Alternatives to Traditional First-Line Treatment
Potential Benefits and Costs

Nonpharmacological Therapy of Hypertension
Commentary on Diet and Blood Pressure

Herbert G. Langford

The use of dietary measures as sole therapy for hypertension has generated much enthusiasm and is supported by epidemiological and anecdotal data but is not substantiated by long-term controlled trials. Dietary measures should be effective in preventing the rise in blood pressure with age in civilized populations, but there are no firm data to substantiate this hypothesis. However, some data do suggest that weight loss and sodium restriction may benefit the drug-treated hypertensive patient. There are strong reasons to consider the use of nonpharmacological measures for the treatment of mild hypertension. Mild hypertension is ubiquitous. Some population subsets have a prevalence of mild hypertension approaching 40%, based on one blood pressure determination, for individuals older than 50 years. Even if the true prevalence is closer to 15%, this is a high proportion of the population in need of drug therapy. If a change in lifestyle could reduce the need for drug therapy by a respectable percentage, then an enormous number of individuals could be spared the necessity of taking antihypertensive medication. (Hypertension 1989;13(suppl I):I-98-I-102)

This article examines the evidence for the utility of dietary change as sole therapy or as a measure used in conjunction with drug therapy in the treatment of hypertension. The strong positive correlation between weight and blood pressure within populations suggests that weight loss should be an excellent means of controlling mild hypertension. Experimental studies on the relation of sodium to hypertension, the efficacy of thiazide diuretic drugs as hypotensive agents, and observations from epidemiological studies of cross-cultural differences in sodium intake and blood pressure suggest that sodium restriction can be valuable for the management of hypertension. Similar arguments can be made for increasing potassium, calcium, and unsaturated fat intake although conclusive evidence for these constituents is lacking.

Measures regarding efficacy, compliance, side effects, and costs that are frequently asked about pharmacological therapy should also be applied to nondrug therapy. If short-term efficacy can be proven, it would be desirable to know if long-term reduction of blood pressure by nonpharmacological means is associated with decreased morbidity and mortality. This finding has been demonstrated for thiazide and β-blocker hypotensive regimens but not for nonpharmacological antihypertensive interventions.

Weight Loss

Most cardiovascular epidemiological studies have shown a strong positive correlation between weight and blood pressure. Many of these studies were reviewed by Chiang et al in 1969. Later studies further documented this correlation. However, there was little interest in weight loss as a therapeutic maneuver from the time Dahl et al published their careful studies in 1958 until Reisin et al’s report in 1978. Dahl et al examined obese women who were confined to the hospital, and they found a pronounced decrease in blood pressure when sodium intake was reduced; however, they observed no decrease in blood pressure when weight was reduced without a decrease in sodium intake. Reisin et al studied hypertensive individuals with uncontrolled hypertension. One half of this cohort was given no dietary advice, and the other half was given dietary advice about weight loss with a stable sodium intake. In fact, kosher dill pickles were permitted for the weight-loss group. There was a significant decline in blood pressure that accompanied the 10-kg weight reduction in the weight-loss group.
Urinary sodium excretion was only measured at the end of the study, and there was no difference in sodium excretion between the two groups. Although there were some flaws in this study, it can be used to conclude that significant weight loss is effective in lowering blood pressure, and that the decrease in blood pressure is not attributable to sodium reduction. In two more recent studies, \(^4\), \(^5\) weight loss was also shown to be effective in slowing or preventing the return of hypertension after prolonged drug treatment had produced a normotensive level. Both studies included participants in the Hypertension Detection and Follow-up Program. \(^6\) The findings of the studies were similar: an approximate 10-pound weight loss was maintained for the duration of the study. Stamler et al's study\(^4\) was especially encouraging because the patients were followed up for 4 years, and they found that, during the 4-year follow-up, 50% of the participants were able to remain normotensive as well as to maintain their weight. This study extended the findings of a previous study that lasted only 1 year.\(^5\)

There are only two published randomized trials concerning the use of weight loss as primary treatment for the patient with essential hypertension. Haynes et al\(^7\) recruited mildly hypertensive subjects who were moderately obese into a study based in the Toronto, Canada area. The mean weight loss in those who received dietary counseling was 5 pounds. There were no differences noted in the blood pressure levels of those who received dietary advice compared with those randomized to no dietary advice, despite the fact that the latter group did not lose weight. The study's power was quite high, and thus it can be used to suggest that modest weight loss (10 pounds) is ineffective in lowering blood pressure in patients who had not been taking medication. In yet another study, MacMahon et al\(^8\) randomized participants to a weight-loss program, to a no-intervention group taking a placebo tablet, or to a no-dietary-intervention group taking metoprolol. At the end of 6 months, the weight-loss group lost an average of 15 pounds, and this loss was associated with a 15-mm-Hg decline in blood pressure.

Several other studies have demonstrated the ability of marked weight loss to lower blood pressure. Most of these studies are not controlled, but the decrease in blood pressure in these studies is nevertheless impressive. These investigations of marked weight loss in the severely obese have usually used formula diets. Such diets are undoubtedly effective in short-term weight change, but they are rarely followed by prolonged maintenance of the weight loss.

To extrapolate beyond the published material on weight loss and blood pressure, an argument can be made for using weight loss as sole therapy in the uncomplicated mildly hypertensive individual if the following criteria are met: 1) Patients should be followed up closely for their blood pressure response because some patients will not respond to weight loss, just as some do not respond to other antihypertensive modalities. 2) Weight loss probably should not be considered as sole therapy unless a planned continuing program aided by the advice of a trained psychologist and nutritionist is available. and 3) Based on the results of MacMahon et al's study,\(^8\) the weight loss goal in a person of average size should be at least 15 pounds.

**Decreased Sodium Intake**

The interest in sodium as a major factor in hypertension can be ascribed to a number of facts: 1) Increased sodium intake in some but not all experimental animals leads to elevated blood pressure. 2) Extremely low intakes of sodium like those used in the Kempner diet lead to blood pressure reduction. 3) Individuals living in remote areas who take little sodium do not develop hypertension, and hypertension appears to be especially prevalent in areas of high salt consumption like northern Japan. and 4) Probably most important, diuretic agents regularly lower blood pressure and are a mainstay of antihypertensive therapy.

However, there is a paucity of well-controlled studies that address the use of sodium restriction alone as antihypertensive therapy. Parijs et al\(^9\) found a significant decrease at 1 month in home but not in office blood pressure. MacGregor et al\(^10\) found that lowering sodium intake from 180 to 80 meq/day produced a significant decrease in mean blood pressure. They did not report the change in diastolic pressure, presumably because it failed to reach significance. Two carefully done studies\(^11\),\(^12\) failed to find any benefit in blood pressure reduction from sodium restriction in mildly hypertensive individuals. There appear to be three differences between those studies reporting positive results and those reporting negative results: 1) The initial sodium intake was higher, and the final sodium intake was lower in the positive studies. 2) Patients in the positive-result studies had higher blood pressures, and 3) The positive results were obtained in smaller short-term studies.

This analysis paints a discouraging picture because continued long-term therapy of the mildly hypertensive patient is what one would want from dietary change. Sodium restriction as sole therapy for the mildly hypertensive individual is eminently reasonable, but sodium restriction cannot be considered as proven therapy. However, a basic problem connected with recruiting individuals for clinical trials impedes testing the utility of restricting sodium intake. Volunteers for nutritional-change programs tend to be health-conscious individuals who have already made numerous changes in their diets. The studies of mild hypertension quoted above fail to address the utility of reducing sodium intake from "high-normal" (~200 meq Na/day) to "low-normal" (~100 meq Na/day).

Decreasing sodium intake by one half may well be an effective antihypertensive therapy for mildly hypertensive patients. Unfortunately, studies are not avail-
able to document the utility of such a large change in sodium intake, nor is there any easy and accurate way to determine compliance with a low-sodium diet. Even several 24-hour urine samples will give only a rough estimate of habitual sodium intake.

The situation is more encouraging for the combination of drug therapy and sodium restriction. Fallis and Ford\(^{13}\) and Ram et al\(^{14}\) showed that sodium restriction could be combined with a lesser dose of a diuretic agent to obtain a blood pressure reduction equivalent to that obtained with a full dose of the thiazide diuretic agent and unmodified sodium intake. Moreover, the degree of hypokalemia was less on the former regimen. Langford and colleagues\(^{5}\) showed that a low-sodium diet in patients who had been treated for hypertension for 5 years and who were then withdrawn from medication substantially slowed the relapse of hypertension.

It is difficult to follow a diet that is markedly depleted in sodium. Reduction of sodium intake to about 100 meq/day is the best that most American studies have obtained in free-living individuals. There are no data on the cost and inconvenience of such a diet. Compliance is difficult and rarely ascertained in clinical practice. From the studies listed above and from the considerations just noted, I conclude that sodium restriction should be considered as an addition to drug therapy of hypertension and not as sole therapy.

Potassium

In 1928, Keith and Binger\(^{15}\) reported the value of potassium supplements as natriuretic agents in congestive failure. Uncontrolled studies have also been published on the value of potassium supplementation as therapy for hypertension. The famous Kempner diet could be described as a high-potassium diet. In fact, Kempner, in some of his writings, commented on the diet’s favorable sodium-potassium ratio. Dahl et al\(^{16}\) showed that, when potassium supplementation was given to rats of his salt-sensitive strain, the potassium appeared to blunt the blood pressure–elevating effects of sodium. Several epidemiological studies showed a better correlation between blood pressure and sodium-potassium ratio than with sodium alone. More recently, Tobian\(^{17}\) reported that potassium supplementation, even when it fails to lower blood pressure in spontaneously hypertensive rats, protects against cardiovascular end points. Even more exciting, Khaw and Barrett-Connor\(^{18}\) found that a high-potassium intake as determined by dietary history is correlated with a lower stroke rate.

Despite the numerous considerations listed above, trials of potassium supplementation have been effective only in perhaps one of two of the reported studies. Moreover, the studies that have attempted to increase potassium intake by diet have met with minimal if any success. I conclude that, on theoretical grounds, an increased dietary potassium intake appears desirable, but it has not been shown to be effective or feasible in lowering blood pressure in the long term.

Calcium

McCarron and Morris\(^{19}\) have revived and energetically supported the concept that a large calcium intake will lower blood pressure. The epidemiological data on this topic remain difficult to interpret. Part of the problem is the phenomenon noted by Reed et al\(^{20}\) of the colinearity of calcium ingestion with the ingestion of potassium, protein, and several other nutrients. Another problem may be due to the phenomenon that Resnick and Laragh\(^{21}\) described in a small, well-controlled, center study. Patients with low ionized calcium benefited from calcium supplementation. Those whose ionized calcium level was above the median did not benefit. Perhaps this phenomenon was the cause of the inconsistent results noted by Langford et al\(^{22}\) a number of years ago. (In one sample, we found a negative correlation between calcium excretion and blood pressure; in another sample, we found a positive correlation.)

It is difficult to interpret the results of calcium supplementation on blood pressure. About half the studies have yielded positive results, which may not be apparent until the 3rd month of therapy. No prospective randomized studies have been published on the effect on blood pressure of increasing calcium by dietary means and, therefore, no studies of compliance with a high-calcium diet. The entire topic of calcium and its role in blood pressure regulation remains unsettled, and at this time, calcium supplementation cannot be considered as a way to treat hypertension.

Unsaturated Fats

An intriguing dietary component possibly related to blood pressure regulation is unsaturated fats, some of which are involved in prostaglandin synthesis. Increased excretion of certain prostaglandins has been demonstrated as a consequence of increased unsaturated fat intake. The prostaglandins themselves are important for sodium excretion. Despite the intriguing conceptual basis for manipulating the distribution of dietary fats, studies to date have not demonstrated the beneficial effects on blood pressure of increasing unsaturated fats.\(^{23}\) Rouse et al\(^{24}\) have shown that Seventh Day Adventists, who eat no meat, have lower blood pressures than other comparable people in their geographic area. They have also found a blood pressure–lowering effect from a vegetarian diet, an effect that cannot be adequately explained by increased unsaturated fats (although these were increased) or by increasing potassium (although this was also increased). Fiber was also increased on this diet.
**Why Are Dietary Changes Ineffective as Sole Antihypertensive Therapy?**

There are a number of possible answers for this question: 1) Dietary change is difficult to implement, and continued compliance is often poor. 2) Perhaps all the dietary components listed above need to be changed. Suppose, for example, that weight loss to a normal weight will lower diastolic blood pressure by 3 mm Hg, that sodium restriction to 70 meq/day will lower diastolic blood pressure by 2 mm Hg, an increase in potassium intake to 100 meq/day will lower diastolic blood pressure by 1.5 mm Hg, doubling of calcium intake will cause another 1.5-mm-Hg decrease, increasing unsaturated fats will lower the diastolic blood pressure by 1.5 mm Hg, and increasing other dietary components like fiber and magnesium will result in another 1.5-mm-Hg decline. The numbers above are conjecture, but meta-analysis could give a better approximation of the change that could be expected from each individual dietary modification. However, the point is that there are many possibly offending or beneficial classes of food, and all of them may be involved. 3) An equally important concept is the difference between dietary causes and dietary treatment of hypertension. The attained blood pressure can be considered due to at least three factors, namely, genetic causes, environmental causes (i.e., the dietary components listed above plus psychological factors), and perpetuating or multiplicative factors. Folkow\(^2\) pointed out how a given stimulus will produce more vasoconstriction in an arteriole with hypertrophied walls than in an arteriole without hypertrophy. The practical translation of this concept is that a lesser stimulus is required to maintain hypertension than to cause it. I suggest that the last point is all-important in considering the place of nonpharmacological modalities (including psychological measures) in the therapy of hypertension. We can theoretically prevent hypertension if we can change the environmental factors that interact with the genetic components to raise blood pressure. We can add to the benefit of drug therapy by diet and stress changes. We may be able to treat individuals whose arteriolar hypertrophy has regressed after prolonged antihypertensive therapy by dietary changes. At this time, however, dietary change is not generally adequate as sole therapy for hypertension because the efficacy of dietary change has not been proven.

The conclusion that nutritional changes have a limited role as sole initial therapy for hypertension does not mean that nutritional change is unimportant in the care of the hypertensive patient. Weight loss and sodium restriction, as noted, can allow better blood pressure control or reduction in antihypertensive drug dosage. Weight loss also helps lower serum cholesterol. Weight loss and sodium restriction can allow continued normotension after discontinuation of antihypertensive therapy. Perhaps most importantly, there is every reason to think that prudent consumption of a low-sodium high-potassium diet that does not lead to obesity may prevent much of the hypertension seen in clinical practice.

**References**


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H G Langford

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