Nutritional Factors in the Control of Blood Pressure and Hypertension

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▪ ABSTRACT

Differing hypertension prevalence rates between certain population and age groups are partially due to differences in the intake of certain nutrients. Blood pressure is positively associated with higher sodium, alcohol, and protein intakes; it is inversely associated with potassium, calcium, and magnesium intakes. Salt may lead to an increase in blood pressure in the presence of salt sensitivity, but there is no inexpensive or easy strategy to identify salt-sensitive patients. Other risk factors for hypertension include obesity and lack of regular physical activity.

The best strategy appears to be moderate salt restriction (6–7 g/day) in *combination* with an optimal compliance of the antihypertensive drug therapy, as well as adoption of the combination diet of the DASH study—a diet rich in fruits and vegetables, and thus rich in potassium. Current evidence does not support the increased intake of Ca^{2+} or Mg^{2+} for blood-pressure-lowering purposes only; however, calcium and magnesium may represent important components in the combination diet of the DASH study. It seems that it is the combination of these nutrients that is of crucial importance for the achievement of optimal blood-pressure reduction. Also recommended is a decrease in alcohol consumption and an increase in regular physical activity. Instead of a severe intervention with regard to 1 risk factor alone, positive changes in 5 habits *combined*—high salt intake, high sodium-to-potassium ratio, alcohol intake, calorie imbalance, and a sedentary life—may be the most realistic and effective strategy to counteract the present hypertension epidemic. *Nutr Clin Care.* 2002;5:9–19 ▪

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Cardiovascular mortality remains the major cause of death in the United States. Elevated blood pressure represents one of the major risk factors for atherosclerosis. Currently, about 23% of the U.S. population has elevated blood pressure, and 64% of men aged > 75 years can be classified as hypertensive.[1] Hypertension is most prevalent among African-Americans, with African-American, non-Hispanic men having a prevalence of 35%. Different prevalence rates of hypertension between certain population and age groups may be due to a variety of modifiable and nonmodifiable factors, including differences in the intake of certain nutrients.[2] Hajjar et al[2] analyzed data from the Third National Health and Nutrition Examination Survey (NHANES III) and found that systolic blood pressure was positively associated with higher sodium, alcohol, and protein intakes and inversely associated with potassium intake. Diastolic blood pressure was negatively associated with potassium and alcohol intakes. The age-related increase in blood pressure was associated with calcium intake.[2]

According to present guidelines, all hypertensive patients should be treated—first by nonpharmacological means and, if necessary, with pharmacological antihypertensive therapy.[3] The effect of a nutritional intervention on blood pressure was documented more than 50 years ago in the classical studies by Kempner,[4] which reported an impressive reduction in blood pressure with use of a low-sodium, rice-based diet. However, it is important to

remember that each of these older studies altered several nutritional factors, so the blood-pressure-lowering effects reported are most likely the result of several nutritional changes rather than a single dietary approach.

Most—if not all—nutrients or combinations of nutrients have been implicated both in the pathogenesis of and therapy for hypertension. At the population level, however, only a few nutrients are of clinical importance. This short review will focus on potassium, sodium, calcium, and magnesium, as well as the factors of body weight and alcohol consumption. Although the dietary factors will be discussed separately, it should be remembered that all nutrients show a high degree of co-linearity,[5] so any separation is somewhat artificial.

SODIUM

Sodium (Na^+) is the nutrient most often related to hypertension, although other nutrients or factors—such as potassium, obesity, or alcohol consumption—may be of equal, if not greater, importance. Sodium and chloride are the most important cations in the extracellular space, playing a central role in maintaining the electrochemical gradient between the intra- and extracellular space. The body's sodium stores are limited; thus, a continuous dietary supply of this cation is essential for survival. Accordingly, humans have evolved a variety of sodium-preserving mechanisms, which, in the modern environment of sodium excess, may represent a risk for hypertension in certain individuals.

The first relationship between blood pressure and salt intake was reported by Dahl et al[6] in the 1950s. Many subsequent studies showed a similar relationship between sodium chloride (table salt) intake and blood-pressure levels.[6] One of the largest, the INTERSALT Study, included more than 10,000 subjects in 52 research centers around the world.[7–9] This study reported a positive, although weak, relationship between salt intake (measured with urinary sodium excretion) and blood pressure. However, the populations studied were very heterogeneous and included some nonacculturated populations such as the Yanomamo, an Amazonian tribe, and population groups of Papuans from New Guinea. The latter populations are characterized by low blood pressure, a very low sodium intake (in the range of 1–3 g per day) and a high potassium in-

take, along with a low body weight and a high level of physical activity—all lifestyle factors associated with low blood pressure. Omitting these populations from the statistical analysis of the INTERSALT data set led to a loss of the positive relationship between sodium intake and blood pressure.[10] Contrary to the INTERSALT Study, other large-scale studies of less heterogeneous populations, such as the Scottish Health Trial,[11] failed to find a relationship between sodium intake and blood pressure.

In view of the differences in the individual handling of sodium as a function of miscellaneous endogenous and exogenous factors, these controversial results are not surprising (see Table 1). In addition, cross-cultural studies are more prone to positive results and should be used very carefully for interfering causality between sodium and blood pressure.

Nevertheless, a meta-analysis of different studies found an overall positive relationship between salt intake and blood pressure.[12] Although the latter study is widely cited, it is surprising that the INTERSALT study was not included in this meta-analysis. It is possible the results would have been different had it been included.

Table 1. Indicators and Determinants of Salt Sensitivity

Factor	Comment
Genetic background	
Race	African-Americans have a higher prevalence of salt sensitivity.
Blood-pressure level	The higher the blood pressure, the higher the probability of salt sensitivity.
Obesity	Body weight changes affect salt sensitivity.
Age	Salt sensitivity increases with age.
Potassium intake	A diet high in potassium is usually low in sodium. Potassium has a direct natriuretic effect.
Calcium intake	Calcium may elicit a natriuretic effect. The natriuretic effect of calcium is small, compared to that of potassium.
Expansion of extracellular volume	Increased salt sensitivity.
Low renin hypertension	Low renin concentration reflects a volume overload, which is associated with an increased salt sensitivity.
Kidney function	Impaired kidney function (eg, hypertensive nephropathy).

Several meta-analyses examining the pooled effect of interventional trials again reached contradictory conclusions.[13] There may be several explanations. First, not all subjects seem to be responsive to a modification of salt intake; this corresponds to the concept of "salt sensitivity" (see below). In addition, interventional studies do show different results as a function of the duration of the intervention. In longer-term intervention studies with salt restriction, a smaller blood-pressure-lowering effect was observed than in short-term interventions.[6,14] This may be partially attributable to time-dependent adaptive phenomena and, even more so, to study participant's insufficient long-term compliance with the low-sodium diet. The latter factor is of great interest since long-term compliance with low-salt diets is often expected, but is—as daily practice shows—an unrealistic expectation for most patients.

Sodium may lead to an increase in blood pressure by different mechanisms, such as expansion of the extracellular volume, increased vascular resistance, hormonal factors involving the sympathetic nervous system, vasoactive hormones from the endothelium, or abnormalities in ion handling and transport at the level of the vascular smooth muscle cells.[15-17] There seems to be no single mechanism responsible for the blood-pressure-increasing effects of sodium, and it seems clear that these effects may be strongly modulated by genetic factors as well as environmental factors, including other dietary components (eg, potassium intake) or acquired kidney damage (eg, hypertensive nephropathy).

In most acculturated populations, salt intake varies between 8–15 g per day and, according to data from NHANES III, current sodium intake in the United States (including all ages and races) is 3289 mg per day, corresponding to 8.3 g salt per day. The highest salt intake—12.5 g per day—was found in young, non-Hispanic, African-American males aged 16 years to 19 years.[18]

Up to 50% of sodium intake comes from processed food products, including bread, bakery products, and meat, as well as other convenience foods; an additional 40 to 45% is added during the food preparation process. A comparatively small amount is added at the table (5–15%). Unprocessed food products are a negligible source of salt (5–10% at most). The abundance of sodium in processed foods and its common addition during the cooking process suggest an easy strategy to attain lower sodium intakes.

About 40% of hypertensive subjects are "salt sensitive." In the presence of salt sensitivity, salt restriction can be expected to lead to a reduction in blood pressure. Attempting to implement salt restriction in all patients is not realistic and, in any case, only a minority would profit from the intervention. Therefore, limiting salt restriction to salt-sensitive subjects is the most promising and satisfying strategy.

Unfortunately, however, there is no inexpensive or simple way to classify patients as salt-sensitive.[19] Several possible predictors of salt sensitivity are summarized in Table 1. Major predictors seem to be body weight (obesity), age, blood pressure, kidney function (creatinine levels), and race (African-Americans). With increasing obesity, age, and blood-pressure levels we find an increasing probability that the patient will be salt sensitive. In such patients, a trial of stricter sodium restriction is more than warranted and may indeed represent an important and effective adjunct in the treatment of hypertension. A reduction in salt intake to evaluate the possibility salt sensitivity is probably indicated in any patient with mild hypertension who reports a high salt intake and in any difficult-to-treat hypertensive patient.

In daily practice, correct assessment of salt intake is one of the major difficulties. Dietary history of sodium intake is usually of no clinical value since sodium occurs so widely and because the sodium content of many foods is not known precisely and may vary from one occasion to the other. The only useful methodology for assessing sodium intake is a 24-hour urinary sodium excretion measurement.[20] Although dietary sodium intake may vary considerably from one day to the next, a single 24-hour collection may reflect usual sodium intake as long as the patient is in a steady-state condition regarding his or her food intake. If a hypertensive patient's creatinine clearance or protein/albumin excretion is being determined with a 24-hour urinary collection, it may be reasonable to measure the sodium chloride as part of the same analysis. Shorter periods of urinary collection are not reliable for the assessment of daily sodium intake; however, the measurement of sodium in spot urine samples may be helpful for assessing compliance with a low-salt diet.

The capacity to handle a sodium load is modulated by a variety of factors.[21] One important and often neglected factor is urinary volume. A recent study showed that sodium excretion is facilitated by abundant water intake; the authors assume that low sodium excretion in the case of low fluid intake (and, thus, low urinary volume) is caused by vasopressin.[22] The capacity of the kidney to excrete sodium seems to be limited in the setting of a low water supply. Accordingly, on a high sodium intake—in other words, a typical, modern American diet—more than abundant water must be consumed, assuming there is no medical contraindication for high fluid intake. Potassium intake is another important factor modulating dietary sodium intake (see below).

In view of the wide occurrence of sodium in food, a dietary prescription for severe sodium restriction is not simple to accomplish. However, considerable reduction in sodium intake can be achieved by the avoidance of salty foods, by not adding table salt to food, and by avoiding processed foods (except low-sodium foods). Further, a diet high in fresh fruits and vegetables is necessarily low in sodium. The consumption of a diet rich in fruits and vegetables—such as that recommended by the DASH study (see below)—in combination with moderately reduced sodium intake may be a more realistic and effective strategy, as far as long-term feasibility, than severe sodium restriction.

To what levels should salt intake be reduced in salt-sensitive subjects? This question cannot be answered easily and, indeed, is probably the wrong question. In view of the strong interrelationship between different cations, it makes more sense to ask this question: To what extent should we modify the sodium-to-potassium ratio? It is possible that in an older obese patient with grade I hypertension and high sodium intake, blood pressure could be lowered significantly with severe sodium restriction. But while the outcome may be positive as far as the absolute blood-pressure value is concerned, we know that strict sodium restriction is not realistic for most patients and may even have unfavorable effects. For elderly patients, low sodium content may make food less attractive and palatable, leading to reduced food intake with the potential risk of malnutrition. In view of the present evidence, it would be more effective to optimize the dietary ratio of sodium to potassium, rather than fo-

cussing on sodium alone.[23] The maintenance of an adequate potassium nutriture is discussed below.

Despite ongoing controversy,[24,25] the following conclusion can be formulated. In view of the evidence, we would advocate that hypertensive patients, as well as normotensive patients, adopt moderate salt restrictions (6–7 g/day) in *combination*[26] with an optimal compliance with an antihypertensive drug regimen and a diet rich in fruits and vegetables (and thus rich in potassium).[27]

POTASSIUM

Potassium (K^+) is the most common intracellular cation, playing an essential role in the maintenance of the electrochemical excitability of muscles and nerves. Many factors influence the K^+ concentration in the plasma and in different parts of the body. The usual dietary K^+ intake is in the range of 50–100 mmol per day. In a healthy person, most of the K^+ is absorbed completely, and in the physiological situation of a steady state, the identical amount is excreted in urine. Thus, the urinary measurement of K^+ represents an important and reliable tool for the assessment of dietary intakes.[20] The renal excretion of K^+ represents the central regulatory mechanism for maintenance of the potassium balance and, as long as kidney function is normal, potassium status and balance are easily maintained. However, in many hypertensive patients with renal insufficiency this is not the case.

Many factors influence the urinary excretion of K^+, including sodium (Na^+) concentration. A high concentration of Na^+ in the distal tubules of the kidney—as occurs with a high-sodium diet—enhances urinary K^+ excretion; in contrast, potassium has direct natriuretic effects.[28] The latter physiological interaction suggests the importance of the interaction between these two cations and, accordingly, it is the sodium-to-potassium ratio that is crucial in the diet, rather than either single cation alone.

Aside from sodium, potassium is probably the most important electrolyte for the nonpharmacological control of blood pressure. In epidemiologic studies, an inverse relationship between potassium intake and blood-pressure status has been observed, and the blood-pressure-lowering effects have been reproduced in supplementation trials.[29-32] A recent meta-analysis of 33 randomized clinical trials that assessed the effect of oral supplementation of po-

tassium on blood pressure reported a significant reduction of the systolic and diastolic blood pressure of 3.11 mm Hg (1.91–4.31 mmHg; 95% confidence interval) and 1.97 mmHg (0.52–3.42 mmHg), respectively.[29] The blood-pressure-lowering effects of potassium are especially seen in subjects with high sodium intake, which is in agreement with earlier observations of the interaction of sodium and potassium.

There probably are multiple mechanisms for potassium's blood-pressure-lowering effects. Suggested mechanisms include modulation of baroreceptor sensitivity, direct vasodilatatory functions, modulation of the catecholamine metabolism, improvement of glucose tolerance, and modulation of the renin-angiotensin-aldosterone system. In addition, consumption of a potassium-rich diet has a rather strong natriuretic effect, and diets that are high in potassium usually are low in sodium, as long as unprocessed foods are consumed.

The blood-pressure-lowering mechanism that prevails differs from one individual to another, which may also, in part, explain the lack of an effect of potassium supplementation in some studies. A low-sodium diet is usually not associated with increased urinary loss of potassium, thus leading to a relative lack of response to supplementation as far as blood pressure is concerned.[32] Furthermore, the potassium-induced natriuresis may lead to a decline in plasma volume, thus leading to a stimulation of renin, which abolishes the blood-pressure-lowering effects of potassium as well as other blood-pressure-lowering strategies. The observed potassium effects on blood pressure fulfill the criteria of causality and are readily reproducible in supplementation trials. These supplementation trials are helpful in clarifying potential mechanisms and interactions of potassium with the different blood-pressure regulatory circuits. However, the results of these trials should not lead to the conclusion that potassium supplements are necessary for the prevention and or treatment of hypertension.

The average potassium intake in the United States varies widely—between 30–100 mmol per day. In the absence of disease, a healthy individual can maintain short-term potassium balance with intakes as high as 10 mmol/kg body weight. Potassium balance is influenced mainly by intake and urinary excretion. In the presence of normal kidney function, most of the potassium is excreted in urine. Despite these regulatory capacities, potassium supplementation could be dangerous and should not be prescribed as a blood-pressure-lowering therapy.

Whenever possible, potassium should be obtained from the diet. Although the potassium content of food may vary widely, milk, fruits, grain products, vegetables, and even some meats represent fairly good sources. In estimating potassium content of various foods, there are a few general rules we can follow. Fruits and vegetables are usually high in potassium and low in sodium, as long they are not processed. The higher the degree of food processing, the lower the potassium content and the higher the sodium content. This is nicely illustrated by the low sodium-potassium ratio in raw potatoes, and the high ratio in potato chips. We find the same ratio in comparing raw tomatoes and commercial tomato sauce or ketchup. The dietary sodium-potassium ratio primarily determines the blood-pressure effects of a certain food item, rather than the potassium or sodium content alone. A high sodium-potassium ratio has a high potential to be associated with blood-pressure elevation, whereas a diet with a low sodium-potassium ratio is associated with potential blood-pressure-lowering effects. The latter type of diet should be favored, and these diets are usually plant and vegetable based. Accordingly, vegetarian diets provide larger amounts of potassium than meat-based diets. It has even been suggested that the relatively high potassium content of vegetarian diets is responsible for the beneficial blood-pressure effects of these diets.[33,34] These findings suggest that there is no single dietary factor or no single "miracle nutrient" that is responsible for blood pressure lowering or increased blood pressure; rather, it is the diet as a whole. The co-linearity of nutrients is a well-known phenomenon in nutritional epidemiology.[5] Nevertheless, it is possible to eat a diet that contains the ideal nutrient composition.

These longstanding observations about the effects of different nutrients on blood pressure have been implemented in the DASH trial (Dietary Approaches to Stop Hypertension).[35] The DASH trial compared the effect of a typical U.S. diet (comparatively low in fruits and vegetables) with a diet high in fruits and vegetables as well as low-fat dairy products. In addition, the DASH diet contains plenty of whole-grain products, poultry, fish, and nuts;

red meat is eaten only rarely. The diet is comparatively high in potassium, as well as other nutrients such as vitamins and calcium, all eliciting potential blood-pressure-lowering effects.

The blood-pressure effects of the DASH diet were shown to result in a reduction in systolic blood pressure by 5.5 mmHg and diastolic blood pressure by 3.0 mm Hg. Although the DASH diet is higher in potassium, the blood-pressure-lowering effect may be due, in part, to other nutrients. The DASH diet concept has been integrated into guidelines for the treatment of hypertension.[3]

A recent study looked at the effect on blood pressures of a combination of the DASH diet with sodium restriction.[27] In this study, the reduction of sodium intake was associated with a reduction of blood pressure as a function of the level of sodium reduction. Consumption of the DASH diet led to a further reduction of blood pressure at any level of sodium intake.[27] As compared to the control diet, which had a high sodium content, the DASH diet, in combination with a low sodium intake, was associated with a 7.1 mmHg lower systolic blood pressure in normotensive subjects. In hypertensive subjects, the combination diet reduced systolic blood pressure by 11.5 mmHg. These findings are very impressive and of great public health importance. However, the duration of the DASH trial was only 30 days, and it remains to be shown whether individuals can follow the combination diet over longer periods of time. For now, it is probably best to follow the DASH diet in *combination* with *moderate* sodium restriction. The public health importance of the DASH trial is also reflected in the concept of the trial—that it is the *whole* diet that counts, with the importance of single nutrients less stressed.

Renal insufficiency is one of the major endpoints of hypertension. Many hypertensive patients may have undiagnosed renal insufficiency, putting them at a high risk for potentially deadly hyperkalemia. Accordingly, no potassium supplements should be taken except in the presence of a medical indication (such as diuretic-induced hypokalemia). The ingestion of salt substitutes, especially in combination with certain drugs (eg, potassium-sparing diuretics, ACE-inhibitors), may represent a dangerous combination with a high risk for hyperkalemia.[36] In the absence of renal insufficiency, the moderate use of potassium-containing salt substitutes may be helpful in subjects with a known high sodium intake. Potassium salts elicit a bitter aftertaste, however, so for some subjects they may reduce palatability and lead to reduced food intake.

Another favorable effect of the increased intake of potassium-rich food may be a reduction of the need for antihypertensive medications, meaning fewer potential side effects.[37]

Maintaining an adequate level of potassium intake is of central importance in any patient because evidence suggests that the cardiovascular protection in treated hypertensive subjects exhibiting plasma potassium concentration < 3.5 mmol/L is reduced or even lost.[38] These observations are in agreement with the importance of potassium in cardiac function and performance as well as other issues, such as stroke protection.[39] Accordingly, any patient not at risk for hyperkalemia should increase his or her dietary intake of potassium from natural sources. This strategy has a high potential for reducing cardiovascular risk independent of possible blood-pressure-lowering effects.

CALCIUM

Calcium (Ca^{2+}) is known to play a major role in the function of the smooth muscle cells and, thus, may be of importance in the modulation of blood pressure. Various studies have reported an inverse association between calcium intake and blood pressure; in addition, cardiovascular mortality has been related to calcium content of drinking water.[40] Further interventional trials with calcium supplements reported only a small reduction or no change in blood pressure in most cases.[41] Only 2 prospective studies found an inverse relationship between Ca^{2+} intake and blood pressure.[42] One meta-analysis was able to show a small, but significant, reduction of systolic blood pressure only upon daily supplementation with 1000–2000 mg of Ca^{2+}.[43] The effect of Ca^{2+} on blood pressure is comparatively small (in the range of 1–2 mmHg) and inconsistent. The effect of calcium supplements on blood pressure varies according to the level of blood pressure[44] and other population characteristics, such as pregnancy-related blood-pressure changes[45] or high salt intake.[46] Similar blood-pressure effects were found for a Ca^{2+}-rich diet or the same amount of Ca^{2+} intake through supplementation.[47] In agreement with these observations, one blood-pressure-lowering nutrient in the DASH diet may have been calcium.[27,48] Again, however, we

must remember that Ca^{2+} is only one component of the DASH diet, and, accordingly, increased Ca^{2+} intake represents only one component of a blood-pressure-lowering diet—and a minor one at that.

In view of the only small blood-pressure-lowering effects of Ca^{2+}, current evidence does not support increased Ca^{2+} intake for blood-pressure-lowering purposes alone. Nevertheless, increased Ca^{2+} intake in form of the DASH diet may represent an important dietary strategy,[27,35] and a calcium intake in the range of present recommendations should be assured.[49-51] The easiest strategy for increasing calcium intake is the consumption of low-fat dietary products.

MAGNESIUM

The usual daily dietary intake of Magnesium (Mg^{2+}) is about 300 mg. Serum Mg^{2+} represents only 0.3% of the total body Mg^{2+} content and is regulated very tightly without reflecting intracellular Mg^{2+} status. Accordingly, the diagnosis of Mg^{2+} deficiency is very difficult, especially in the setting of epidemiological studies, but also in clinical practice.[52] Since changes in extracellular and intracellular concentration of Mg^{2+} modifies the vascular tone and contractility, magnesium has been postulated to have blood-pressure-modulating effects, as well as effects on cardiovascular function.[53]

As early as 1925, Blackfan[54] reported an improvement in malignant hypertension after Mg^{2+} infusion.[54] However, experimental and epidemiological studies could not consistently find blood-pressure-lowering effects of oral Mg^{2+} in normotensive as well as hypertensive patients. The Nurse's Health Study reported a 23% reduction of the risk to develop hypertension in women with a daily Mg^{2+} intake of >300 mg as compared to an intake of <200 mg per day.[55] The DASH diet is high in Mg^{2+}, which may be one causal dietary factor for the blood-pressure-lowering effect seen in the DASH trial,[27,35] but, once again, Mg^{2+} represents just a single component that may lead to blood-pressure reduction. As compared to sodium or potassium, the blood-pressure-lowering effects of Mg^{2+} are rather small.

A large retrospective study found that the Mg^{2+} levels in drinking water were inversely related to the risk of death from hypertension.[56,57] On the other hand, several studies did not report a blood-pressure-lowering effect of Mg^{2+} supplementation.[58,59]

As with other nutrients, some subgroups of the population may consistently show favorable blood-pressure effects upon an increased Mg^{2+} intake—particularly African-American patients[60] or patients with hypomagnesiemia.[61]

Present evidence suggests only inconsistent and weak hypotensive effects of Mg^{2+}. In view of this, it is impossible to make a statement about the exact role of magnesium in the regulation of hypertension; therefore, supplementation is not warranted. A Mg^{2+} intake in the range of the present recommendations is the best approach for the time being.

ALCOHOL

Alcohol represents one of the most important pressor agents in our diet.[62] As early as 1915, the French researcher Lian[63] observed a significantly higher prevalence of hypertension in heavy drinkers (defined as more than 3 L wine per day) as compared to the control group, which consumed 1 L of wine per day. Many cross-sectional studies have confirmed this relationship.[64] The pressor effects have been induced in interventional studies,[65] and it has been observed that heavy alcohol consumers do show a blood-pressure reduction upon hospital admission and cessation of alcohol intake.

While the pressor effects of alcohol are not in doubt, the exact relationship between alcohol and blood pressure remains controversial: Is the relationship linear, curvilinear, or is there a threshold level below which no pressor effects are observed? In addition, due to methodological problems in the assessment of alcohol intake, we do not know whether low levels of alcohol intake (light consumption) are associated with increased blood pressure. The phenotypic expression of hypertension is the result of a complex interplay of different blood-pressure regulatory systems, which are themselves influenced by environmental as well as endogenous (eg, genetic predisposition) factors. Accordingly, several potential mechanisms for the blood-pressure effects of alcohol have been identified, including hormonal effects, catecholaminergic effects, altered baroreflex sensitivity, increased vascular tone, or an enhancement of abdominal obesity.[66] Alcohol may influence the metabolism of antihypertensive drugs (eg, decreased absorption and increased metabolic clearance of metoprolol), thus reducing their effect. The pressor effects are much

more pronounced in older individuals than in younger subjects. In view of the strong and consistent effects of alcohol on blood pressure, alcohol as been characterized as one of the major causes of essential hypertension.[67]

Any hypertensive patient consuming more than moderate amounts of alcohol should reduce alcohol intake independently from blood-pressure control. Any patient with difficult-to-treat or resistant hypertension should, at least during the period of work-up, avoid alcohol intake completely. In hypertensive patients with optimal blood-pressure control, light to moderate alcohol consumption should be allowed. Alcohol should not be consumed daily and, whenever possible, the alcohol should be consumed with meals only.[68]

BODY WEIGHT

Obesity is increasing in the United States,[69] representing what is probably the most important and most prevalent cardiovascular risk factor. An increased body weight has been identified as one of the major modulators of blood pressure,[70-72] especially in the presence of abdominal obesity.[73] The increase in blood pressure with increasing age is due in part to an increase in body weight with aging. A reduction in body weight is usually associated with a reduction in blood pressure.[74]

Despite the potential of weight loss to induce a reduction in blood pressure, most weight-reduction strategies are unsuccessful in the long run. One of the characteristic features of aging is a steady increase in body weight. It is known that the risk of developing hypertension increases according to the weight change during adulthood. The combined effect of aging and weight gain probably has a stronger effect on blood pressure than each factor alone.

Accordingly, a stabilization of body weight represents the best, and also most realistic, initial strategy for many hypertensive patients. After successful weight stabilization is attained, the chance for successful weight loss with maintenance of body weight at the lower level is increased. This is due to the positive experience and learning effects of the former weight stabilization. On the other hand, in a patient who is unable to lose weight and maintain his or her weight, a stabilization of body weight must be judged as success.

The ideal choice of antihypertensive agent for obese hypertensive subjects is still a matter of some controversy. However, some patients may react to beta-blocking agents with an increase in body weight.[75]

Regular physical activity is the cornerstone for the stabilization of body weight.[76,77] Aside from its favorable effects on body weight, regular physical activity is associated with reduced blood pressure and overall improvement of cardiovascular risk. The control of overweight and obesity should have a high priority in any hypertensive patient.

CONCLUSION

In view of the complexity of blood-pressure regulation, we must remember that many different nutrients and diets may affect blood pressure (eg, vitamin C, fish oil, dietary fiber, amino acid composition of the diet).[78,79] Nevertheless, from the clinical point of view, the factors discussed in this article represent the most promising strategies for blood pressure lowering in most individuals (see Table 2).

With potent antihypertensive drugs so readily available in the clinical setting, nonpharmacological strategies of blood-pressure control are often forgotten, as a recent Finish study reports.[80] The underuse of nonpharmacological means of blood-pressure control will add to the suboptimal control of hypertension and to atherosclerosis in general.

It is never too late to implement nonpharmacological strategies for the prevention and treatment of hypertension. In addition, it is never *early enough* to implement these strategies, since most risk factors track from childhood to adulthood.[81] In view of the often inefficient lifestyle interventions in adulthood, certain health-prone behaviors—including diet and food choices—should be learned in childhood so that these healthy habits are carried into adulthood. In our society, blood pressure increases with aging, and this phenomenon is often wrongly regarded as normal or physiological. However, this rise in blood pressure is not an inevitable consequence of the aging process but, rather, a consequence of our lifestyle. In the MRFIT study, only 5 lifestyle habits—high salt intake, high ratio of sodium to potassium, alcohol consumption, calorie imbalance, and a sedentary life—were identified as adversely affecting blood pressure.[26] Accordingly, moderate positive changes in all 5 of these habits combined (instead of severe intervention involving 1 factor alone, such as salt) would be the most realistic and effective

Table 2. Key Recommendations for the Dietary Modulation of Blood Pressure

Nutritional Factor	Recommendation
Body weight	• Stabilize or if overweight/obese reduce body weight • Avoid weight gain with aging
Control of alcohol intake	• Avoid daily alcohol intake • Drink only with meals • Drink only in combination with at least the same volume of nonalcoholic beverages • Avoid highly concentrated alcoholic beverages • Drink slowly • Do not increase alcohol consumption over time; avoid excessive alcohol intake
Sodium reduction	• Avoid addition of sodium in form of table salt • Promote the consumption of fresh, unprocessed foods • Implement strict sodium reduction only in subjects with salt sensitivity • Increased sodium intake should be followed by abundant nonalcoholic fluid intake • Use a salt replacer such as potassium chloride
Fruits and vegetables	• Consume vegetables with any meal • Add fruits to each main meal and use to replace any type of snack
Potassium	• Increase potassium intake by increasing consumption of fruits and vegetables • Avoid excessive salt intake to minimize potassium losses • Potassium supplements are not routinely recommended
Fluid intake	• Abundant water intake should be pursued every day, especially in the setting of a high sodium intake • Consume noncalorie-containing fluids • Distribute fluid intake over the day
Physical activity	• Pursue regular physical activity whenever possible, and at least 30 minutes every day • Integrate physical activity into the daily schedule • Reserve time in your daily schedule for physical activity
Calcium	• Pursue dietary intake of calcium according to the present recommendations by the consumption of low-fat dairy products

strategy to counteract the present hypertension epidemic.[27,82] In view of the ongoing debate about salt and hypertension, this approach seems not only the safest, but also the most promising.[83]

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