 140 mm Hg) when both occur in the same person.(1)

Systolic hypertension may be as important as diastolic blood pressure as an index of prognosis. (7) Treatment goals should include normalization of systolic blood pressure (<140 mm Hg) as well as diastolic blood pressure. Systolic hypertension should be evaluated and treated with nonpharmacologic and pharmacologic interventions as necessary.

Although general guidelines are useful to establish criteria for treatment, each patient should be considered individually. The late Sir George Pickering had long challenged the assumption that a particular blood pressure level should be considered a cutoff for treatment. He postulated that the risk associated with high blood pressure is a relative risk and should be viewed on a continuum: the higher the pressure, the greater the risk. (11) Rose subsequently identified the operational definition of hypertension as: "the level at which the benefits ... of action exceed those of inaction." (12)

Current trends

The approach to the management of hypertension has changed significantly in the last two decades. Previously, the major emphasis was placed on the detection of curable forms of hypertension (coarctation of the aorta, pheochromocytoma, primary aldosteronism, Cushing's syndrome, and renovascular hypertension) and treatment of premalignant and

malignant hypertension. Subsequently it has been demonstrated that the treatment of primary or essential hypertension results in a significant reduction in stroke and coronary artery disease (7), and the major focus has shifted to the detection, evaluation and treatment of all types of hypertension. Since 1972, there has been an increased public awareness of high blood pressure and an increasing number of individuals being treated for high blood pressure. These factors have likely contributed to the decline in the national age-adjusted stroke mortality (50%) and the decline in coronary artery disease mortality (35%) in the United States over the last two decades. (1)

Expert panels in the last three years have convened around the world to evaluate the most efficacious way in which to treat hypertension. The US Joint National Committee notes that if the decline in stroke and coronary artery disease noted in the 1988 report is to continue, that a number of events must occur including:

- * The hypertension control process must be extended to the entire population, and
- * Aggressive treatment must also take into consideration the life-styles and concomitant conditions of individual patients.

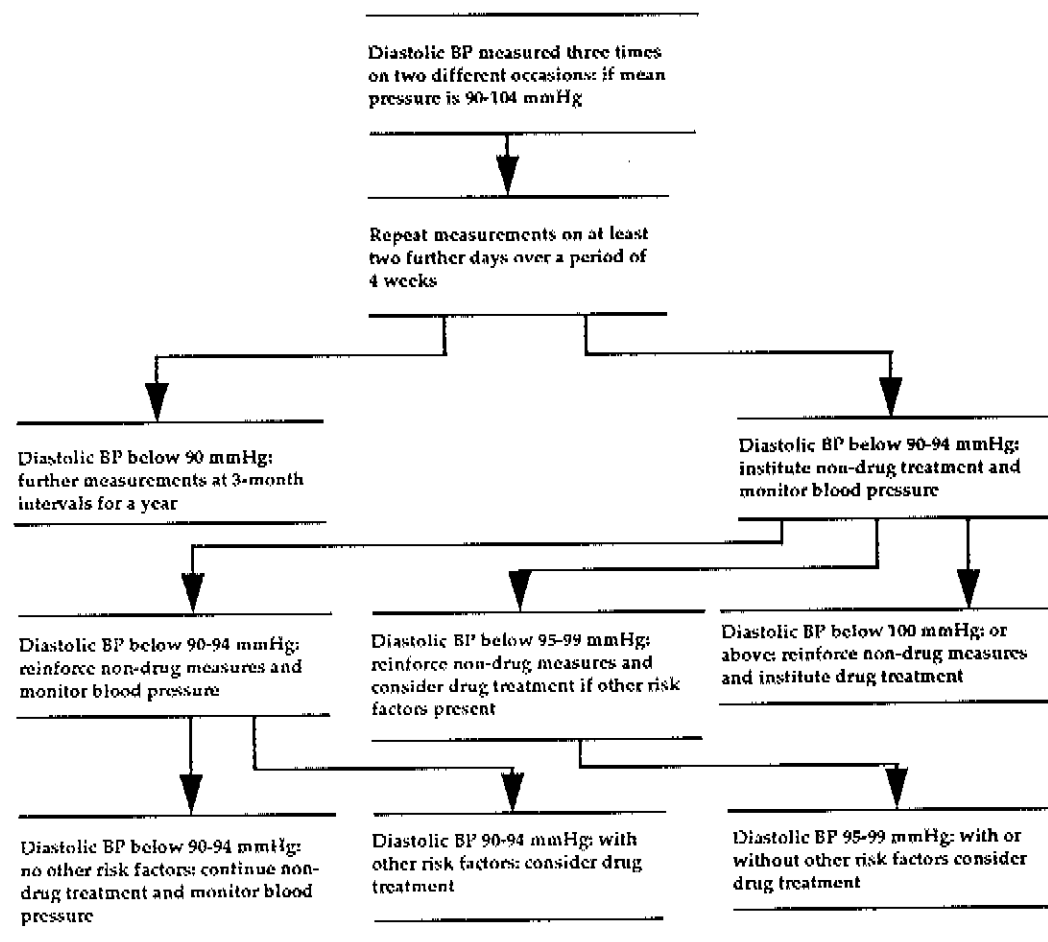
The increased numbers of therapeutic options, nonpharmacologic as well as pharmacologic, provides the opportunity to improve hypertension control while minimizing adverse effects that may influence the efficacy of therapy and incur complications.

Endorsing the philosophy that the treatment options for each person with hypertension must be considered individually, Dr. Alan Brett wrote; "It is widely acknowledged that, with successively higher blood pressure levels, the risk of complications increases gradually rather than abruptly. Therefore, the reasons to intervene should be viewed as gradually more compelling as blood pressure rises, rather than suddenly compelling at a specific level such as 90 mm Hg. Each decision

must be individualized, depending on the patient's aversion to risk, perception of the intrusiveness of medical care in his life, tolerance for discomfort or untoward drug effects, etc." (13) Careful attention need therefore be taken with each patient to choose the most appropriate, least intrusive and specific nonpharmacologic and/or pharmacologic intervention(s).

The goal of treating patients with hypertension is to prevent morbidity and premature mortality associated with elevated blood pressure. The objective is to achieve and 'maintain blood pressure measurements below 140/90 mm Hg. (1) In choosing the appropriate treatment one must consider the severity of blood pressure elevation and the presence of other complications.

The following flow chart illustrates this concept.



Recommendations for the definition and management of mild hypertension by participants at the Third Mild Hypertension Conference of the World Health Organization and the International Society of Hypertension. (From World Health Organization/ International Society of Hypertension. *J Hypertens* 7:689, 1989).

Within the context of this treatment paradigm, the present paper will focus on the nonpharmacologic interventions. After a general introduction, each intervention will be discussed separately and in detail, considering ways to apply it to a general population and taking into consideration the lifestyles and concomitant conditions of individual patients.

Nonpharmacologic treatment of hypertension

There is endorsement from the World Health Organization (6), the Joint National Committee for the Detection, Evaluation and Treatment of Hypertension (1), the Canadian Consensus Report Society for Hypertension (9) and the British Hypertension Society Working Party (8) concerning the efficacy of nonpharmacologic approaches. Recent evidence cited by these panels suggests that nonpharmacologic therapies may lower elevated blood pressure and improve the efficacy of pharmacologic agents. In this context, nonpharmacologic interventions can be used both as a definitive intervention in mild to moderate hypertension (diastolic ≤ 114), and as an adjunct to pharmacologic therapy for those individuals with severe hypertension (diastolic ≥ 115 mm Hg) or the presence of other significant risk factors.

In view of the fact that 80% of those individuals diagnosed with hypertension have mild to moderate elevations of blood pressure (90 - 104 mm Hg), nonpharmacologic therapies should be used as initial therapy for most patients, at least for the first three to six months after recognition of their hypertension. For those 40% of patients with diastolic levels between 90 and 94 mm Hg, nonpharmacologic therapy may be sufficient to reduce blood pressure to the recommended normotensive level. (14) Systolic blood pressure reductions between ten and 15 mm Hg and diastolic blood pressure reductions between six and 10 mm Hg are typically achieved in nonpharmacologic intervention programmes. (15) This is clinically significant since it has been estimated that each four mm Hg reduction in blood pressure is accompanied by a 10.8% decrease in cardiovascular risk. (16) The modest decrease in blood pressure associated with nonpharmacologic therapies has an additional benefit since it has been demonstrated that reductions of greater than 10-20 mm Hg are associated with increased morbidity and mortality. As many as 20 to 25% of patients with mild hypertension can become normotensive with nonpharmacologic interventions. Medicated hypertensive patients who add nonpharmacologic treatments to their regimens may be able to maximize blood pressure control on a minimum of antihypertensive medications.

Advantages of nonpharmacologic approaches are reduction in adverse pharmacologic side effects as well as medication costs. In addition, and significantly, nonpharmacologic interventions generally address the entire risk profile.

Effecting behavioural change

Clinician responsibility

While most clinicians treating hypertension now consider nonpharmacologic treatments to be effective, particularly for patients whose diastolic pressures are between 90 and 94 mm Hg, the practical issues regarding patient motivation, monitoring, and engineering behaviour change can appear formidable. A legitimate concern of many clinicians is whether such interventions can be successfully and practically accomplished. They are not comfortable instituting and monitoring the behavioural changes required. Whereas most clinicians support, at least in theory, the use of nonpharmacologic treatment of mild hypertension, very little time is usually spent counseling patients with respect to the behavioural interventions required.

The reasons for this discomfort are threefold. First, an overwhelming majority of clinicians (particularly physicians) feel that their training did not adequately prepare them for counseling patients on how to bring about the important behavioural changes required. Consequently, the use of nonpharmacologic treatment approaches for hypertension may be more inhibited by physicians discomfort with teaching behavioural change than by the ability of the interventions to bring about the desired goal. Second, many physicians report little confidence in the effectiveness of their recommendations for nondrug approaches to hypertension control. Third, most physicians report that there is little time in an office visit to bring about behavioural change. Nurses feel better prepared to address behavioural change and endorse its value and utility, but also find little time to accomplish this objective.

To address these issues clinicians need to consider the scope of what can be accomplished in the office as well as to consider referring their hypertensive patients for nonpharmacologic treatment to centers or practitioners who routinely engage in this type of endeavor.

The role of belief

The personal bias of the clinician toward the cost/benefit of changing a specific behaviour also needs to be considered. If a clinician believes that exercise is unpleasant, that losing weight means giving up everything that tastes good, and that it is difficult to change a lifestyle behaviour, it is likely that this practitioner will not be as effective in helping a patient engineer behavioural change as will a clinician who holds a more positive view. An explanation stems from an understanding of the mechanisms responsible for the placebo effect.

Eliciting the placebo effect is dependent on three factors: the belief of the practitioner, the belief of the patient, and the relationship between the patient and practitioner. (17) In studies conducted by investigators who enthusiastically endorsed the validity of the intervention being studied, the results were more likely to be positive than studies done by skeptics. (18) It is likely that the enthusiastic clinicians conveyed their belief in the value of the intervention to the patient, and that this directly influenced the outcome. Thus, the belief of both clinician and patient is inherently important.

A recent study illustrates this point. Horowitz et al reanalyzed the results of the Beta Blocker Heart Attack Trial, controlling for adherence. (19) The relationship of medication adherence to mortality in the first year after a myocardial infarction was investigated among 2,175 participants. Those who did not adhere well to their medication regimen (i.e., who took less than 75% of their medications) were 2.6 times more likely to die than were those who did adhere to their medication regimen (i.e., who took more than 75% of their medication). The surprising finding in this study was that those participants assigned to placebo had approximately the same death rate when they did not adhere to their placebo medication regimen (2.5% incidence of death) as did those assigned to a Beta-blockade group (3.1% incidence of death). This would suggest that not adhering to a prescribed medication regimen increased mortality in the first year following myocardial infarction and that the increase in mortality occurred irrespective of whether the person took placebo or active ingredient. From this it might be construed that the act of taking medication (belief) might be the active ingredient rather than the pharmacokinetics of the drug itself. In the current environment where both patient and clinician highly value medication in the treatment and cure of illness, the depth of this belief becomes apparent. This study illustrates the utility of harnessing the power of the patients belief in effecting optimal outcomes.

Therefore it becomes more important for the clinician to engender belief that the intervention is valuable, that the benefits of the treatment exceed the cost, and that the patient can be successful in effecting the change.

Data describing reactions to the diagnosis of hypertension also emphasize the role of beliefs. One risk attendant in labeling an individual as "hypertensive" is to evoke the assumption of the sick role. There has been so much, albeit necessary, education about the dangers and risks associated with hypertension, that much of the public holds a strong opinion about this diagnosis. In one frequently cited study, the diagnosis of hypertension, without any change in symptoms, was accompanied by an immediate increase in absenteeism from work. (20) This is not a trivial effect. In a Canadian study the average yearly income of 230 hypertensive steel workers was an average of \$1093 less than for a matched group of normotensives, despite similar incomes in the year before diagnosis. (21) MacDonald et al concluded in a review article that people who are labeled, but who receive appropriate counseling, support and care and who are compliant with therapy usually do not have increased absenteeism or psychological distress. (22) Clearly this phenomenon needs to be considered and clinicians need to address this directly with patients. In this context, family, societal and culture beliefs which also profoundly affect the patient's perception of health, illness and treatment need to be considered.

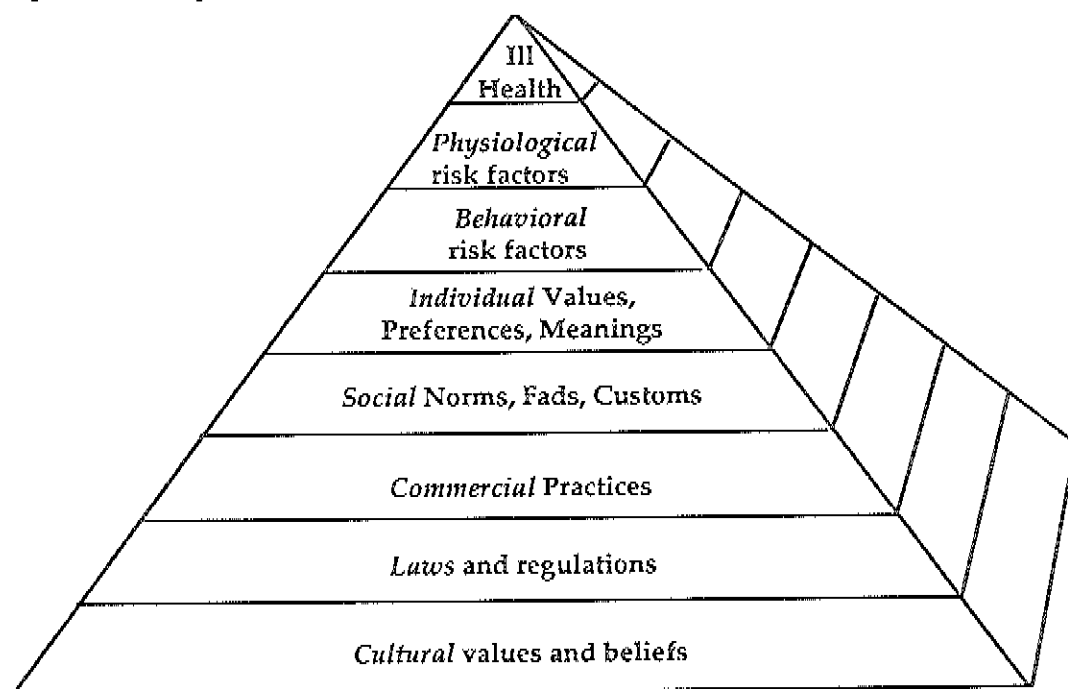
Antecedent factors which influence the treatment of hypertension

In assessing an individual's motivation and readiness to adopt healthy behaviours to prevent and/or adjust to illness and to change adverse lifestyles, the role of antecedent factors must be considered.

It is perhaps a paradox that in the health care system the greatest percentage of resources are allocated to medical care, while precious few resources are dedicated to understanding and treating those behaviours which underlie illness. In 1979 Joseph Califanos MD, then the US Surgeon General, issued *Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention*. (23) This report noted that as many as half the premature deaths in the United States may be due to unhealthy behaviours or lifestyles. It suggested that of the ten leading causes of premature death in the United States, at least seven could be substantially reduced if Americans altered their lifestyles. These lifestyles included poor diet,

smoking, lack of exercise, alcohol excess, and unhealthy responses to tension and stress. In 1991, while the technology available to treat the complications of hypertension have advanced tenfold since 1979, there are still few monetary resources dedicated to understanding and promoting health seeking behaviours.

The following is a conceptual framework developed by Tarlov which allows consideration of relevant factors as they apply to the genesis of illness.



From: Tarlov AR. The rising supply of physicians and the pursuit of better health. *Journal of Medical Education*. 1988;63:104.

In the above illustration, ill health is viewed as being at the apex of a pyramid of antecedent underlying factors, in which the medical/cultural value plays a role. (24) The way in which this model relates to cardiovascular disease in general and hypertension in particular will be described. Priorities in research funding to study the prevention and treatment of hypertension have favoured genetic causes or new interventional techniques. There is no doubt that genetic factors play an important role. But these genetic factors are influenced by a variety of other factors which can trigger and/or overwhelm biology and predilection to develop hypertension.

The physiological risk factors which predict the development of cardiovascular disease include hypertension, hypercholesteremia, smoking, sedentary lifestyle and stress, as well as others. But each physiological risk factor - in this example hypertension - is directly influenced by behavioural antecedent factors including a diet high in sodium, fat and calories with too little exercise, too much stress, and social pressure to smoke and consume alcohol.

Each individual has a set of values, preferences and meanings which greatly influence their attitude toward life, health and health seeking behaviours. Several conceptual models have evolved to explain the role of values and beliefs in predicting treatment outcomes and adherence. One of the most popular, the Health Belief Model proposes that in order to be successful in effecting behavioural change, the individual must perceive the benefits of change as outweighing the cost of change. (25) Other theories, including Locus of Control (26) - either internal or external - and Self-Efficacy (27) - how successful the individual believes that they can be in bringing about change, acknowledge that the individual's perception of their role in their care greatly influences the outcome.

These personal belief systems should be viewed in conjunction with the family, social and cultural norms which nurture them. In the society of two-income families, for example, fast food and prepared food are becoming the norm. These foods are high in calories, fat and sodium, and lacking in potassium and other important nutrients. This eating pattern is reinforced by commercial practices which market the

attractiveness of these products to busy families, and laws which do not promote labeling which might educate the consumer. Underlying and supporting the pyramid are cultural values and beliefs which support the notion of freedom of choice with minimal regulation and restriction.

Clearly, the primary concern of the health care system and the clinician has been at the top of this pyramid, the diagnosis and treatment of ill health. In order to treat hypertension effectively, considering the multiplicity of antecedent factors, it is important to view each individual patient as a biopsychosocial being, and to intervene with a specific understanding of these myriad factors. In addition, clinicians should concern themselves with the broad public health policies which influence progress towards achieving the desired blood pressure objectives.

Stages of change

Clinicians need not become behavioural change experts to endorse the utility of nonpharmacologic therapy, or to treat their patients using these approaches. Once the specific problems have been identified, the first step for the clinician is to endorse the value of nonpharmacologic therapy. It is important to engender confidence on the part of the patient that they can be successful in changing adverse behaviours, and that the practitioner will be there to help them.

A second step involves the endorsement of the patient's participation in a programme that is specifically designed to bring about the desired changes.

The third step is a willingness to reinforce the changes once the programme is completed when the patient returns for follow-up care.

These are realistic goals for a clinician to accomplish within the context of a brief office visit. It is not always practical or cost effective to establish programmes within their office to bring about the behaviour change required for successful nonpharmacologic interventions. It is, however, crucial to appropriately motivate and empower patients.

Patients who are appropriate candidates for nonpharmacologic intervention may be at a variety of stages regarding their readiness to change. It is imperative for the clinician to assess the patient's readiness to change and to intervene in the most effective manner. Drs. Prochaska and DiClemente, have described four stages of change. These are: precontemplation (no information, or not interested, not motivated), contemplation (interested, motivated), action (ready to change, motivated), and maintenance (including relapse prevention) (28).

The relationship between implementation of nonpharmacologic treatments for hypertension and the decision making stage of a hypertension patient is crucial. The first goal of the clinician would be to assess a patient's stage regarding his condition.

For some hypertensive patients, the initial issue involves presenting the importance of lowering blood pressure and reducing other cardiovascular risk factors. Some patients simply do not know that high blood pressure is dangerous and should be lowered. Others may appreciate the fact that high blood pressure should be lowered, but will assume that only drugs can be effective. Both types of patients are at the precontemplation stage. The clinician's primary responsibility is to communicate the importance of reduced blood pressure and to indicate the effectiveness of nonpharmacologic interventions. For patients in the precontemplation stage, the clinician should not only communicate the effectiveness of such interventions but also assess the patient's willingness to help achieve the goals.

Once patients have moved past the precontemplation stage into the contemplation stage, more specific information is required. For example, at this stage, a discussion about the ways in which dietary salt can be reduced should occur. The patient's anticipated difficulties associated with making behavioural changes should also be considered. Precise information concerning the ways in which the programme can be implemented should be discussed.

During the contemplation stage, the clinician should explain the potential benefits of exercise, techniques to elicit the relaxation response, and stress management to the patient. The transition from the contemplation stage to the action stage requires a presentation which does not threaten the patient. Of course, behavioural change can be difficult and patients should not be misled. However, systematic programmes on life-style change to reduce cardiovascular risk need not be overwhelming. In the contemplation stage, the clinician must carefully set the stage for action. He or she must let the patient know that the action plan will occasionally cause some frustration and relapsing and although this is normal and expected, success is possible.

Many patients tend to approach the contemplation stage with an ambivalent attitude. They may perceive attempts to intervene nonpharmacologically as attempts to "get me to stop doing everything I enjoy and get me to do everything I hate." Obviously, this would need to be addressed in order to move a patient to the action stage. It is often useful at this point to have the patient complete some type of values

clarification exercise. In this way the patient can view the cost/benefit ratio of changing in relation to the larger issue of what they value. Another useful strategy is to have the patient review past experiences with successful behaviour change, and then to engender the expectancy that they can be successful in this endeavor. Presenting the intervention in light of what patients **can do** instead of focusing on with they **cannot do**, is important in engendering a positive attitude and engineering a successful outcome.

Once patients have entered the action phase, specific, measurable, incremental goals should be mutually agreed upon, stated and monitored. During the contemplation stage, patients become convinced, for example, that sodium restriction or weight loss is both necessary and possible. During the action stage, agreed upon reductions below 2,000 mg per day and weight loss of one and a half pounds per week must be monitored. Clinicians should focus on imparting critical information, teaching appropriate self-care skills and engendering a positive attitude in order to accomplish these behavioural changes. (29) The patient should feel a sense of partnership in achieving goals, and a sense of individual responsibility consistent with a selfcare approach should be developed.

The clinician's knowledge concerning nonpharmacologic interventions and their utility in hypertension treatment is usually all that needs to be communicated to the patient to move him or her from precontemplation to the contemplation stage. More of a dialogue concerning feasibility and more appreciation of the patient's particular circumstances and strengths is usually required to move the patient to the action stage. Once there, however, a fundamental decision needs to be made. The decision concerns the implementation of the intervention plan. In many, if not most cases, the clinician will not be practically prepared to initiate the type of systematic and comprehensive plan which will most likely lead to success. In these cases we recommend referral to specialized programmes which assess the patient utilizing a biopsychosocial model, whose interventions address knowledge development, skills acquisition, and attitude, and which include the patient as a partner in care. A group format may be most advantageous in this regard, providing structure and social support as well as being more cost effective.

For those patients who are motivated and interested in pursuing behavioural change on their own, the referring clinician should follow a similar format but more of the responsibility for knowledge development and skills acquisition falls to the patient. Goals should be mutually agreed upon. The patient

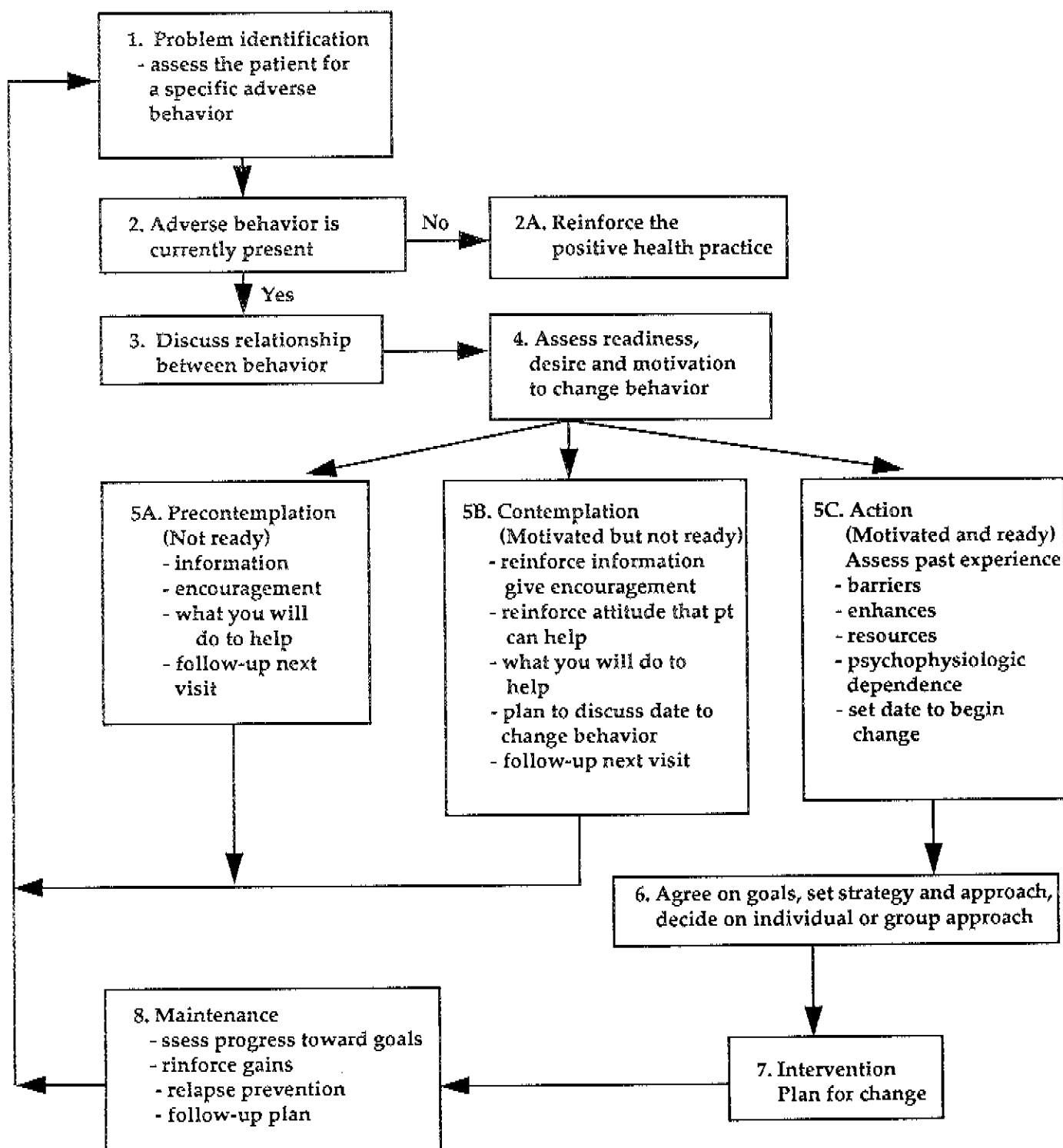
should seek out information and resources for self-help which will provide the structure for their intervention. The clinician should assess and monitor the patient's progress.

Prior to completing the intervention or action phase, it is important to provide patients with a framework for maintenance and relapse prevention. They need to learn specific self-monitoring skills such as diary keeping, home blood pressure measurement, weekly weights, and fat-gram counting, as well as many others. Patients need to learn to anticipate high risk situations when lapses and relapses may occur, and they need to know when to seek help to prevent a lapse becoming a more entrenched situation. The patient should understand that movement through the stages of successful behaviour change is bidirectional and circular, not linear. This is important. When a person does encounter a lapse, they should not feel that it is the end of the line, but rather that they can move back into action, or around to precontemplation and begin again. The referring clinician is critical in assisting the patient to maintain the health gains which they have achieved. Periodic review of the patients progress and self-monitoring data are important to validate the self-care role of the patient and reinforce the hard won gains which they have achieved. Adherence to nonpharmacologic intervention strategies will not be uniform and estimates of compliance vary widely. While adherence to nonpharmacologic therapy is certainly an issue, adherence to antihypertensive pharmacologic regimens also represents a significant challenge to the clinician. This however is hardly a reason to abandon either pharmacologic or nonpharmacologic treatment.

In spite of a clinician's best efforts, there are some patients who do not desire to approach their medical problems from a nonpharmacologic, behavioural perspective. Some cannot easily accept the rationale for reducing medical symptoms using behavioural strategies; others may see the "cost" in personal terms as outweighing the benefits. For a variety of reasons, the patient may be unwilling or unmotivated to make the required behavioural changes at this time. It is, however, vital in these situations to follow-up at the next visit, because the patient may be ready at that time. Additionally, a referral to a Behavioural Medicine clinician might be helpful.

The following is a flow chart which delineates the decision making process for effective behavioural change.

Decision making matrix for behaviour change



Nonpharmacologic intervention strategies

There are some well-established, nonpharmacologic, antihypertensive therapeutic interventions: weight reduction, sodium restriction, reduced alcohol consumption, exercise, elicitation of the relaxation response, stress management, and smoking cessation. The procedures by which these nonpharmacologic interventions can be coordinated into an effective treatment programme will be described later.

In prescribing these nonpharmacologic interactions for individual patients, one problem which remains is the heterogeneity of response. A fundamental question concerning the utility of nonpharmacologic treatment strategies for hypertension concerns therapeutic efficacy for the individual as well as the aggregate. In addition, the diagnostic process and prescription of nonpharmacologic therapies often lacks precise sensitivity and specificity. An obvious example is that of weight reduction. While an obese patient can significantly reduce blood pressure with weight loss, a thin hypertensive patient would be unlikely to benefit from this strategy. Due to differences in underlying pathophysiologic mechanisms, some hypertensive patients may be more or less likely to profit from exercise training, sodium restriction or elicitation of the relaxation response.

While the presence or absence of some relevant characteristics are easily determined such as obesity and smoking, other factors are less easily observed, for example, sodium insensitivity and exercise conditioning. Therefore, careful attention needs to be paid to assessing the risk profile as carefully as possible and intervening in the most specific manner.

In the following pages, each of the nonpharmacologic components will be described separately before presentation of a comprehensive treatment approach.

Weight reduction

Scientific Rationale for Weight Reduction

Weight reduction for obese hypertensive patients should be the first treatment strategy for mild hypertensive patients and a complementary treatment strategy in all obese patients requiring drugs. There is consensus in the literature that obesity is commonly associated with hypertension. (30) A strong correlation between body weight and blood pressure, and between increased body weight and increased blood pressure has been demonstrated. (31)

Conversely, a decrease in body weight correlates with a decrease in blood pressure. (32) The mean effect of a one Kg fall in body weight is a 1.6/1.3 mmHg fall in systolic/diastolic blood pressure. (33) Further, significant reductions in blood pressure can be achieved with only modest decreases in weight. (34) Weight reduction intervention is recommended for patients who are more than ten to 20% above ideal body weight (IBW). Body mass index (BMI), hip/waist measurement and percent body fat should also be taken into consideration.

The issue of whether weight reduction will lower blood pressure without sodium restriction is still debated. The US Joint National Commission stated that weight reduction may occur with calorie restriction alone, even without reduction in sodium intake and before ideal body weight is achieved. Kaplan (33), however, reviewed a variety of studies including one by Fagenberg et al who found little antihypertensive effect associated with weight loss without concomitant sodium restriction. (35) The majority of studies reviewed do not isolate the two interventions. (36)

There appear to be three mechanisms by which weight loss effects lowered blood pressure. The initial response to a low calorie diet is natriuresis, with an accompanying decrease in weight. (37) Thereafter, a decrease in sympathetic nervous system activity (38), and a decrease in plasma insulin occurs. (39)

Behavioural Strategies for Weight Reduction

Clinicians should vigorously promote weight control, particularly in those with a family history of obesity. This recommendation is made with full understanding of the difficulties in engineering this change, and the high rate of relapse. Weight loss programmes for hypertension must be considered in conjunction with the cultural and socioeconomic situations confronting patients. In some impoverished societies, obesity is not a widespread problem. In other societies, these may be little social pressure to lose weight, while the social pressure to be thin in other societies may be great.

It is unlikely that obese hypertensive patients will substantially alter their behaviour concerning body weight just because they became aware of the relationship between body weight and blood pressure. Clinicians must not only educate, but also motivate patients to begin the difficult behaviour change process. Taking the time and effort to appreciate the patients perspective on weight loss is important. Developing a practical and effective programme requires sensitivity to the life circumstances of the patient. Very low caloric formulas can be

effective (short term) and safe. (40) However, these formulas are expensive and require careful medical supervision. Less stringent diets combined with exercise and behavioural techniques also work. (41) However, these programmes must be rigorously and systematically implemented. Weight reduction should be approached as a multifaceted problem. In addition to education regarding the nutritional, caloric and fat content of food, the patient should be instructed in label reading, menu selection and food preparation. They should be encouraged to exercise, elicit the relaxation response and investigate the behavioural aspects of their eating patterns. Assess not only what people eat, but where, when and how they eat. It has been estimated that 50% of meals eaten by the typical American are eaten away from home or in front of a television. In addition to addressing individual differences, food plans should be flexible, enjoyable and focus on those things the patient can eat rather than those things that they can not.

The specifics of the intervention for obese hypertensive patients do not differ greatly from those usually applied to other obese patients. Patients need to be apprised of healthy eating choices available to develop an increased awareness of dietary habits. This requires careful attention to monitoring food intake and behavioural habits on a consistent basis. Self-monitoring both food intake and progress towards goals - through a food diary and weekly weights - are critical to a successful programme. The food recall diaries should be for three or more days. Some clinicians express reservations about the accuracy of these recalls. However, many intervention programmes report that the technique can be successful if it is introduced as a way for the patient to contribute to their care, and if they understand that successful weight loss depends on accurate record keeping.

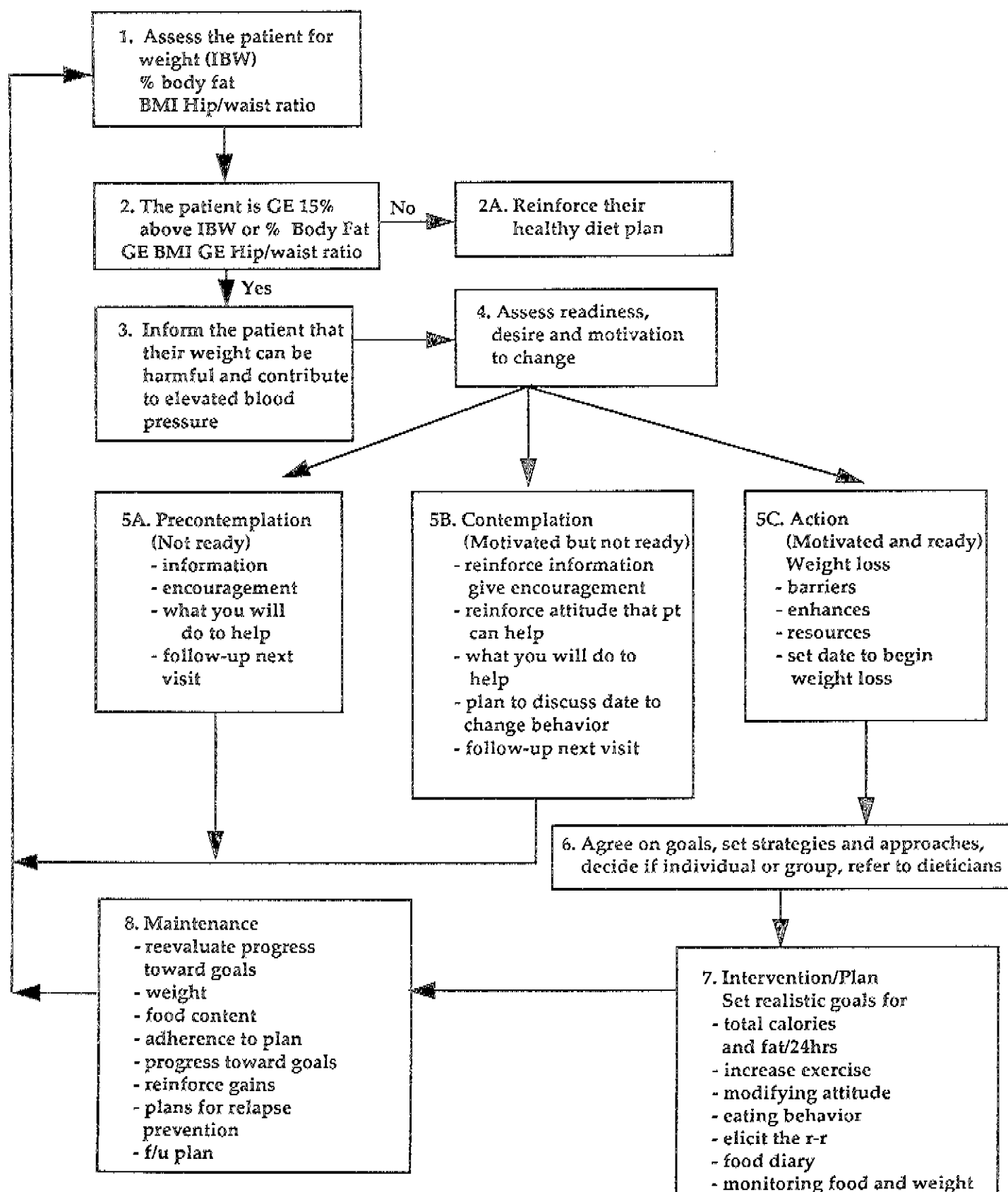
The positive aspects of diet planning should be stressed, and the patient should be encouraged to design their own strategies for change. It is not enough to tell the patient that he should not eat high calorie snacks. The patient needs to plan what he will snack on when he sits down to watch television, or to avoid the television by taking a walk. Anticipating hurdles and high risk or tempting situations is important, and contingency plans should be developed. Initial adherence to the

food plan is highly predictive of long-term compliance, therefore it is important to programme an early success. (42) The patient should learn that foods lower in calories also tend to be lower in saturated fat and sodium thereby maximizing cardiovascular benefits. Impress upon the patient that dietary change requires dedication and commitment. Again, the dietary changes recommended must be developed with a sensitivity to the environment in which the patient lives.

An example of the sensitivity required is illustrated by the important difference a clinician would need to maintain in developing a programme for a group of affluent hypertensive women in a suburban United States community as compared to a group of poor inner-city women in the United States. Advice concerning available food choices, specific exercise activities and medical supervision should account for the dramatic differences in personal resources, food choices, community resources and access to medical care between the two groups. Monitoring progress on a regular basis is necessary to achieve optimal adherence. Dietitians are important collaborators in achieving these ends. Realistic expectations concerning weight loss are also necessary. The short-term goal is weight loss of between one to 1.5 pounds each week. The long-term goal is to achieve and maintain body weight within ten percent of ideal body weight.

Advice to "lose weight" or "cut back," no matter how well-intentioned, will not likely yield positive results. Most weight loss programmes succeed in the short term, but fail in the long run. Those programmes based on behaviour modification principles and which include exercise and psychological factors such as elicitation of the relaxation response and stress management work better than approaches which do not include these components. In our experience, patients do best when referred to programmes which offer a comprehensive approach to weight loss as part of our overall risk profile. The referral process for this intervention and for all other nonpharmacologic therapy is a crucial aspect of the treatment. Clinicians should enthusiastically endorse weight loss as a way to reduce blood pressure and lower cardiovascular risk. They should also convey their firm belief that the patient can succeed. Long-term maintenance is enhanced with appropriate follow-up support.

Decision making matrix for weight reduction



Sodium restriction

Scientific Rationale for Restricting Sodium

The US Joint National Commission stated that "a high sodium intake plays a critical role in maintaining the elevated blood pressure of some hypertensive patients and in limiting the effectiveness of certain antihypertensive drugs." (1)

The Canadian Consensus Report (9), the World Health Organization (7), and the Australian National Health and Medical Research Council (43), concurred. Kaplan points out that some controversy about the utility of sodium restriction remains, however. (44) This controversy is most likely due to the fact that precise measures to determine who will benefit from this intervention are not available. The effects of sodium restriction on individual patients cannot be predicted with certainty. Although this diagnostic ambiguity exists, it is also true that sodium restriction has been shown generally to be beneficial in lowering blood pressure, and moderate restriction has not been associated with any serious adverse consequences. (1)

There is a consensus that modest restriction of dietary sodium - 70 to 100 mmol/day - will lower blood pressure four to

eight mmhg below levels seen with a baseline diet which is high in sodium. (32) Not only is there a correlation between decreased sodium intake and decreased blood pressure, but most patients can successfully adhere to this diet. As noted previously however, not all patients will show a decrease in blood pressure with a modest reduction in sodium. Initial blood pressure may be the most important factor related to the effect of sodium restriction; the higher the initial level, the greater the reduction. (45) In general, elderly and black hypertensive patients seem to benefit from sodium restriction more than young and white patients. Genetic factors have been cited as predisposing individuals to respond to sodium restriction, but this theory is not universally endorsed. (36)

Although the precise mechanism by which sodium restriction lowers blood pressure is not agreed upon, there are several hypotheses which have evolved to explain the relationship between sodium intake and blood pressure regulation. Of these, the autoregulation theory, the natriuretic hormone hypothesis and altered cellular membrane function have been widely accepted. (36)

The following chart summarizes the recommended changes in the consumption of dietary fats, cholesterol, sodium and fiber, and demonstrates the potential impact of these changes. (42)

Current and Recommended Dietary Intakes

<i>Nutrient</i>	<i>Current Intake</i>	<i>Recommended</i>
Dietary fat	80-100g (40%)	40-60g (30%)
Cholesterol	350-400mg	300mg
Sodium	150-200meq	70-100meq
Fiber	10-15g	25-30g

Potential Benefit From Diet Changes

<i>Diet</i>	<i>Diet Intake</i>	<i>BP/Cholesterol Change</i>
Sodium	70-100meq/day (20-40% reduction)	DBP ↓ 2-8mm Hg
Weight	10-15 lb (4-7 kg) weight loss	DBP ↓ 4-10mm Hg cholesterol ↓ 30-60 mg/dL
Fat	30% fat (phase 1) 20% fat (phase 2)	cholesterol ↓ 30-45 mg/dL cholesterol ↓ 45-60 mg/dL

From: Leon AS. Weight Loss In: New Approaches to Cardiovascular Risk Management.

Behavioural strategies to reduce daily sodium intake

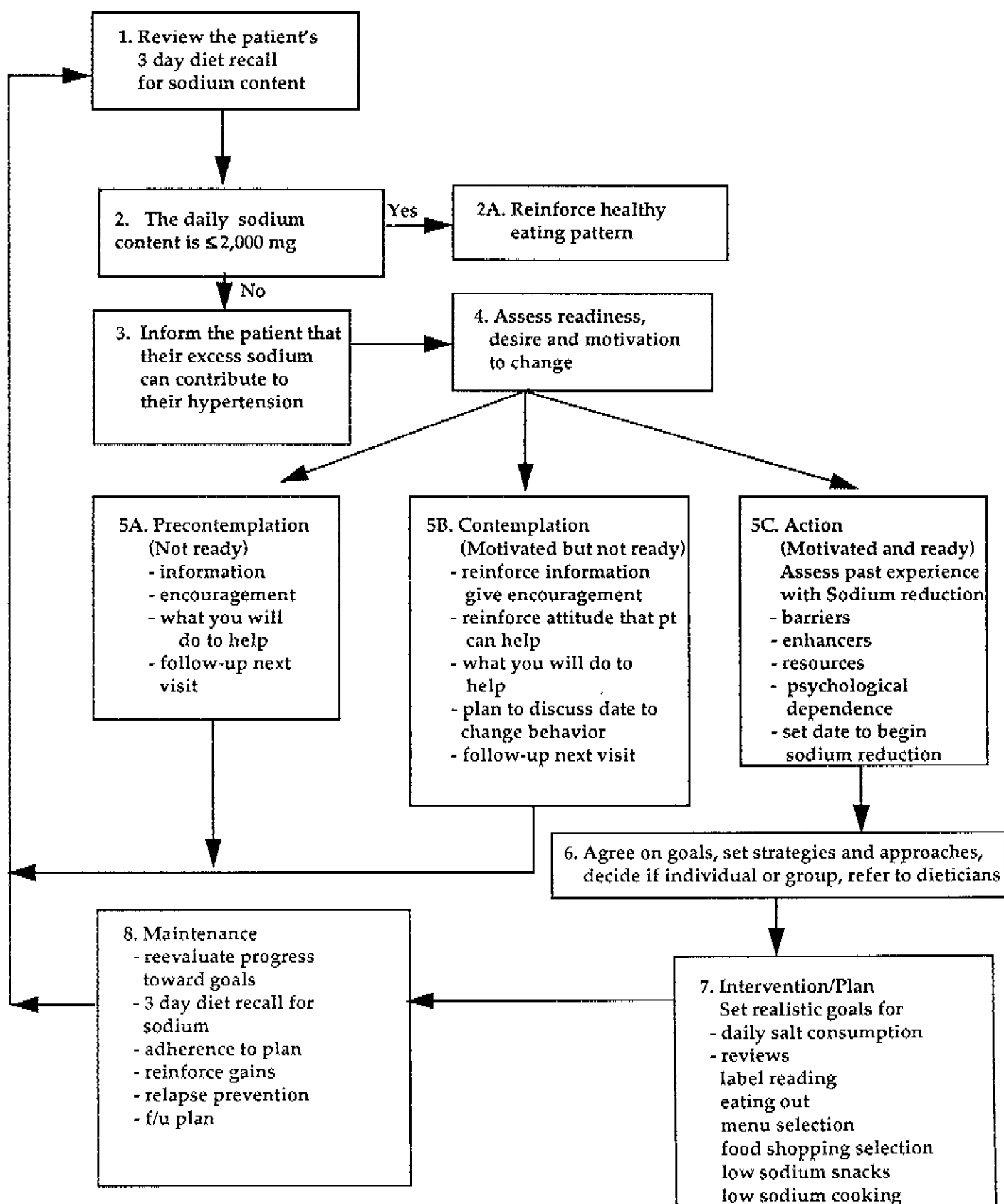
Most agree that for sodium restriction to be therapeutically efficacious daily consumption needs to be reduced to or below 2,000 mg. Some patients find this difficult, although in our experience, it is a realistic and attainable goal. To place this goal in perspective, 2,000 mg per day is ten times above the necessary metabolic requirements. The problem however is that the average American may ingest between 8,000 and 9,000 mg of sodium per day, and their taste preferences are nurtured to enjoy salt. In view of the benefits of restricting sodium, why are these diets not more widely followed? In 1972 Dahl (46) put forth three reasons which are still appropriate. First, we are accustomed to the taste of salty food since infancy. Second, most clinicians prescribe low sodium diets unenthusiastically and haphazardly. And third, these diets may initially seem complex, and discourage patients. In 1991, we might add a fourth reason, social and cultural norms which promote fast/prepared/processed food. Currently there is a compelling need to consider additional, powerful deterrents to reducing sodium. Nearly 60 to 80% of our daily sodium intake in the United States (47), Canada (48), and England (49), comes from processed food, and this food may be inadequately labeled. In addition, in the United States, it is estimated that twenty percent of our meals are "fast food," which are typically high in sodium and fat (49). The sensitivity to the individual circumstances of a hypertensive patient, which we discussed regarding weight loss, is also required when addressing sodium restriction. There are occasionally practical barriers to sodium restriction. For example, in some cultures and among some ethnic groups, salty food is the norm. Asking patients to shop differently, cook differently and eat in different restaurants, impacts on family members as well as patients themselves. Careful planning and cooperation between clinician and patient can usually overcome these difficulties.

In spite of these difficulties, the successful completion of many of the trials cited in this paper demonstrates the ability of people to adhere to a moderate sodium restricted diet. While ethnic and cultural preferences need to be considered, this need not be a barrier to treatment. Rose has described a successful community intervention trial in rural Portugal where the predominant local diet was high in sodium. (50)

Begin by evaluating the patient's diet recall for a minimum of three days assessing sodium content and the presence of prepared/processed foods. Ask the patient how often they eat out. Positive feedback may be provided by checking the random urine sodium which may demonstrate adherence to a low sodium diet. Although only useful as a relative number, random sodium provides feedback and can be a positive incentive for patients if they are adhering to their diet and see the numbers go down. (51) Simply modifying food purchases and preparation can achieve a reduction in sodium and an accompanying decrease in blood pressure. (52) In addition, patients should also be instructed in label reading, menu selection, and food preparation. It is helpful to let patients know that it takes approximately one month after reducing sodium for the taste buds to begin to recover and to appreciate flavors once again.

Sodium restriction pays dividends in a variety of ways. When patients become more aware of their sodium ingestion, they have a tendency to restrict their total caloric intake as well, thereby facilitating weight loss. Additionally, there is a tendency to eat less "fast" and/or "junk" food.

Decision making matrix for sodium reduction



Alcohol

Scientific rationale for reduction of alcohol

Alcohol consumption has been associated with elevations in blood pressure in a number of large population studies (53-56) and a reduction in alcohol consumption is associated with a reduction in blood pressure. (57,58) The relationship between alcohol consumption and elevations in blood pressure is more evident for systolic than diastolic blood pressure. (36) There are differences in response to alcohol. The strongest association between alcohol consumption and elevations in blood pressure have been shown in men, white people, and those over 55 years of age. (59) The mechanism by which alcohol appears to work is an acute pressor effect. (56) Each daily alcoholic drink increases the systolic blood pressure by 0.9 mmhg. (60)

In addition to elevations in blood pressure, excess alcohol intake may decrease adherence to therapy (1), reduce the effectiveness of antihypertensive medications (61), and occasionally increase the incidence of refractory hypertension. (1)

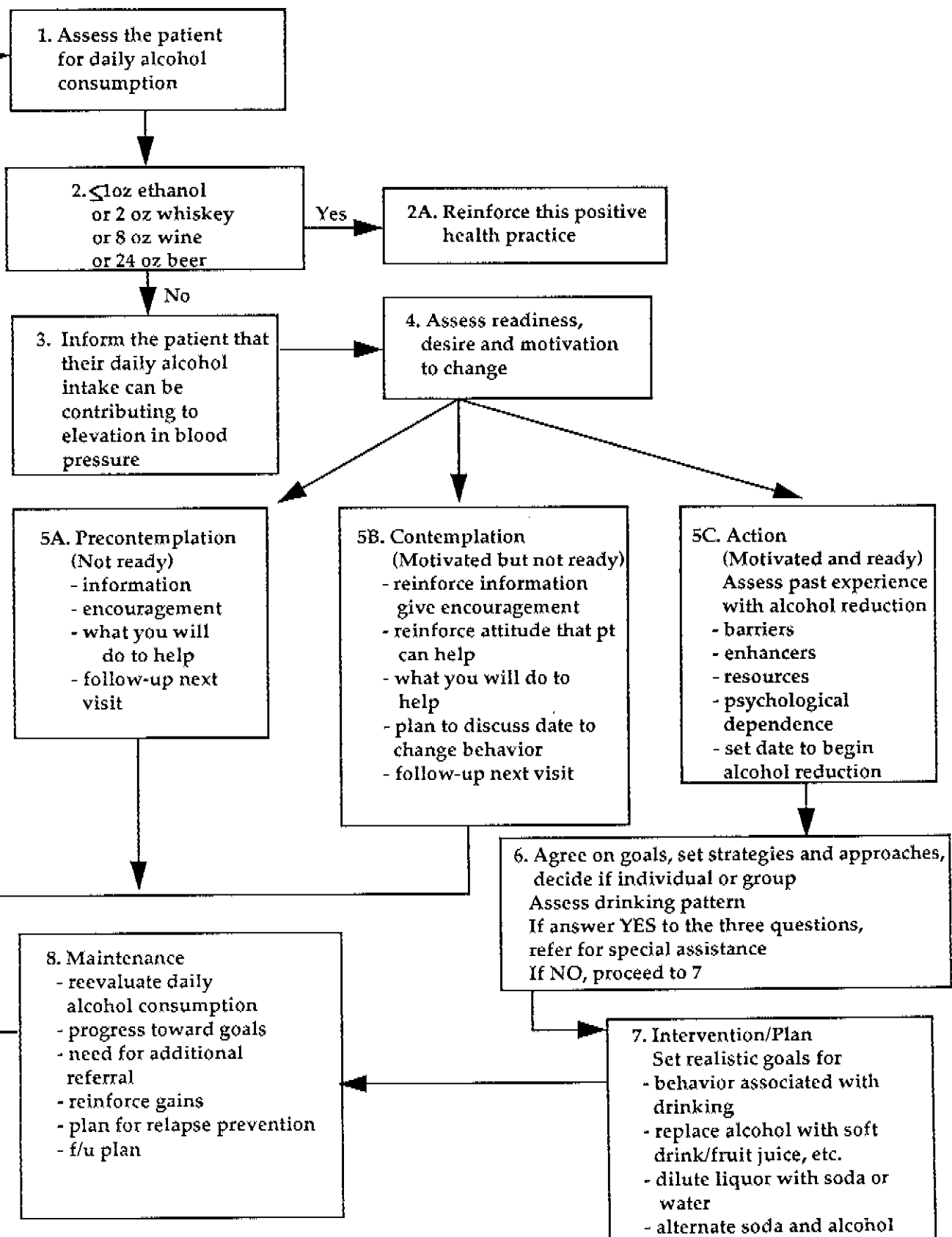
Behavioural strategies for reducing alcohol intake

Excess alcohol consumption is one of the most treatable causes of hypertension and modification of this behaviour should always be attempted. To lower blood pressure in those who consume

alcohol, limit their alcohol intake to one ounce (30 ml) of ethanol daily. One ounce (30 ml) of ethanol is two ounces (60 ml) of 100-proof whisky, eight ounces (240 ml) of wine or 24 ounces (720 ml) of beer. (1) Modest variations of these specific recommendations appear in other reports. (6,8,9) The clinician should address this issue with any patient who has hypertension and who consumes alcohol, because when made aware of the relationship between alcohol and elevated blood pressure, most patients who are not alcohol dependent can make appropriate adjustments. (62)

As was the case for weight loss and salt restriction, engineering reduced alcohol consumption requires sensitivity to individual patient needs. While most patients can easily adjust alcohol consumption to help control hypertension, there are those who cannot. Alcohol consumption is for many people an important aspect of social and family life. It is also, unfortunately, a coping mechanism for many. Patients dependent on alcohol may have difficulty making the necessary adjustments. To ascertain this, the following questions may be asked and have been shown to predict alcohol dependence: 1) Has your family ever objected to you drinking? 2) Did you ever think you drank too much? 3) Have others (friends, physicians, clergy) ever said you drank too much? (63) If the answers are affirmative, the individual should be referred for counseling.

Decision making matrix for alcohol reduction



Exercise

Scientific rationale for exercise

Although regular physical exercise is standard practice to lower blood pressure, controversy remains regarding its effectiveness as a singular therapy. In their most recent position statement, the Joint National Committee on Detection, Evaluation and Treatment of Hypertension concluded that "regular aerobic exercise (walking, bicycling, jogging or swimming) facilitates weight control and may be helpful in reducing blood pressure." (1) The World Health Organization (6) and the Canadian Consensus Panel (9) concluded that the effectiveness of exercise in lowering blood pressure has not been convincingly demonstrated, and stated that they could not issue any recommendation at this time.

Interest in exercise as a therapeutic modality to lower blood pressure has been longstanding. Several large cohort studies have suggested that sedentary individuals were at greater risk for developing hypertension (64), and that active individuals had lower blood pressures than their sedentary counterparts. (65-72) These studies are likely confounded by the fact that those who exercise and become fit may be more predisposed to manage their weight, eat a healthier diet which is low in sodium, and fat, consume less alcohol, and be less stressed. Many studies of both normotensive and hypertensive people have demonstrated a moderate, clinically significant reduction in systolic and diastolic blood pressure with regular aerobic exercise. (73) However the results have not been clearcut and the studies are flawed by methodologic issues. (74) These studies have demonstrated the positive effects of exercise on weight reduction, insulin resistance (75), blood lipids (76), and psychological stress (77,78).

The precise mechanism by which exercise may lower blood pressure is unclear. During exercise, systolic blood pressure rises, and diastolic blood pressure remains the same or rises slightly. In individuals with hypertension, the exercise response may be exaggerated, or be blunted secondary to antihypertensive medications. After aerobic exercise, vasodilatation persists short term and systolic blood pressure remains 25% lower than the preexercise blood pressure for approximately 90 minutes. (79) The mechanisms which may be responsible for the long term effects of exercise on lowering blood pressure are also not clearly identified, but several hypotheses have been offered: 1) decreased sympathetic nervous system activity, 2) increased levels of vasodilating prostaglandins, 3) decreased levels of plasma renin activity, 4) decreased plasma volume and

viscosity, and 5) decreased insulin resistance and decreased weight. (73)

In several large population studies, physical fitness has been associated with lower rates of cardiovascular disease, particularly of stroke and coronary artery disease. (80) This phenomenon has been observed in diverse socioeconomic groups and different parts of the world, including: middle-aged Finnish men (81), Belgian factory workers (82), United States railroad workers (83), and Harvard University graduates. (84) In view of the salubrious effect that exercise may have on weight, insulin resistance and blood lipids, it appears reasonable to include exercise as part of a comprehensive approach to treating hypertension.

Behavioural strategies for implementing an exercise programme

The American College of Sports Medicine Guidelines recommend a full symptom limited exercise tolerance test for anyone over 45 years of age, or for anyone over 35 years of age with a history of coronary artery disease, smoking, obesity, hypertension, hypercholesterolemia, or a family history, and who wishes to begin a new exercise programme. (85) Although the absolute risk is very low, individuals with coronary artery disease or associated risk factors are at risk for sudden death with strenuous physical exercise. (86) Preparing a hypertensive patient for regular exercise requires addressing psychological attitudes toward exercise which can undermine success. Many patients will suggest that they are "too busy" to exercise. Similarly, many patients will claim that their normal activities keep them so active that they get all the exercise they need. Patients should be encouraged to consider these objections in the context of their overall values and goals. As with all behavioural interventions, personal circumstances must be considered. There are some patients who will need to learn the difference between taking a two minute walk to the stock room 15 times a day and taking a 30 minute sustained walk. There are other patients who will indicate that they cannot exercise outside the home. The important points to emphasize are that physical fitness should be fun, should be personally relevant and also will pay great dividends in lower cardiovascular risk.

To avoid untoward cardiac events, as well as musculoskeletal injuries, patients should start slowly and be taught important self-monitoring techniques.

The following can serve as guidelines for prescribing exercise in both healthy and sedentary adults. (87)

Exercise prescription for healthy adults

Frequency	3 to 5 x per week
Intensity	60 to 90% max HR mod-heavy RPE
Time (Duration)	15 to 60 minutes (continuous)
Type (activity)	Run, jog, walk, cycle, swim or endurance sports

Exercise prescription for sedentary adults

Frequency	Start with sessions every other day Remain at 3 days per week or progress to 5 days per week
Intensity	60 to 70 percent of maximal heart rate or less Light to moderate RPE Gradually achieve target zone 70-85 % of maximal heart rate over weeks or months Moderate RPE
Time (Duration)	Start with 10-to 15-minute sessions Progress to 20- to 60-minute sessions
Frequency	Start with sessions every other day Remain at 3 days per week or progress to 5 days per week

Moderate exercise yields about the same blood pressure-lowering benefits as vigorous exercise and is preferred, even for the active, healthy adult. (89,89) Instruction in body awareness and rating of perceived exertion (RPE) (90) are useful in teaching patients to stay within their target zones, to monitor for symptoms and to adjust to changes in extrinsic demands. Yoga and other techniques which elicit the relaxation response are effective methods to teach body

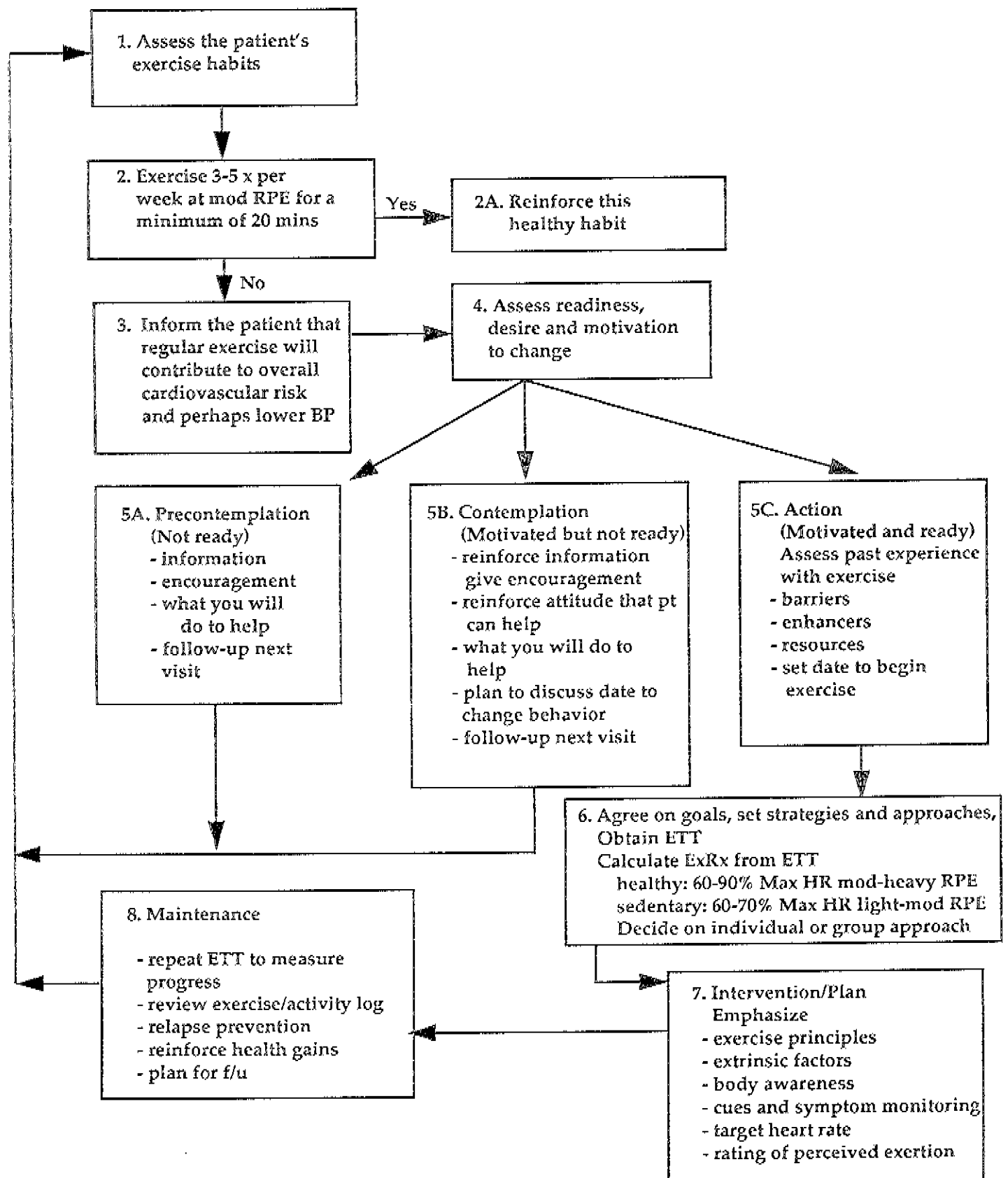
awareness. For patients who are on beta-blocking medication, rating of perceived exertion in fact may be the most effective way to measure exercise work load.

Patients should be encouraged to participate in the development of their prescription. Individual differences and preferences should be respected and initial level of fitness, including daily pattern of activity, general health and age should factor into the exercise prescription. Patients should identify the type of exercise they most prefer, keeping in mind what is most reasonable and feasible to maintain throughout the year. Patients should make every attempt to incorporate physical activity into their daily schedule in such a way that it becomes a pleasure as opposed to work.

Modern technology has largely eliminated the need for physical exertion at work and in our daily life. Fifty years ago, factories and farms were supplied by muscle power, today, however, few jobs require physical strength. The trade-off is that leisure has its price and we have noted the increased risk for cardiovascular disease over this time period. For this reason, it behooves us to look for ways in which to increase the amount of physical activity we engage in during the course of a day. Attention needs to be paid, however, to cultural and societal influences.

In the United States, convenience is highly valued while physical activity is devalued. In other cultures, this is not the case. Personal and family beliefs also should be considered. In some families, activity is valued whereas in other families, it is not. Patients who express a desire for structure or support tend to do better exercising with a group. Adherence and outcome improve with a significant other participants with the patient in their exercise programme. As was the case for sodium restriction, the institution of an exercise programme for the treatment of hypertension has multiple benefits. Individuals who begin regular exercise have a tendency to lose weight and consequently other cardiovascular risk factors are reduced. Another important asset is that the institution of a regular exercise programme can result in a reduction of psychological stress. Regular exercise can reduce negative psychological symptomatology thereby setting the stage for more effective behavioural change.

Decision making matrix for exercise



Eliciting the relaxation response and stress management

Scientific rationale for eliciting the relaxation response and stress management

Exposure to psychologically stressful situations which cause anxiety can result in an acute elevation in blood pressure. The relationship between emotional arousal and a transient rise in blood pressure is well established. (91) The presumed mechanism for emotional arousal is repeated elicitation of the "emergency response" or "fight or flight response." (92,93) First described by Walter B. Cannon, this response is characterized by arousal of the sympathetic nervous system (increased sympathetic nervous system activity) and manifested by increased oxygen consumption, blood pressure, heart rate, respiration rate and a 300 to 400 percent increase in skeletal muscle blood flow.

This relationship has led many clinicians and researchers to hypothesize that repeated or prolonged exposure to emotional arousal might result in chronic hypertension. (91) There is a substantial animal experimental literature (94) and human observational literature (95) which supports this hypothesized interaction between psychological stress and the pathogenesis of essential hypertension. In fact, many patients believe that psychological stress plays an important role in the etiology of hypertension. Not surprisingly, these etiologic relationships have prompted attempts to use relaxation-response therapy and other stress management approaches as intervention strategies for elevated blood pressure.

The relaxation response is the physiologic counterpart of the emergency response. (96) It is characterized by decreased oxygen consumption, blood pressure, heart and respiratory rates and a stabilization of muscle blood flow. (97) There are two common steps in techniques that elicit the relaxation-response: 1) the repetition of a word, sound, prayer or phrase and 2) the passive disregard of intruding thoughts and a return to the repetition. (98) Relaxation therapies include procedures such as progressive muscular relaxation, yoga, diaphragmatic breathing, mental focusing, meditation, repetitive prayer, biofeedback and autogenic training. All of these procedures may elicit the relaxation response when appropriately applied. (99)

In the discussion that follows, the terms relaxation therapy, relaxational therapies and relaxation training are all used for techniques that elicit the relaxation response. The suffix "which elicit the relaxation response" may be added as a suffix to these terms. The use of this suffix is to remind the reader that irrespective of

the specific procedure employed e.g. meditation, autogenic training, yoga, or biofeedback, the common presumptive mechanism associated with the lowered blood pressure from these relaxation therapies is elicitation of the relaxation response.

The understanding of this common response has further import in that every culture has its own techniques of eliciting the relaxation response. obviously, techniques can and should be chosen which respect the cultural as well as personal beliefs of the individual.

Stress management involves a wide variety of psychological and behavioural procedures including self-monitoring, time management techniques, cognitive restructuring, as well as elicitation of the relaxation response.

Following initial positive reports in 1974 on the effectiveness of yoga and other relaxation-response approaches in the treatment of hypertension (100), clinical studies proliferated with more than 60 outcome reports in the literature. (101) While the efficacy of these interventions is still debated, there are positive reports in the literature which strongly suggest that procedures which elicit the relaxation response can have a clinically significant effect on blood pressure and that they can be an effective adjunctive treatment when used in conjunction with pharmacotherapy. (102-105) More recently, some of these positive results have been criticized on methodological grounds. (101) Although a critical review is not possible here, an overview of the most current conceptualizations concerning the relevance of relaxation therapies and stress management for the treatment of hypertension can be presented.

There are several fundamental rationales for using relaxation techniques in hypertensive patients. These involve the presumed relationship between essential hypertension and arousal of the sympathetic nervous system. (106) If relaxation therapies and other stress management interventions are hypothesized to reduce sympathetic activity (107), then, it would be expected that psychological interventions would be most effective for patients with evidence of higher arousal of the sympathetic nervous system. There have been conflicting reports concerning this relationship. (108,109) An indirect test of the sympathetic arousal hypothesis can be made by examining the relationship between anxiety levels and response to treatment (110), because elevated sympathetic nervous system activity might be expected to be associated with higher anxiety. Whereas some studies report a positive association between anxiety and response to treatment (110,111) other studies report that lower levels of anxiety predicted better treatment response. (112,113)

Another rationale for the use of these therapies is more recent and involves the concept of the reactive or "white coat" hypertensive patient. There are apparently a relative large number of patients who carry the diagnosis of hypertension, but whose blood pressure is elevated only during clinical evaluation by a physician. (114) The use of ambulatory monitors has allowed the examination of this phenomenon and as many as 20% of all diagnosed hypertensive patients exhibit this effect. (115) Since these episodic and situation specific elevations are most likely due to anxiety associated with the medical evaluation, relaxation therapies and other psychological interventions obviously are relevant.

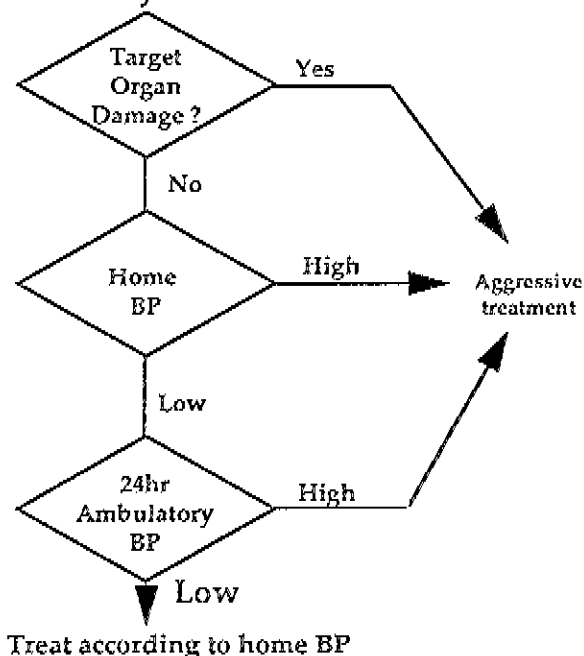
A recent comprehensive review of the literature indicated that at present, no psychological variable could discriminate responders to psychological treatment from non-responders. (116) Most researchers agree that exposing hypertensive patients to relaxation training and stress management will result in reduced blood pressures. However, the effects observed may be the result of research designs which fail to accurately account for blood pressure variability. (101) The positive results reported may be due to the fact that at the onset of most studies, patients are more aroused, stimulated and reactive to the measurement of blood pressure. Hence, pretreatment to post-treatment reductions would be expected to occur irrespective of the nature of the interventions (pharmacologic/nonpharmacologic). Indeed, there is a meaningful relationship between the length of pre-treatment evaluations and the magnitude of intervention-mediated blood pressure reductions. (101) Thus, relaxation therapies and stress-management therapies may act by achieving decreased sympathetic nervous system arousal. If this be the case, hypertensive patients should be exposed to those therapies before pharmacologic therapy is initiated or expanded.

In other words, the issue of blood pressure reactivity to the measurement procedure itself is becoming increasingly important. If the positive results of any clinical trials (pharmacologic or nonpharmacologic) are indeed the result of a progressive decrease in arousal as the patients proceed in the study, then it may be particularly important to acknowledge that for many patients, relaxation and stress management may be a very effective way to facilitate reduced arousal.

Clinicians must treat the blood pressures that they observe. Pickering (117) has offered a schema for evaluation of patients with elevations in blood pressure but who may be exhibiting spurious elevations due to exaggerated arousal. The schema is presented below:

Evaluation of "resistant" hypertension by ambulatory and home BP monitoring

Persistently elevated clinic BP



From Pickering TG: Hypertension 11 (Suppl II):11-96, 1988

According to this procedure, the patient is given the opportunity to demonstrate that the pressure is characteristically low under conditions of low arousal and that the high values which prompted clinical concern were due to the unusual circumstances associated with the clinic visit. Home monitoring doesn't necessarily eliminate anxiety and arousal nor is it often economically feasible. Even the use of ambulatory monitoring cannot completely overcome anxiety and arousal and is a very expensive assessment procedure for routine use. Therefore, reduced arousal during the clinic assessment becomes an important behavioural goal for many patients. White coat hypertensive patients not only have clinic-related arousal as an issue, but apparently, repeated clinic visits fail to result in a decrease. For this category of patients, relaxation training directly associated with clinic visits may be an important aspect of treatment. (115)

Despite the complexity and uncertain nature of the research involving psychological variables in the treatment of hypertension, the effect of psychological arousal on blood pressure must be considered if only to prevent misdiagnosis. To the extent that psychological arousal is a component of a patient's blood pressure level, relaxation therapy in particular

and stress management in general should be considered part of the comprehensive intervention package.

Still another rationale for the use of relaxation therapies and stress management in hypertension is the effects of these interventions on other nonpharmacologic treatment approaches. Nonpharmacologic interventions of any sort require behavioural change. Behavioural change is stressful. (118) Relaxation and stress management may very well facilitate the dietary and exercise changes discussed above (119), since regular use of relaxation therapy reduces negative psychological symptoms of anxiety, depression and hostility in hypertensive patients (105) and may make the prescribed anti-hypertensive behavioural adjustments easier. (119)

There is no consensus concerning the use of psychological interventions such as relaxation therapies and stress management in hypertensive patients. The Canadian Consensus Conference did not recommend that people participate in such interventions to prevent hypertension and considered it premature to recommend that hypertensive patients use them therapeutically. (9) The most recent JNC conclusion was equivocal. It recommended that "all patients be introduced to some type of psychological or behavioural therapy", but also indicated that "these promising methods have yet to be subjected to rigorous clinical trial evaluation and should not be considered as definitive treatment for patients with high blood pressure". However, despite this equivocation, there was a continued recommendation to use relaxation therapy and stress management for the treatment of mild hypertension and as adjunctive therapy for more severe hypertension. (1)

Neither of these reports specifically addressed the importance of these therapies as they relate to the issue of sympathetic arousal and white coat hypertension nor do these reports comment on the facilitating effect that these treatments may have on other nonpharmacologic interventions in reducing cardiovascular risk profile. These latter two concerns coupled with the suggestion of the JNC prompts us to recommend that relaxation therapies and stress management continue to be included in comprehensive treatment programmes.

Behavioural strategies for implementing relaxation therapy and stress management

No other intervention requires as much concern for individuality of patient needs as do the psychological therapies. To suggest that psychological interventions can importantly impact on physiology triggers many reactions.

Some patients will enthusiastically endorse the suggestion that stress may play some role in their hypertension and that relaxation therapy and stress management may help control it. Many patients will also readily acknowledge the role that stress plays in promoting smoking, overeating and lack of exercise. For these patients, relaxation and stress management can easily become an integral part of treatment.

For other patients, due to personal perspective or family and cultural influences, the suggestion that stress may be a consideration in hypertension may seem inappropriate, blaming, or even threatening. For these patients, the rationale for elicitation of the relaxation response and stress management must be presented in a practical, non-threatening manner. It should be recognized that for many patients the concept of psychophysiology is new. Many are comfortable with the distinction between physical illnesses such as hypertension and mental issues such as anxiety. To suggest that they influence each other may be seen as problematic by some and may be perceived as blaming by others. In our experience however, taking the time to explain the interaction between thoughts, feelings, emotions, behaviour and physiology is a crucial first step and usually pays worthwhile dividends.

For those with suspected "white coat hypertension", the stimuli associated with the measurement procedures cannot easily be eliminated. But repeated measurements over time is a standard strategy used to eliminate these anxiety-induced effects. Home blood pressure monitoring provides useful clinical information, and also allows the patient to feel a sense of partnership in their care while at the same time becoming less sensitive to the procedure. Ambulatory monitoring is another strategy which can be used to circumvent the white coat effect. Furthermore, physician referral for treatment to behavioural specialists is an option. In this case, the nonpharmacologic treatment is more focused on the relaxation response and desensitization to the measurement procedure itself and special attention can be given to the home blood pressures obtained by the patient.

Stress management and the relaxation response should be carefully introduced in a manner which acknowledges the biopsychosocial model and its application to the etiology of hypertension. Emphasis should be placed on establishing a biopsychosocial or psychophysiological link between stress, sympathetic arousal, increased anxiety and elevations in blood pressure. The relationship between stress and adverse lifestyle behaviours (stress-disinhibition) should be explained. After establishing these important links and once

patients are comfortable with the concept of relaxation training and stress management, a formal programme can be instituted.

Because stress causes both psychological distress and physiological arousal a phenomenon called the "stress-disinhibition" effect has been described. (120) Upon exposure to environmental demands which are perceived as stressful, previously inhibited behaviour patterns are likely to emerge. Hence, a patient's ability to exercise, elicit the relaxation response daily, restrict salt intake or reduce calories in general may be compromised by stress exposure. Relaxation therapies and stress management procedures directly address this issue and may therefore enhance the effectiveness of other behavioural interventions. This issue will be discussed in more detail when the concept of high risk situations is presented.

We recommend including four basic components in a formal programme. These are: 1) systematic self-monitoring of circumstances which are perceived as stressful; 2) a systematic attempt to alter the environmental demands so that stressful circumstances are less likely to occur; 3) a formal relaxation training programme in which the relaxation response can be elicited daily in order to reduce psychological and physiological arousal and; 4) exposure to cognitive restructuring exercises.

In addition to eliciting the relaxation response on a regular basis, it is also useful to instruct patients to utilize the relaxation response as an immediate stress reliever in specific situations. For example, when they have their blood pressure measured, encounter a traffic jam, or engage in a particularly difficult conversation they can stop, take a few deep breaths and thus bring forth the physiology of the relaxation response, mitigating some of the acute psychophysiological effects of stress.

The practicing clinician can accomplish a great deal in a short office visit. Begin by simply asking patients if they are stressed. Then ask them to rate the amount of stress in their life on a scale of 1 to 10. If they indicate that they are stressed, ask them to rate the amount of stress that they experience at home, work, socially, interpersonally and in regards to their health on a scale of 1 to 10. Ask them if they think that they are coping with the stress in their life. Research has shown that if the patient perceives themselves as being stressed, they are. Simply by asking about the amount of stress in their life, the clinician is educating the patient to the fact that stress can have an impact on a patient's health. The clinician is also creating the opportunity to allow the patient to discuss their concerns and to be empathetic. This in and of itself can be very helpful in treating stress. If the patient is stressed the clinician might then ask

the patient to identify the pattern of stress which they are experiencing. Is the stress they are experiencing acute—in response to a specific situation—or is it in a more pervasive pattern which is impacting on their daily life? This will provide the clinician with the information needed to determine whether they should work with the patient one-on-one or refer them to a formalized programme.

For those patients who are in the precontemplation and contemplation stage, it is important to acknowledge their stress and to establish the connection between stress and adverse psychophysiological effects. Engender a positive attitude so that the patient feels that they can be successful in altering their pattern of stress. In those patients who are ready to change, the clinician should choose an approach to managing stress which respects the individuals interests, beliefs and lifestyle. Patients who are concrete and who have difficulty acknowledging the psychophysiological component to stress may do best, initially, to approach stress management through a programme of exercise, reduction in caffeine, alcohol and cigarettes. Once they have begun, the clinician can then introduce the notion that these approaches are likely most effective when used in conjunction with regular elicitation of the relaxation response. Patients can then be instructed in a variety of techniques which can elicit the relaxation response. Basic instruction can be given in the office and the patient can be sent home with written instruction and a tape of relaxation-response exercises. Other patients will do best initially to begin with cognitive approaches and eliciting the relaxation response, then to address additional components including exercise and dietary modification.

Simple techniques which elicit the relaxation response such as progressive muscle relaxation, diaphragmatic breathing and meditation can be easily learned in an office visit. Techniques which may require more supervision and follow-up such as yoga or autogenic training may be best addressed in a group or structured format. It has been our experience with hypertensive patients that it is more effective to start by using concrete technique such as progressive muscle relaxation and diaphragmatic breathing although individual differences should be acknowledged.

The following are the basic prescriptive components for eliciting the relaxation response:

Relaxation response prescription

The Basics

- passive mental attitude to intruding thoughts
- repetition of a simple mental stimulus such as a word, sound, prayer, phrase or muscular activity

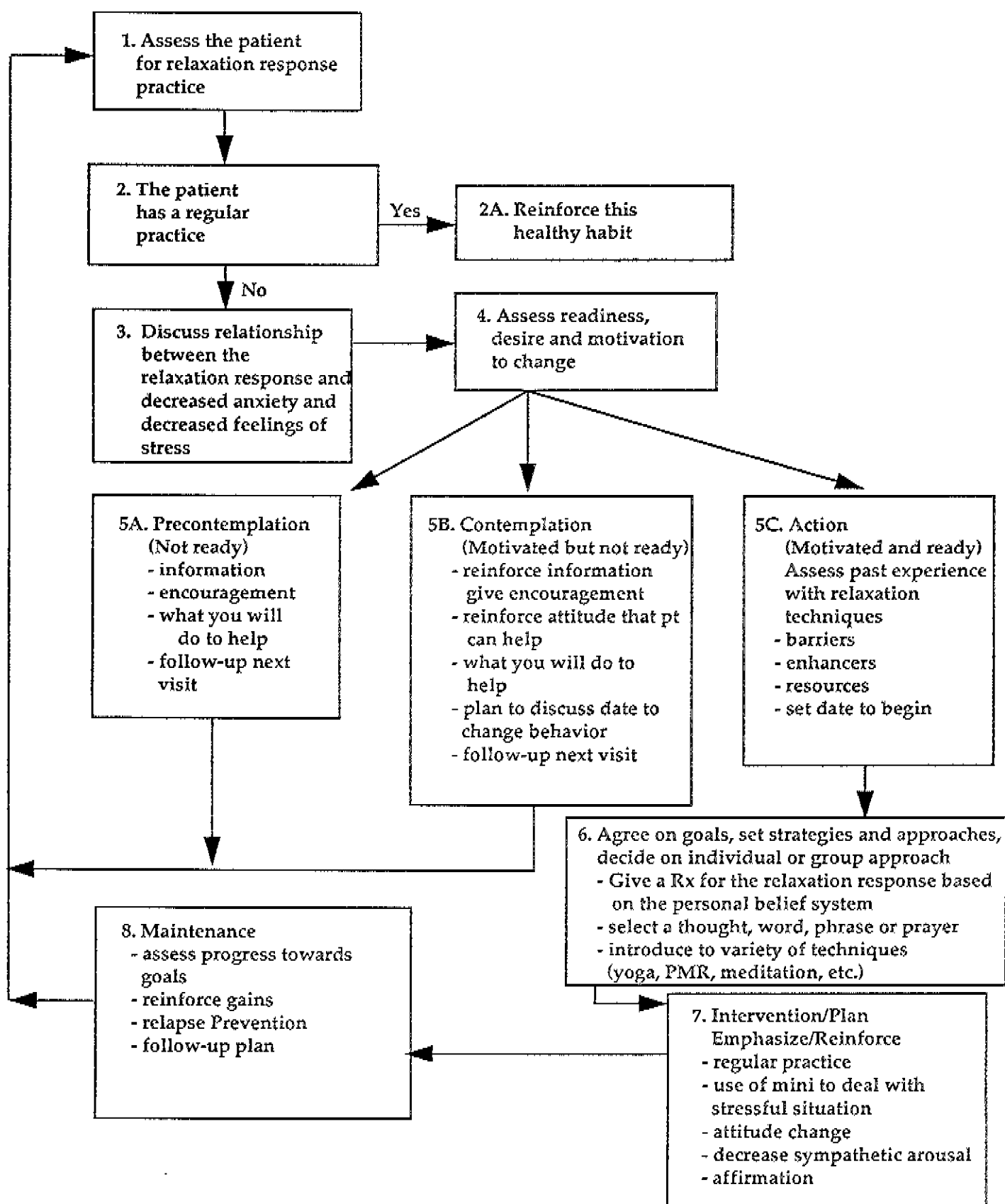
General prescription

- practice once a day for 20 minutes; alternative would be twice a day for 10 minutes each;
- do it in the same place every day;
- do it first thing in the morning;
- take the phone off the hook;
- tell the family, secretary, etc not to bother you;
- don't use the alarm clock; set a watch in front of you; alternatively, put a pillow over an alarm clock so it rings softly;
- don't do it right after a large meal;
- if you are uncomfortable, make yourself comfortable.

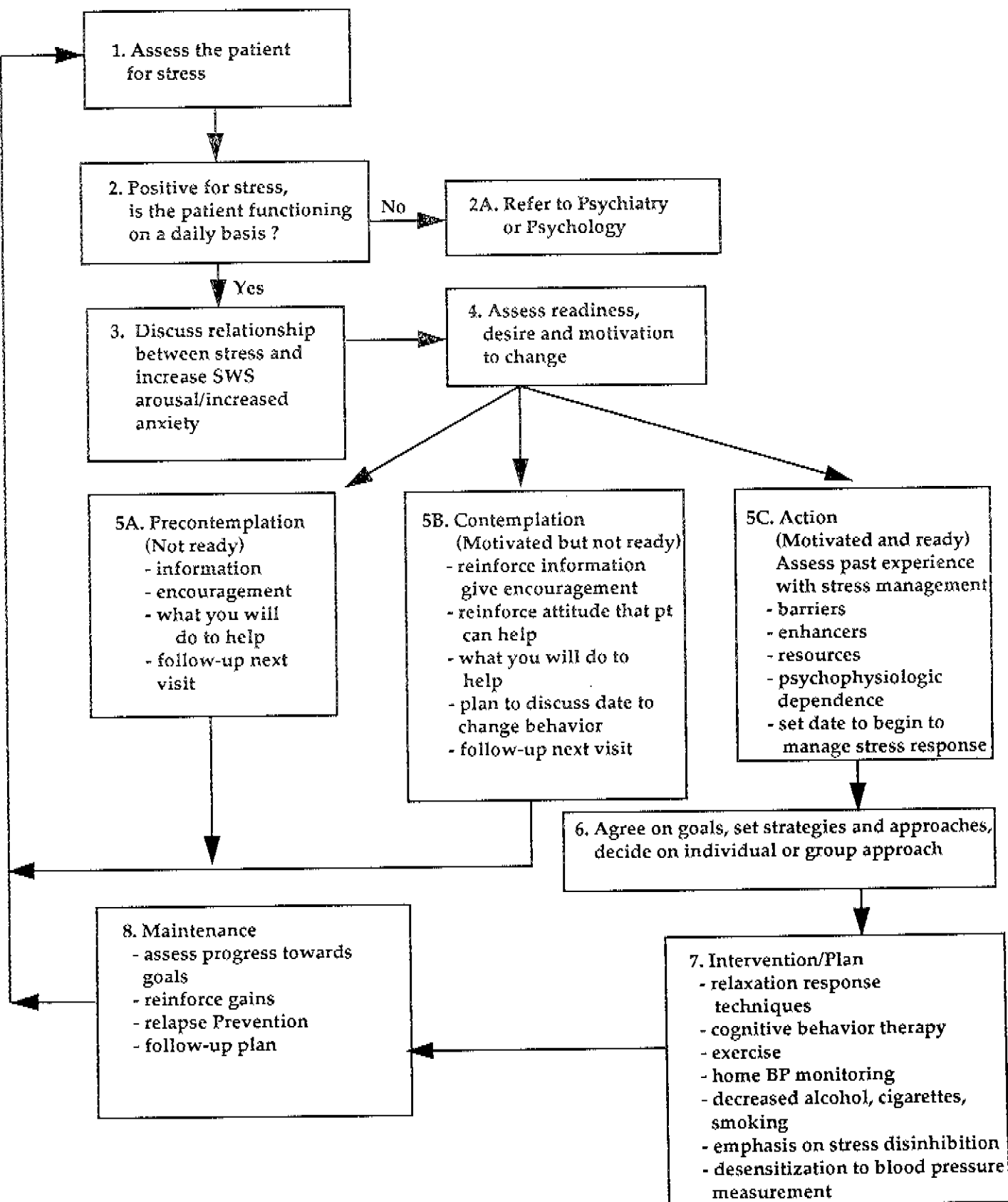
Cognitive behavioural approaches to stress management stem from the viewpoint that what we think influences our emotions, which in turn influence our physiology, and ultimately our health. Although it is difficult to carry out a systematic approach to cognitive restructuring in the confines of a short office visit, the clinician can endorse the principles of cognitive restructuring and encourage patients to participate either in a self-instructional programme or a group which addresses this therapeutic approach. Encourage patients throughout the process of making changes in stress management. Patients often want immediate success. If patients appear upset that

stress is still a problem, encourage them to think of stress management as a process rather than an immediate cure. Even if patients have not totally mastered the stresses in their life, encourage them in whatever changes that they have made. As previously indicated, these relaxation and stress management components are most effectively used when they are embedded within a comprehensive intervention programme addressing other cardiovascular risk factors and employing other nonpharmacologic interventions.

Decision making matrix for the relaxation response



Decision making matrix for managing stress



Tobacco

Scientific rationale

There have been no definitive studies indicating that cigarette smoking causes hypertension. Smoking does induce an acute pressor effect, but the body habituates to this effect so that chronic smoking is not associated with elevations in blood pressure or increased risk of developing hypertension. (121) Nonetheless, those who have hypertension and who smoke significantly increase their risk for coronary artery disease, myocardial infarction, sudden death and stroke. (122) In addition, individuals with hypertension who smoke have an increased incidence of malignant hypertension and subarachnoid bleeding. (123) Smoking also decreases the effectiveness of selected antihypertensive medications. (124)

As noted, the cardiovascular risks associated with hypertension coupled with smoking are high. An individual with hypertension who smokes has an increased risk of myocardial infarction and stroke three to five times greater than a hypertensive patient who does not smoke. This data led the Joint National Committee to state that, "the benefits of tobacco avoidance have been proven conclusively, and smoking cessation is strongly recommended. A key component of every therapeutic regime for hypertension should include counseling to help patients stop smoking". (1)

The direct relationship between smoking and blood pressure is less important than the relationship among multiple cardiovascular risk factors. The need to engineer smoking cessation as part of a nonpharmacological treatment programme in hypertension highlights the need to consider the patient from a multidimensional perspective. In addition to smoking being one of several risk factors, the behaviours associated with these risk factors are interrelated.

Behavioural strategies for smoking cessation

Smoking cessation approaches for hypertensive patients are no different than other smoking cessation programmes. In some cultures, there is increasing social pressure to stop smoking although commercial advertisements persistently encourage it. On the other hand, there are cultures in which smoking is considered quite appropriate. The approach recommended to help patients to stop smoking should be made with these cultural differences in mind. All patients who are currently smoking should be advised to stop. It is important for the clinician to express their concern and strong conviction that it will benefit the patient's overall health and prevent premature stroke and cardiovascular disease. United States Surgeon General C. Everett Koop (125) felt strongly enough to state that the single most important thing that one can do for their health is to stop smoking. The clinician is paramount in motivating the patient to stop smoking.

Once you have determined the patient's motivation, you can create the specific plan. If patients are not yet ready, it is important to give information, encourage patients to stop smoking and engender the belief that they can be successful at smoking cessation. It is also important to follow up on smoking status on each subsequent visit. For the patient who is motivated and ready to change, the first step is to take a complete smoking history. Within this history it is important to get an accurate picture of how addicted they are to nicotine as well to get a perspective on the biopsychosocial factors influencing their smoking pattern. Establish realistic expectations and a specific date to quit. Encourage the patient to complete a smoking self-assessment and begin to prepare for nicotine withdrawal. Nicotine is considered to be an addictive drug. It has recently been reported to be as addictive as either heroin and cocaine. (126) The physical symptoms of nicotine addiction subside in one to three weeks but psychosocial addiction lasts much longer. Patients should be prepared for the withdrawal symptoms including: cravings, anxiety, irritability, headache, drowsiness, restlessness, GI symptoms and difficulty concentrating. The following chart presents some useful interventions to deal with the symptoms of nicotine withdrawal. (127)

Symptom	Prescription	Symptom	Prescription
Anxiety irritability	Diaphragmatic breathing Relaxation techniques 6-8 hours sleep Decrease or eliminate stimulants Exercise (eg walking) Avoid unnecessary stress Nicotine gum Accept as normal Talk with support person	Constipation	Increase fluids High fiber diet
Headaches	Avoid caffeine	Fatigue	Maintain regular work / relaxation / sleep schedule
Sleep disturbance	Deep breathing / relaxation, especially before retiring Avoid unnecessary stress Regular exercise	Drowsiness	Aerobic exercise
Increased appetite	Increased water intake	Decreased concentration	Decrease or eliminate depressants Break from work and do light calisthenics Change activity Do not start new projects
Gnawing stomach	Low-calorie snack	Cough	Accept as natural part of nicotine withdrawal
Nausea	Noncalorie oral substitutes		

From McKool K. Facilitating Smoking Cessation. *Journal of Cardiovascular Nursing*. 1987; 1:36

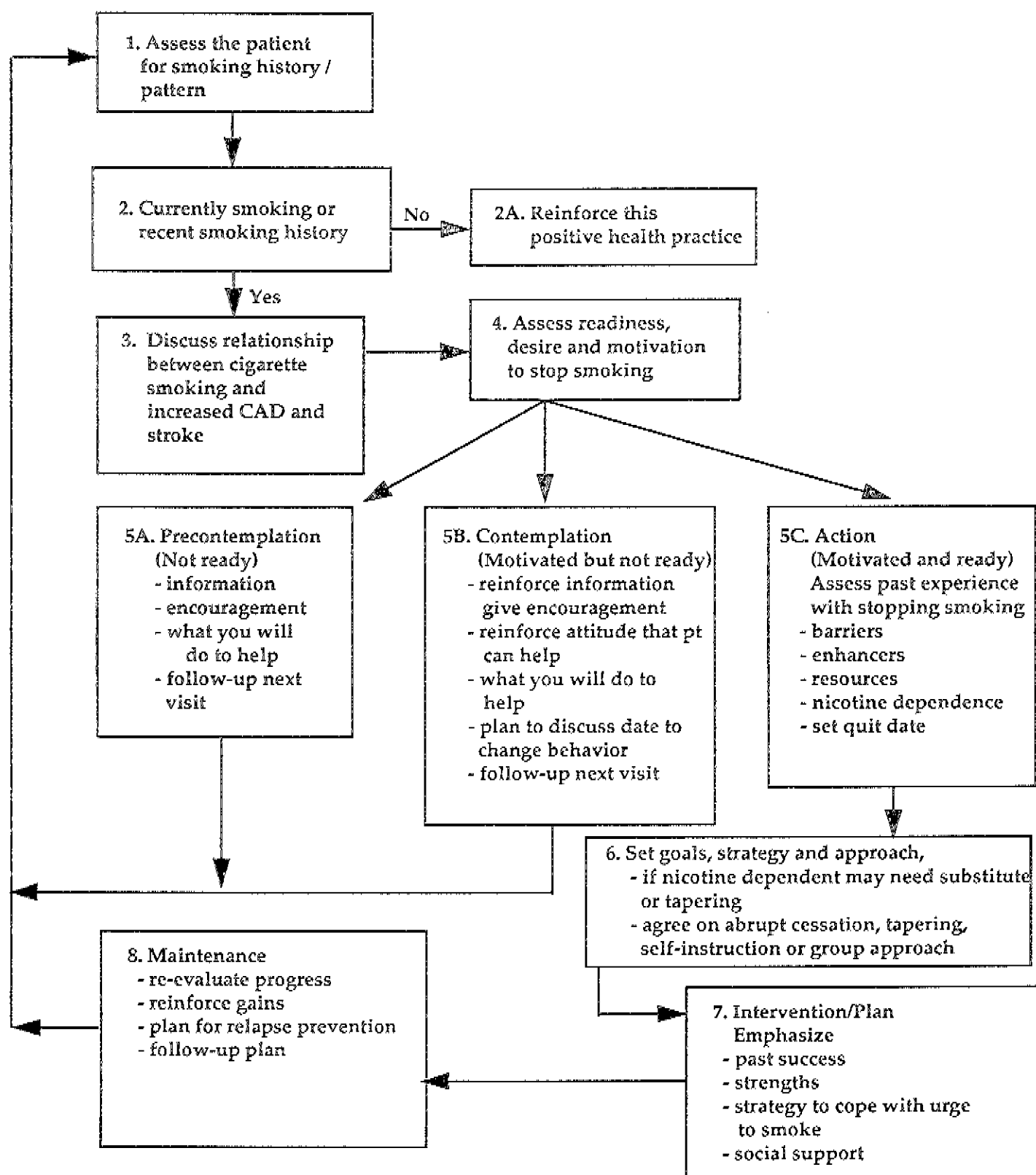
Nicotine chewing gum can be used as a short term weaning mechanism but should not be used as a long term substitute. The average smoker will chew six to ten pieces per day, begin to decrease this amount after two to three months and completely discontinue the chewing gum by six months. It is also useful to switch to a low tar, low nicotine brand of cigarette and/or to begin switching brands. Tapering the number of cigarettes smoked each day is another helpful weaning technique.

Patients should practice coping skills for high risk situations such as negative affect, sudden stress, boredom, eating/drinking, and

social situations. They need to have a variety of behaviour and cognitive coping skills, including distraction, delay, not giving in to the urge, deep breathing, escape, positive self-statements, cognitive restructuring, and recommitting to the benefits of quitting.

Exercise and eliciting the relaxation response have been found to be particularly helpful in dealing with the symptoms of nicotine withdrawal. Again, as is the case for weight loss, referral to a multi-factorial, nonpharmacologic treatment programme or a smoking cessation programme may be useful.

Decision making matrix for smoking cessation



Other dietary considerations

Potassium supplementation

Recently it has been suggested that the addition of potassium, calcium and magnesium to the diet of the hypertensive patient might be beneficial. There is a growing interest in potassium supplementation as a treatment modality for lowering blood pressure. (128) Several large population studies have suggested that dietary potassium is negatively associated with blood pressure (129,130) and that the sodium/potassium ratio is positively associated with elevated blood pressure. (131) Some studies have suggested that the 20th century diet of processed and fast food which is high in sodium and low in potassium is responsible for these findings. Diets high in potassium have been associated with a reduction in stroke-associated mortality with one study reporting a 40% reduction in stroke with the addition of 10 mmol/day of potassium. (132,133) A similar protective effect was noted in Japanese men living in Honolulu. (134) Increases in potassium intake (80mEq or 3-4 g/day) have been shown to lower systolic and diastolic blood pressure by 10 mmHg. (135) This inverse effect of potassium to blood pressure was noted to be greater in blacks. (135)

The mechanisms by which potassium may lower blood pressure and contribute to reduction in risk are not precisely understood. Several hypotheses have evolved including: potassium produces a natriuretic effect thereby decreasing extracellular volume and blood pressure (136), potassium alters the activity of the renin/angiotensin system, potassium relaxes smooth blood vessels thereby decreasing peripheral vascular resistance. There is some evidence that increased potassium may modify central and peripheral neural mechanisms that regulate blood pressure. (36)

Specific recommendations have been made by the Canadian Consensus Group (9) and the Joint National Committee (1) to increase potassium through simple dietary modification along with decreased dietary sodium intake. Caution should be exercised in patients with abnormal renal function or on potassium sparing diuretics. It has also been suggested that modification of the diet should concentrate on reducing the amount of processed and fast food since they tend to be high in sodium and low in potassium. Kaplan warns of the dangers inherent in using supplements to meet the recommendation to increase potassium. Although this may sound easier, he notes that they are costly and potentially hazardous and should be reserved for diuretic-induced hypokalemia. (10)

Calcium, Magnesium, Zinc, and Lead

The evidence regarding the blood pressure lowering effect of these cations or their impact on the overall cardiovascular risk profile is meager and mixed. The Joint National Committee (1), Canadian Consensus Panel (9), and World Health Organization (6) did not issue any recommendations regarding these cations.

Dietary fats

There is evidence in population studies that dietary fats influence blood pressure. Specifically, decreased consumption of fat and an increased polyunsaturated/saturated (P/S) ratio decreases blood pressure. (137) However the evidence from clinical trials is conflicting and controversial. Several mechanisms have been postulated to explain the effect of dietary fat on lowering blood pressure: an influence on renal excretory function, altered prostaglandin metabolism, and alterations in the composition of cell membranes. (138) The results of clinical studies, however, have not been definitive.

The general recommendation issued by the Joint National Committee (1), Canadian Consensus Report (9) and the World Health Organization (6) concludes that the evidence is inadequate to recommend decreased dietary intake of fat and increased P/S ratio to lower blood pressure. They did, however, agree that decreasing total dietary fat is important as a means to decrease cholesterol, decrease weight and impact favourably on the overall cardiovascular risk profile. The goal is to reduce cholesterol to less than 200 mg/dl, reduce LDL to less than 140 mg/dl, increase HDL to greater than 40 mg/dl and reduce the total cholesterol to HDL ratio in females to less than 3.5 and in males to less than 4.5.

Fish oil

Several population studies have shown an association between increased consumption of fish (particularly cold water fish) and decreased incidence of cardiovascular disease. Cold water fish are rich in omega-3 fatty acids, which have been widely reported to: decrease platelet aggregation, reduce vascular occlusive disease, and decrease blood pressure. The presumptive mechanism for these effects is the production of endogenous eicosanoids, mainly prostaglandins. (139)

It is the consensus (1,6,8,9) that the benefits of fish oil supplementation have not been clearly demonstrated, and that there was a significant risk of bleeding with these

supplements. It has therefore been recommended that individuals increase their consumption of fish (particularly oily fish), but not use fish oil supplements. (1,9)

Caffeine

Decreasing daily caffeine intake is popularly believed to reduce blood pressure. It has been demonstrated that ingestion of caffeine can have an acute pressor effect, elevating blood pressure five to 15 mmHg after two to three cups (250 mgm) of coffee. The blood pressure can then remain elevated for as long as two hours. However, chronic coffee ingestion of six cups/day (500 mgm) over four weeks was not found to be associated with sustained increased in blood pressure. (140) Additionally, there are no population studies that show a positive association with coffee consumption and prevalence of hypertension. (141)

Therefore reduction of caffeine is not specifically recommended to lower blood pressure or impact on the overall cardiovascular risk profile. (1,9)

Combined therapies

Primary or essential hypertension is not a unitary disorder. Therefore it is not surprising that there is uncertainty about the efficacy of each nonpharmacologic intervention. It may also explain why monotherapy may not be as effective as a combined, or multifactorial approach. Understanding the etiology and treatment of hypertension requires an appreciation of the complexity of the circulatory system with overlapping control mechanisms, intrinsic and extrinsic, and influenced by the central nervous system which is in turn influenced by the patient's perceptions and cognitions. In addition, the goal of hypertension treatment is twofold: first to lower blood pressure and secondly to favourably impact on the overall cardiovascular risk profile.

There have been studies which combine therapies in a multifactorial treatment approach even though these studies have methodological caveats. It has been demonstrated that patients could successfully lower blood pressure through a variety of nonpharmacologic interventions and combinations of interventions, and that they could successfully maintain these reductions in blood pressure over time. (142-144) Some of these same studies demonstrated a concomitant reduction in antihypertensive medications.

Two large recent trials have viewed the effectiveness of combining nonpharmacologic and pharmacologic therapies. In the Trial of

Antihypertensive Intervention and Management (TAIM), 878 patients were randomized to one of three antihypertensive drugs; chlorthalidone, atenolol, placebo and one of three antihypertensive diets; reduced calorie, reduced sodium/increased potassium, usual. After six months, those randomized to chlorthalidone or atenolol plus reduced calorie diet - and who had lost a mean of 10 pounds - had the greatest reduction in blood pressure. In addition, weight reduction was an independent predictor of pressure reduction. (145,146)

The Treatment of Mild Hypertension Study (TOMHS) studied 468 patients with mild hypertension. They were first prescribed a programme of weight reduction, sodium reduction, alcohol reduction, and therapeutic exercise, and were then placed on either placebo or one of five antihypertensive medications. After eighteen months, both the placebo plus nonpharmacologic group and the active drug plus nonpharmacologic group had achieved statistically and clinically significant reductions in blood pressure. The active drug plus nonpharmacologic group achieved the greatest reductions. (147)

The Canadian Consensus Panel (9), the Joint National Committee on Detection, Evaluation and Treatment of Hypertension (1), the World Health Organization (6), and the British Hypertension Working Party (8), have stated that hypertension is only one factor in the genesis of cardiovascular disease, and that it should be seen in the context of a multifaceted approach to the prevention and treatment of cardiovascular disease. Therefore, it is logical to approach treatment in this manner.

We recommend a comprehensive nonpharmacologic treatment programme as being more effective than unidimensional interventions for several reasons: 1) there are few precise diagnostic methodologies to identify specific nonpharmacologic interventions; 2) the goal is to both lower blood pressure and reduce overall cardiovascular risk; and 3) the interventions work synergistically.

The 13-session programme entitled the Cardiac Risk Reduction Clinic developed by the authors and their colleagues at the New England Deaconess Hospital, incorporates the multifactorial components of nonpharmacologic treatment for hypertension described throughout this paper.

Cardiac risk reduction clinic

The philosophy of this clinic is to provide a comprehensive, integrated, biopsychosocial approach to the care of the patient needing to modify adverse cardiovascular risk factors and

attain better control over their blood pressure. We emphasize that the clinic is interdisciplinary staffed by nurses, physicians, psychologists, nutritionists and exercise therapists. The specific aims of the Cardiac Risk Reduction Clinic are to:

- * reduce the cardiovascular risk profile, specifically to:
 - lower elevated blood pressure,
 - normalize serum lipids,
 - eliminate smoking,
 - reduce sodium, fat, and calories if appropriate,
 - reduce alcohol if appropriate,
 - increase exercise capacity,
 - reduce elevated stress levels
 - stabilize glucose tolerance
- * demystify care,
- * engender a positive attitude,
- * promote self-care,
- * empower patients to participate in their care, and
- * teach patients to elicit the relaxation response

Emphasis is placed on self-assessment, goal setting, selfmonitoring skills, self-care strategies and a variety of coping and relapse prevention skills. Each clinic session is designed to model and value the goals we have described. we incorporate therapeutic exercise, body awareness, affirmations, elicitation of the relaxation response and a discussion series into each session. Patients are asked to monitor and record their own symptoms and progress on diary cards and are encouraged to participate in their treatment planning.

All aspects of the curriculum - therapeutic exercise, body awareness, training in the elicitation of the relaxation response, affirmations, nutrition education and stress management are designed to emphasize:

- * Knowledge
 - that information necessary to understanding the biopsychosocial dimensions of interventions, the risks attendant in not treating hypertension and the essentials of self-care.
- * Skills
 - strategies for change
 - self-monitoring skills including:
 - symptom monitoring
 - home blood pressure measurement
 - home glucose monitoring
 - body awareness
 - rating of perceived exertion
 - stress warning signals
 - weight
 - heart rate monitoring

eliciting the relaxation response
therapeutic exercise
expanded coping styles
stress management
relapse prevention

* Attitude

utilizing cognitive restructuring and the relaxation response to promote an attitude conducive to mind/body health.

Family and/or significant others are encouraged to participate as a way of supporting the patient in making these changes.

Prior to entering the programme each patient is evaluated individually to confirm the diagnosis of hypertension, rule out secondary causes for the diagnosis, and ascertain the patients readiness and motivation for behavioural change. At this intake evaluation patients are invited to become participants in their care planning. At this time, the patient's medication use is discussed and a strategy for drug titration is considered. Throughout the programme, the weekly blood pressures of medicated patients should be evaluated. Consistent reductions can prompt reduced medication requirements. Since patients are frequently seen in the programme, this represents a particularly good time to evaluate medication use.

Following the intake evaluation, in preparation for the first clinic session patients;

- * view an instructional videotape on a simple technique to elicit the relaxation response
- * listen to an audiotape each day for 20 minutes which guides them in a simple breath focused technique to elicit the relaxation response
- * read Chapters 2, 4, and 5 in a manual entitled *The Wellness Book*. (148)

Session 1

Introduction to the clinic

- * staff introductions who we are, what we expect from them, what they can expect from us
- * patient introductions - who they are, what motivated them to come, what they expect from us
- * general information and expectations

Data collection

- * blood pressure, heart rate, weight, blood sugar

Goal setting lecture

- * With an emphasis on: values clarification, life meaning and purpose, long range goals, short term strategies, strengths/weaknesses, social support and monitoring parameters.

In preparation for Session 2

- * read Chapters 1 and 3 in The Wellness Book
- * complete the values clarification exercise
- * complete the self-portrait
- * begin goal sheet
- * purchase home blood pressure kit
- * complete daily diary

Session 2

Individual Prescription session

- * review of medical treatment
- * data collection
- * set specific goals and agree upon strategies and plan
- * exercise prescription
- * nutrition prescription
- * relaxation response/body awareness prescription
- * answer specific questions

In preparation for Session 3

- * read Chapters 7 and 22 in The Wellness Book
- * complete daily diary
- * purchase home blood pressure monitoring kit and bring to the next session

Session 3

Self-monitoring of blood pressure

Simple instruction in home blood pressure monitoring and recording.

Exercise lecture, with an emphasis on;

- * principles of therapeutic exercise
- * body awareness
- * cues for perceived exertion
- * self-monitoring of symptoms including:
 - angina
 - hyper/hypoglycemia
 - hyper/hypotension
 - musculoskeletal
 - snacks/insulin/exercise
- * healthy pleasures - exercise for fun

In preparation for Session 4

- * read chapters 8 and 9 in The Wellness Book
- * complete three day diet recall
- * complete daily diary

Session 4

The remaining sessions (4 through 13) follow the following format:

- data collection (10 minutes)
- warm-up (5 minutes)
- therapeutic exercise (30 minutes)
- cool-down/body awareness (10 minutes)
- relaxation response (15 minutes)
- discussion series (50 minutes) - topics change each week

Discussion topic

Heart Healthy Nutrition with an emphasis on low sodium;

- * 2,000 mg sodium food plan
- * label reading
- * food preparation
- * menu selection
- * behavioural factors

In preparation for session 5

- * read Chapter 23 in The Wellness Book
- * complete three day diet recall
- * complete daily diary

Session 5

Therapeutic exercise/relaxation response

Discussion topic

Heart healthy nutrition, reducing calories and cholesterol including:

- * food plan less than 20 - 30% of calories from fat
- * instruct in fat gram counting
- * label reading
- * food preparation
- * menu selection
- * behavioural factors/food choices

In preparation for Session 6

- * read Chapter 21 in The Wellness Book
- * complete risk factor checklist
- * complete daily diary

Session 6

Therapeutic exercise/relaxation response

Discussion topic

The pathophysiology of cardiovascular risk factors specifically:

hypertension, dyslipidemia, and diabetes

- * Biopsychosocial model for cardiovascular disease with emphasis on antecedent factors and synergy of factors.

In preparation for Session 7

- * review Chapter 6 in The Wellness Book
- * complete assessment for relaxation response
- * complete weekly diary
- * complete mid-programme evaluation of goals

Session 7

Discussion topic

Midway evaluation of progress toward goals

- * reassess goals
- * self measure of progress toward stated goals
- * reconfirm commitment
- * adjust goals as necessary

Hatha Yoga Exercises

- * Developing body awareness, muscle stretching, joint mobility and a method for aerobic cool-down

- * Eliciting the relaxation response

In preparation for Session 8

- * complete weekly diary
- * read Chapter 10 in The Wellness Book

Session 8

Discussion topic

Stress physiology; cardiovascular reactivity to physical and emotional stress.

- * The relationship of stress, sympathetic arousal, and elevations in blood pressure,
- * general psychophysiological reactions to stress
- * strategies to reduce stress - relaxation response, exercise, cognitive, and behavioural
- * the use of minis to alleviate acute stress

In preparation for Session 9

- * complete weekly diary
- * complete biodots exercise
- * read Chapters 11 and 12 in The Wellness Book
- * complete irrational beliefs inventory

Session 9

Therapeutic exercise/relaxation response

Discussion topic

Cognitive restructuring: automatic thoughts, irrational beliefs and distorted thinking styles.
In preparation for session 10

- * complete weekly diary
- * read chapters 13 and 14 in *The Wellness Book*
- * complete 'challenging stress and winning' worksheet

Session 10

Therapeutic exercise/relaxation response
Discussion topic
Coping and problem identification

In preparation for Session 11

- * complete weekly diary
- * complete anger expression scale

Session 11

Therapeutic exercise/relaxation response
Discussion topic
Coronary prone behaviour pattern: how thoughts, feelings and emotions affect cardiovascular arousal and health.

In preparation for Session 12

- * complete weekly diary
- * read Chapters 15 and 24
- * confirm follow-up appointment
- * make appointment for blood-work
- * complete post-programme evaluation of goals, questionnaires, and self-portrait
- * prepare a favourite heart healthy snack to bring to the 12th session

Session 12

Therapeutic exercise/relaxation response
Discussion topic
Humor as a coping tool and relapse prevention
Programme conclusion

- * general review of progress
- * programme evaluation form

- * collect post-programme evaluation of goals, questionnaires and self-portrait

- * confirm post programme lab work
- * confirm follow-up appointment
- * congratulations to one and all

In preparation for the follow-up visit;

- * complete programme evaluation
- * complete post-programme evaluation of goals, questionnaires and self-portrait
- * complete blood work at least three days prior to follow-up appointment

Follow-up session

One hour individual session

- * individual review of progress toward goals
- * medical update
- * sub-max exercise tolerance test (ETT) for measuring exercise conditioning and upgrading exercise prescription
- * Written plan for maintaining health gains and preventing relapse
- * medication consideration

References

1. The 1988 Joint National Committee. The 1988 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* (1988) 148:1023.
2. Kirkendall WM, Hammond JJ. Hypertension in the elderly. *Arch Intern Med* (1980) 140:1155.
3. Neaton JD, Kuller LH, Wentworth D, et al. Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years. *Am Heart J* (1984) 108(3 Pt 2):759.
4. Ribeiro AB, Ribeiro MBD. Epidemiological and demographic considerations: Hypertension in underdeveloped countries. *Drugs* (1986) 31(Suppl 4):23.

5. Akinkugbe OO. World epidemiology of hypertension in blacks. *J Clin Hypertens* (1987) 3:15.
6. Guidelines Sub-Committee of the WHO/ISH Mild Hypertension Liaison Committee. 1989 Guidelines for the Management of Mild Hypertension: Memorandum from a WHO/ISH Meeting. *J Hypertension* (1989) 7:689.
7. Gifford RW Jr, Kirkendall W, O'Connor DT, et al. Office evaluation of hypertension: A statement for health professionals by a writing group of The Council for High Blood Pressure Research. American Heart Association. *Circulation* (1989) 79:721.
8. British Medical Research Council Working Party. Stroke and coronary heart disease in mild hypertension: Risk factors and the value of treatment. *Br Med J Clin Res* (1988) 296:1565.
9. Chockalingam A, et al. Recommendations of the Canadian consensus conference on nonpharmacological approaches to the management of high blood pressure. *Can Med Assoc J* (1990) 142:1397.
10. Kaplan NM. *Clinical Hypertension*. 5th Ed. Baltimore: Williams & Wilkins, 1990:14.
11. Pickering G. Hypertension. Definitions, natural histories and consequences. *Am J Med* (1972) 52:570.
12. Rose G. Concepts in Hypertension. In: CJ Mathias, PS Cevr (Eds.) *Concepts in Hypertension*. 5th Ed. New York: Springer-Verlag, 1989:392.
13. Brett AS. Ethical issues in risk factor intervention. *Am J Med* (1984) 76:557.
14. Smith WM. Treatment of mild hypertension: Results of a ten-year intervention trial. *Circ Res* (1977) 40 (5 Suppl. 1) : 198.
15. Friedman R, Stuart E, Benson H. Non-pharmacologic Adjuncts to Therapy. In: SP Cooke, ED Frohlich (Eds.) *Current Management of Hypertension and Vascular Diseases*. Philadelphia: Mosby Yearbook. In Press.
16. Blackburn H. The primary prevention of high blood pressure—a population approach. *Ann Clin Res* (1984) 16(Suppl 43):9.
17. Benson H, Epstein MD. The placebo effect - a neglected asset in the care of patients. *JAMA* (1975) 232:1225.
18. Benson H, McCallie Jr DD. Angina pectoris and the placebo effect. *N Engl J Med* (1979) 300:1424.
19. Horowitz A, Viscoli C, Berkman L, et al. Treatment adherence and risk of death after myocardial infarction. *Lancet* (1990) 336:542.
20. Rudd P, Price MC, Graham LE, et al. Consequences of worksite hypertension screening. changes in absenteeism. *Hypertension* (1987) 10:425.
21. Johnston ME, Gibson ES, Terry CW, et al. Effects of labelling on income, work and social function among hypertensive employees. *J Chronic Dis* (1984) 37:417.
22. MacDonald LA, Sackett DL, Haynes RB, et al. Labelling in hypertension: A review of the behavioral and psychological consequences. *J Chronic Dis* (1984) 37:933.
23. US Dept of Health Education and Welfare. *Healthy people: The Surgeon General's Report on Health Promotion and Disease Prevention*. Washington, D.C.: US Government Printing Office, DHEW Pub. No. 7, 1979:79-79.
24. Tarlov AR. The rising supply of physicians and the pursuit of better health. *J Med Educ* (1988) 63:103.
25. Rosenstock IM, Becker NH. The social learning theory and health belief model. *Health Education Quarterly* (1988) 15:175.
26. Long JD. Relationship of locus of control to lifestyle habits. *J Clin Psychol* (1988) 44:209.
27. Bandura A. Self-efficacy: Toward a unifying theory of behavioral change. *Psych Rev* (1977) 84:191.
28. DiClemente CC, Prochaska JO. Self-change and therapy change of smoking behavior: A comparison of processes of change in cessation and maintenance. *Addict Behav* (1982) 7:133.
29. Orem DE. *Nursing: Concepts of Practice*. New York: McGrawHill, 1985.
30. 1988 Joint National Committee. The 1988 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* (1988) 148:1026.

31. Aristimuno CG, Foster TA, Voors AW, et al. Influence of persistent obesity in children on cardiovascular risk factors: The Bogalusa Heart Study. *Circulation* (1984) 69:895.
32. Staessen J, Fagard R, Lijnen P, et al. Body weight, sodium intake and blood pressure. *J Hypertension* (1989) 7 (Suppl 1):S19.
33. Kaplan NM. *Clinical Hypertension*. 5th Ed. Baltimore: Williams & Wilkins, 1990:165.
34. Eliahou HE, Ianina A, Gaon T, et al. Body weight reduction necessary to attain normotension in the overweight hypertensive patient. *Int J Obesity* (1981) 5:157.
35. Fagerberg B, Andersson OK, Isaksson B, et al. Blood pressure control during weight reduction in obese hypertensive men: Separate effects of sodium and energy restriction. *Br Med J Clin Res* (1984) 288:11.
36. Pietinen P, Aro A. The role of nutrition in the prevention and treatment of hypertension. *Adv Nut Res* (1990) 8:35.
37. Krieger DR, Landsberg L, Laraugh JH, Brenner BM, Kaplan NM, (Eds). *Perspectives in Hypertension, Vol. 2. Endocrine mechanisms in Hypertension*. New York: Raven Press, 1989:105.
38. Fagerberg B, Andersson OK, Persson B, et al. Reactivity to norepinephrine and effect of sodium on blood pressure during weight loss. *Hypertension* (1985) 7:586.
39. Rocchini AP, Katch V, Schork A, et al. Insulin and blood pressure during weight loss in obese adolescents. *Hypertension* (1987) 10:267.
40. Amatruda JM, Richeson JF, Welle SL, et al. The safety and efficacy of a controlled low-energy (very-low-calorie) diet in the treatment of non-insulin-dependent diabetes and obesity. *Arch Intern Med* (1988) 148:873.
41. Rocchini AP, Katch V, Anderson J, et al. Blood pressure in obese adolescents: Effect of weight loss. *Pediatrics* (1988) 82:16.
42. Leon AS. Weight Loss. In: *New Approaches to Cardiovascular Risk Management: A Mandate for the 90's. Highlights of the Saddlebrook Symposium: Reducing cardiovascular Risk Factors in the Hypertensive Patient*. Secaucus, NJ: Physicians World Communications, 1988:40.
43. Australian National Health and Medical Research Council Dietary Salt Study Management Committee. Fall in blood pressure with modest reduction in dietary salt intake in mild hypertension. *Lancet* (1989) 1:399.
44. Kaplan NM. *Clinical Hypertension*. 5th Ed. Baltimore: Williams & Wilkins, 1990:166.
45. Morgan T, Nowson C. The role of sodium restriction in the management of hypertension. *Can J Physiol Pharmacol* (1986) 64:786.
46. Dahl LK. Salt and hypertension. *Am J Clin Nutr* (1972) 25:231.
47. Atschul AM, Grommet JK. Food choices for lowering sodium intake. *Hypertension* (1982) 4(Suppl II):II116.
48. Shah BC, Belonje B. *Nutr Res* (1983) 3:629.
49. Sanchez-Castillo CP, Warrender S, Whitehead T, et al. A novel technique for assessing the sources of dietary sodium. *Ann Clin Res* (1984) 16(Suppl 43):44.
50. Rose G. Concepts in Hypertension. In: CJ Mathias, PS Cevr (Eds.) *Concepts in Hypertension*. New York: Springer-Verlag, 1989:392.
51. Kaplan NM, Simmons M, McPhee C, et al. *Arch Intern Med* (1982) 142:1638.
52. Ellison RC, Capper AL, Stephenson WP, et al. Effects on blood pressure of a decrease in sodium use in institutional food preparation: The Exeter-Andover Project. *J Clin Epidemiol* (1989) 42:201.
53. Grollman A. The action of alcohol, caffeine, and tobacco on cardiac output (and its related functions) of normal man. *J Pharmacol* (1930) 39:313.
54. Orlando J, Aronow WS, Cassidy J, et al. Effect of ethanol on angina pectoris. *Ann Intern Med* (1976) 84:652.
55. Kupari N. Acute cardiovascular effects of alcohol. *Br Heart J* (1983) 49:174.
56. Potter JF, Watson RD, Skan W, et al. The pressor and metabolic effects of alcohol in normotensive subjects. *Hypertension* (1986) 8:625.

57. Potter JE, Beavers DG. Pressor effect of alcohol in hypertension. *Lancet* (1984) 1:119.
58. Malhotra H, Mehta SR, Mathur D, et al. Pressor effects of alcohol in normotensive and hypertensive subjects. *Lancet* (1985) 2:584.
59. Klatsky AL, Friedman GD, Siegelaub AB, et al. Alcohol consumption and blood pressure: Kaiser-Permanente Multi-Phase Health Examination Data. *N Engl J Med* (1977) 296:1194.
60. Elliott P, Fehily AM, Sweetnam PM, et al. Diet, alcohol, body mass, and social factors in relation to blood pressure: The Caerphilly Heart Study. *J Epidemiol Community Health* (1987) 41:37.
61. Puddy IB, Beilin LB, VanDongen R. Regular alcohol use raises blood pressure in treating hypertensive subjects: A randomized control trial. *Lancet* (1987) 1:647.
62. Wallace P, Cutler S, Haines A. Randomised controlled trial of general practitioner intervention in patients with excessive alcohol consumption. *Br Med J* (1988) 297:663.
63. Gallant DM. *Alcoholism: A Guide to Diagnosis, Intervention and Treatment*. New York: W.W. Norton and Company
64. Paffenbarger RS, Thorne NC, Wing AL. Chronic disease in former college students. VIII. Characteristics in youth predisposing to hypertension in later years. *Am J Epidemiol* (1968) 88:25.
65. Paffenbarger RS, Thorne NC, Wing AL. Chronic disease in former college students. VIII. Characteristics in youth predisposing to hypertension in later years. *Am J Epidemiol* (1968) 88:25.
66. Blair SN, Goodyear NN, Gibbons LW, et al. Physical fitness and incidence of hypertension in healthy normotensive men and women. *JAMA* (1984) 252:487.
67. Anderson KL, Elvik A. The resting arterial blood pressure in athletes. *Acta Med Scand* (1956) 153:367.
68. Hickey N, Mulcahy R, Bourke CJ, et al. Study of coronary risk factors related to physical activity in 15,171 men. *Br Med J* (1975) 3:507.
69. Leren P, Askevold EM, Foss OP, et al. The Oslo Study: Cardiovascular disease in middle-aged and young Oslo men. *Acta Med Scand* (1976) 558 (Suppl):1.
70. Cooper KH, Pollock ML, Martin RP, et al. Physical fitness levels vs. selected coronary risk factors: A cross-sectional study. *JAMA* (1976) 236:166.
71. Criqui MH, Mebane I, Wallace RB, et al. Multivariate correlates of adult blood pressures in nine North American populations: The Lipid Research Clinics Prevalence Study. *Prev med* (1982) 11:391.
72. Cederholm J, Wibell L. The relationships of blood pressure to blood glucose and physical leisure time activity. *Acta Med Scand* (1986) 219:37.
73. Siegel WC, Blumenthal JA. The role of exercise in the prevention and treatment of hypertension. *Ann Behav Med* (1991) 13:23.
74. Blumenthal JA, Siegel WC. Does exercise reduce blood pressure in patients with mild hypertension? Results of a randomized controlled trial. *JAMA* (1991) 266(15):2098.
75. Rodnick KJ, Haskell WL, Swislocki ALM, et al. Improved insulin action in muscle, liver, and adipose tissue in physically trained human subjects. *Am J Physiol* (1987) 253:489.
76. Hespel P, Lijnen P, Fagard R, et al. Changes in erythrocyte sodium and plasma lipids associated with physical training. *J Hypertension* (1988) 6:159.
77. Blumenthal JA, Williams RS, Needles TL, et al. Psychological changes accompanying aerobic exercise in healthy middle-aged adults. *Psychosom Med* (1982) 44:529.
78. Goff D, Dimsdale JE. The psychological effects of exercise. *J Cardio-Pulmonary Rehabil* (1985) 5:234.
79. Bennett T, Wilcox RG, MacDonald IA. Post-exercise reduction of blood pressure in hypertensive men is not due to acute impairment of baroreflex function. *Clin Sci* (1984) 67:97.
80. Kannel WB, Wilson P, Blair SN, et al. Epidemiological assessment of the role of physical activity and fitness in development of cardiovascular disease. *Am Heart J* (1985) 109:876.

81. Pekkanen J, Marti B, Nissinen A, et al. Reduction of premature mortality by high physical activity: A 20-year follow-up of middle-aged Finnish men. *Lancet* (1987) 1:1473.
82. Sobolski J, Kornitzer M, DeBacker G, et al. Protection against ischemic heart disease in the Belgian Physical Fitness Study: Physical fitness rather than physical activity. *Am J Epidemiol* (1987) 125:601.
83. Slattery ML, Jacobs JRDR. Physical fitness and cardiovascular disease mortality. The U.S. Railroad Study. *Am J Epidemiol* (1988) 127:571.
84. Paffenbarger, Jr. RS, Hyde RT, Wing AL, et al. Physical activity, all-cause mortality, and longevity of college alumni. *N Engl J Med* (1986) 314:605.
85. American College of Sports Medicine. **Guidelines for Exercise Testing and Prescription**. 4th Ed. Philadelphia: Lea and Febiger, 1991.
86. Thompson PO, Funk EJ, Carleton RA, et al. Incidence of death during jogging in Rhode Island from 1975 through 1980. *JAMA* (1982) 247:2535.
87. Putter J. Increasing Physical Activity and Exercise. In: *New Approaches to Cardiovascular Risk Management: A Mandate for the 90s. Highlights of the Saddlebrook Symposium. Reducing Cardiovascular Risk Factors in the Hypertensive Patient*. Secaucus, NJ: Physicians World Communications, 1988:22.
88. Pollock MC. How much exercise is enough. *Phys Sports Med* (1978) 6:50.
89. Leon AS, Blackburn H. Physical Inactivity. In: NM Kaplan, in Stamler (Eds.) *Prevention of Coronary Heart Disease*. New York: B.Saunders Co., 1983:86-97.
90. Borg G. Perceived exertion: A note on history and methods. *Med Sci Sports Med* (1973) 5:90.
91. Shapiro AP. Psychological and Social Factors in Hypertension. In: J Genest, O Kuchel, P Hamet, M Canton (Eds.) *Hypertension*. New York, NY: McGraw-Hill, 1983:765-776.
92. Cannon WB. The emergency function of the adrenal medulla in pain and the major emotions. *Am J Physiol* (1914) 33:356.
93. Cannon WB. *Bodily Changes in Hunger, Fear and Rage*. New York: Appleton, 1929.
94. Friedman R, Iwai J. Genetic predisposition and stress-induced hypertension. *Science* (1977) 193:161.
95. Cobb S, Rose RM. Hypertension, peptic ulcer, and diabetes in air traffic controllers. *JAMA* (1973) 224:489.
96. Benson H. *The Relaxation Response*. New York: William Morrow, 1975.
97. Wallace RK, Benson H, Wilson AF. A wakeful hypometabolic physiologic state. *Am J Physiol* (1971) 221:795.
98. Hoffman JW, Benson H, Ams PA, et al. Reduced sympathetic nervous system reactivity associated with the relaxation response. *Science* (1982) 215:190.
99. Benson H. The relaxation response: Its subjective and objective historical precedents and physiology. *TINS* (1983) 6:281.
100. Patel CH. Yoga and biofeedback and the management of hypertension. *Lancet* (1973) 2:1053.
101. Jacob RG, Chesney MA, Williams DM, et al. Relaxation therapy for hypertension: Design effects and treatment effects. *Ann Behav Med* (1991) 13:5.
102. Patel CH, Marmot MC, Terry DJ. Controlled trial of biofeedback-aided behavioral methods in reducing mild hypertension. *Br Med J* (1981) 282:2005.
103. Patel C, Marmot MC, Terry W, et al. Trial of relaxation in reducing coronary risk: Four year follow-up. *Br Med J* (1985) 290:1103.
104. Engel BT, Glasgow MS, Gaarder KR. Behavioral treatment of high blood pressure: III. Follow-up results and treatment recommendations. *Psychosom Med* (1983) 45:23.
105. Stuart E, Caudill MA, Leserman J, et al. Nonpharmacologic treatment of hypertension: A multiple-risk-factor approach. *J Cardiovasc Nurs* (1987) 1:1.
106. Julius S, Esler M. Autonomic nervous cardiovascular regulation in borderline hypertension: Symposium on hypertension. *Am J Cardiol* (1975) 36:685.

107. Caudill MA, Friedman R, Benson H. Relaxation Therapy in the Control of Blood Pressure. In: MD Blafox, HG Langford (Eds.) **Non-Pharmacologic Therapy of Hypertension**. New York: Karger, 1987:106-119.
108. Cottiar C, Shapiro K, Julius S. Treatment of mild hypertension with progressive muscle relaxation: Predictive value of indexes of sympathetic tone. *Arch Intern Med* (1984) 144:1954.
109. McGready AV, Higgins Jr JT. Prediction of response to bio-feedback-assisted relaxation in hypertensives: Development of a hypertensive predictor profile (HYPP). *Psychosom Med* (1989) 51:277.
110. McGready AV, Utz SW, Woerner M, et al. Predictors of success in hypertension treated with bio-feedback-assisted relaxation. *Biofeedback Self Regul* (1986) 11:95.
111. Wadden TA. Predicting treatment response to relaxation therapy for essential hypertension. *J Nerv Ment Dis* (1983) 171:683.
112. Crowther JH. Stress management training and relaxation imagery in the treatment of essential hypertension. *J Behav Med* (1983) 6:169.
113. Egin KS, Kogan NH, Garber A, et al. The impact of psychological distress on the control of hypertension. *J Human Stress* (1983) 9:4.
114. Mancina G, Parati G, Grassi G, Pomidossi, G. *J Hypertension* (1987) 5(Suppl 5):S591.
115. Pickering TG, Friedman R. The White Coat Effect: A Neglected Role for Behavioral Factors in Hypertension. In: PM McCabe, N Schneiderman, JM Field, JS Skyler (Eds.) **Stress, Coping, and Disease**. Hillsdale, NJ: Erlbaum, 1991:35-49.
116. Blanchard EB, McCoy GC, Berger M, et al. A controlled comparison of thermal biofeedback and relaxation training in the treatment of essential hypertension. IV: Prediction of short term clinical outcome. *Behav Ther* (1989) 20:405.
117. Pickering TG, James GD, Boddie C, et al. How common is white coat hypertension. *JAMA* (1988) 259:225.
118. Lazarus RS, Folkman S. **Stress, Appraisal, and Coping**. New York: Springer, 1984.
119. Taylor S. **Health Psychology**. New York: Random House, 1986.
120. Marlatt CA, Gordon JR. Determinants of Relapse: Implications for the Maintenance of Behavior Change. In: PO Davidson, SN Davidson (Eds.) **Behavioral Medicine: Changing Health Lifestyles**. New York: Brunner/Mazel, 1980:410-452.
121. Ballantyne D, Devine BL, Fife R. Interrelation of age, obesity, cigarette smoking, and blood pressure in hypertensive patients. *Br Med J* (1978) 1:880.
122. Kannel WB, Higgins M. Smoking and hypertension as predictors of cardiovascular risk in population studies. *J Hypertens Suppl* (1990) 8(5):S3-8.
123. Isles C, Brown JJ, Cumming AMM. Excess smoking in malignant-phase hypertension. *Br Med J Clin Res* (1979) 1:579.
124. Greenberg G, Thompson SC, Brennan PJ. The relationship between smoking and the response to anti-hypertensive treatment in mild hypertensives in the Medical Research Council's trial of treatment. *Int J Epidemiol* (1987) 16:25.
125. US Department of Health and Human Services. **The Health Consequences of Smoking: Cardiovascular Disease**. : Public Health Service, Office on Smoking and Health, 1983.
126. Ockene JK. Smoking Cessation. In: **New Approaches to Cardiovascular Risk Management: A Mandate for the 1990s**. Highlights of the Baddiebrook Symposium: Reducing Cardiovascular Risk Factors in the Hypertensive Patient. Secaucus, NJ: Physicians World Communications, 1988:15.
127. McKool K. Facilitating smoking cessation. *JCN* (1987) 1:20.
128. Smith SJ, Markandu ND, Sagnella GA, et al. Moderate potassium chloride supplementation in essential hypertension: Is it additive to moderate sodium restriction?. *Br Med J* (1985) 290:110.
129. INTERSALT Cooperative Research Group 1988. INTERSALT: An international study of electrolyte excretion and blood pressure: Results of 24-hour urinary sodium and potassium excretion. *Br Med J* 297:319.
130. Harlan WR, Harlan LC. An epidemiological perspective on dietary electrolytes and hypertension. *J Hypertension* (1986) 4 (Suppl 5):S334.

131. Pietinen P, Aro A. The role of nutrition in the prevention and treatment of hypertension. *Adv Nut Res* (1990) 8:35.
132. Khaw KT, Barrett-Connor E. Dietary potassium in stroke-associated mortality. *N Engl J Med* (1987) 316:235.
133. Lee CN, Reed DM, MacLean CJ, et al. Dietary potassium and stroke (letter). *N Engl J Med* (1988) 318:995.
134. Kihara M, Fujikawa J, Ohtaka M, et al. Inter-relationships between blood pressure, sodium, potassium, serum cholesterol, and protein intake in Japanese. *Hypertension* (1984) 6:736.
135. Svetkey LP, Yarger WE, Feussner JR, et al. Double-blind, placebo-controlled trial of potassium chloride in the treatment of mild hypertension. *Hypertension* (1987) 9:444.
136. Fujita T, Sato Y. Natriuretic and antihypertensive effects of potassium in DOCA-salt hypertensive rats. *Kidney Int* (1983) 24:731.
137. Sacks FM, Rouse JL, Stampfer NJ, et al. Effect of dietary fat and carbohydrate on blood pressure of mildly hypertensive patients. *Hypertension* (1987) 10:452.
138. Heagerty AM, Ollerenshaw JD, Robertson DL, et al. Influence of dietary linoleic acid on leucocyte sodium transport and blood pressure. *Br Med J* (1986) 293:295.
139. Kaplan NM. *Clinical Hypertension*. 5th Ed. Baltimore: Williams & Wilkins, 1990:173.
140. Robertson D, Hollister AS, Kincaid D, et al. Caffeine and hypertension. *Am J Med* (1984) 77:54.
141. van Dusseldorp M, Smits P, Thien T, et al. Effect of decaffeinated versus regular coffee on blood pressure. A 12-week, double-blind trial. *Hypertension* (1989) 14:563.
142. Dodson PM, Pacy PJ, Cox EV. Long-term follow-up of the treatment of essential hypertension with a high-fiber, low-fat and low-sodium dietary regimen. *Human Nutr-Clin Nutr* (1985) 39:213.
143. Vertes V, Frolkis JP, Martin PJ. Clinical utility of nondrug therapy for hypertension. *Mt Sinai J Med* (1988) 55:296.
144. Leserman J, Stuart E, Mamish M, et al. Non-pharmacologic intervention for hypertension: Long-term follow-up. *J Cardio-Pulmonary Rehabil* (1989) 9:316.
145. Blafox MD, Oberman H, Langford H, et al. *Am J Hypertens* (1989) 2:42A.(Abstract)
146. Smoller SW, Blafox MD, David B. *Am J Hypertens* (1989) 2:16A.(Abstract)
147. Grimm R, Neaton J, Almer P, et al. *Circulation* (1989) 80 (Suppl II):II-302.(Abstract)
148. Mind-Body Medical Institute Staff et al. *The Wellness Book: A Comprehensive Guide to Maintaining Health and Treating Stress Related Illness*. (1991) Birch Lane Press. New York.