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Chapter 3

High Blood Pressure

Hence if too much salt is used in food,
the pulse hardens.
Huang Ti (the Yellow Emperor, 2697–2597 B.C.)
*The Yellow Emperor's Classic of
Internal Medicine*

Introduction

Hypertension, the medical name for high blood pressure, is a common chronic medical problem in the United States responsible for a major portion of cardiovascular disease. In recent years, public health efforts have increased public awareness and knowledge of the risks and appropriate treatment of this condition. As a result, almost the entire adult U.S. population has had at least one blood pressure measurement and 73 percent of Americans have had their blood pressure checked within the previous 6 months. By 1985, 77 percent of the public identified high blood pressure as the factor that most increases a person's chances of having a stroke, and 91 percent indicated that high blood pressure increases a person's chances of getting heart disease (Lenfant 1987). The proportion of hypertensive persons who have their high blood pressure under control more than doubled from the early 1970's to 1980 (Subcommittee 1985). The significant decrease in cardiovascular disease deaths and disability that has occurred since the 1970's is believed by many experts to be due to the increased detection and treatment of high blood pressure.

This success in the control of hypertension is generally credited to a combination of improved detection and the use of antihypertensive medication. However, the implications of long-term drug therapy for millions of Americans are unknown. There are documented side effects of the antihypertensive drugs. Thiazide diuretics, for example, can induce short-term increases in serum cholesterol, low density lipoproteins (LDL), and triglyceride levels in some persons. Some studies suggest that these effects

decrease or disappear with long-term therapy, although some clinical trials have shown persistence of the adverse effects (JNC IV 1988). Beta-blockers tend to lower high density lipoprotein (HDL) levels. These and other risks of drug therapy call attention to the potential benefits of nonpharmaceutical treatment of high blood pressure (Kaplan 1985).

Currently, three nondrug methods—weight control, alcohol restriction, and sodium restriction—are recommended as part of the treatment for established hypertension (JNC IV 1988). These measures have also gained support as likely to aid in the prevention of high blood pressure, particularly for those at high risk.

Historical Perspective

The first successful dietary treatment of human hypertension has been attributed to Kempner's rice-fruit diet that provided 20 g of protein, less than 5 g of fat, 150 mg of sodium, 200 mg of chloride, and 3,000 mg of potassium per day (Kempner 1944). Other studies confirmed that a very low sodium intake, for example, 200 mg/day, was effective in lowering blood pressure, although the effect on blood pressure of a moderate sodium intake, for example, 2,000 mg/day, was inconsistent (Watkin et al. 1950). Such studies laid the groundwork for subsequent investigations into the role of sodium in the development of hypertension.

The positive association between body weight and blood pressure was documented more than 60 years ago (Faber 1924). Clinical treatment of hypertension with weight reduction was reported over 60 years ago by Rose, who documented lower blood pressure and relief of edema in obese patients who lost weight (Rose 1922). The historical evidence for dietary associations with high blood pressure has been reviewed extensively (McCarron and Kotchen 1983; McCarron, Filer, and Van Itallie 1982; Horan et al. 1985).

A review of the development of U.S. public health policy on nutrition and hypertension indicates that in a rather brief time period, the focus shifted from acceptance of the association between sodium intake and blood pressure—an emphasis of the 1969 White House Conference on Food, Nutrition, and Health—to congressional attention on the topic of sodium labeling and FDA action to promote sodium labeling to the 1980 issuance of the DHHS/USDA recommendation in the *Dietary Guidelines for Ameri-*

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means to avoid excessive sodium intake. The relatively fast pace of this nutrition policy development can be appreciated by contrasting it with the history of recommendations for dietary change for high blood cholesterol levels (see chapter on coronary heart disease). A few milestones in the development of nutrition-hypertension policy are noted here.

The role of dietary salt in hypertension was a major issue at the White House Conference on Food, Nutrition, and Health and at the Senate Select Committee hearings in 1969 (Mayer 1969). Subsequently, a committee of the National Academy of Sciences recommended that no more than 0.25 percent salt be added to the commercial preparation of infant food (Subcommittee 1970). In 1970, the infant food industry initiated restriction of added salt.

In 1974, the American Academy of Pediatrics Committee on Nutrition recommended dietary modification of sodium intake for the pediatric population at risk for hypertension. The committee also favored the development of guidelines for reducing the use of salt by food processors and recommended that information about the amount of salt added to processed food be made available to consumers (AAP Committee on Nutrition 1974).

The Food and Drug Administration (FDA) sponsored a review of the health implications of added salt as part of an evaluation of substances designated "Generally Recognized As Safe" for use in foods. This evaluation, completed in 1979 by a committee of the Federation of American Societies for Experimental Biology, concluded that consumption of sodium chloride in the United States should be reduced, guidelines should be developed for restricting salt in processed foods, and the sodium content of processed foods should be labeled (Select Committee on GRAS Substances 1979). Subsequently, the FDA proposed that more information on the sodium content of foods be provided as part of nutrition labeling (U.S. Congress 1981; FDA 1982).

Sodium labeling is part of the FDA's five-point sodium program that involves collaboration between the food industry and Government. The effort intends to achieve changes in food labeling and to encourage the food industry to reduce the amount of sodium added to processed foods and to market a greater variety of foods with lowered sodium. The FDA will encourage consumer education about the relationship between sodium and

hypertension. It is intended to monitor changes in the marketplace to see whether efforts to reduce sodium in the food supply and to increase sodium labeling are successful. Legislation to mandate sodium labeling might be considered if voluntary efforts fail (Forbes and Stephenson 1985).

Appropriate caloric intake to maintain desirable weight and avoidance of excessive sodium intake are goals included in the *Surgeon General's Report on Health Promotion and Disease Prevention* (DHEW 1979) and the nutrition component of the 1990 *Objectives for the Nation* (DHHS 1980). These goals are also recommended by the Food and Nutrition Board of the National Academy of Sciences (NRC 1980) and are consistent with the *Dietary Guidelines for Americans*, published by the Departments of Agriculture and Health and Human Services (USDA/DHHS 1980, 1985).

In recognition of the clear social need to reduce illness, disability, and death from uncontrolled hypertension, the National High Blood Pressure Education Program (NHBPEP) was initiated in 1972, led and coordinated by the National Heart, Lung, and Blood Institute. This program includes an extensive network of Federal agencies and major national health organizations. During the time the NHBPEP has been in operation, age-adjusted death rates for stroke and coronary heart disease have substantially declined. The activities of the NHBPEP on improving hypertension control are believed to have contributed to this decline (Lenfant and Roccella 1984).

Significance for Public Health

In the United States, hypertension is a public health problem of enormous magnitude. Estimates of the prevalence of hypertension vary, however, depending upon differences in interpretation and extrapolation of the data. Current definitions of hypertension are listed in Table 3-1. A change in the blood pressure threshold from 160/95 mm Hg (systolic/diastolic measurements in millimeters of mercury) to 140/90 mm Hg was recommended in 1984 (JNC III 1984) and is maintained in the most recent report (JNC IV 1988). According to this definition, almost 58 million individuals have been found to have elevated blood pressure or have reported taking antihypertensive drugs prescribed by a physician (Table 3-2). About 39 million of these people are under the age of 65; less than 3 percent are children. Prevalence of hypertension increases with age in the U.S. population and is higher for black Americans (38 percent) than for white Americans (29 percent) (Subcommittee 1985).

Table 3-1
Classification of Blood Pressure^a in Adults 18 Years or Older

Range, mm Hg	Category ^b
Diastolic	
< 85	Normal blood pressure
85–89	High normal blood pressure
90–104	Mild hypertension
105–114	Moderate hypertension
> 115	Severe hypertension
Systolic, when diastolic blood pressure is < 90	
< 140	Normal blood pressure
140–159	Borderline isolated systolic hypertension
> 160	Isolated systolic hypertension

^aBased on the average of two or more readings on two or more occasions.

^bA classification of borderline isolated systolic hypertension (SBP 140 to 159 mm Hg) or isolated systolic hypertension (SBP > 160 mm Hg) takes precedence over high normal blood pressure (diastolic blood pressure 85 to 89 mm Hg) when both occur in the same person. High normal blood pressure (DBP 85 to 89 mm Hg) takes precedence over a classification of normal blood pressure (SBP < 140 mm Hg) when both occur in the same person.

Source: 1988 Joint National Committee.

Table 3-2
Estimated Prevalence of Cardiovascular Disease in the United States

Hypertension (> 140/90 or on Rx)	57,700,000
Rheumatic fever with or without heart disease	1,500,000
Coronary heart disease	6,700,000
Cardiac arrhythmias	1,400,000
Cerebrovascular disease	2,700,000

Sources: Subcommittee on Definition and Prevalence of the 1984 Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure 1985; National Center for Health Statistics 1986.

Hypertension is a major risk factor both for heart disease, which is the leading category of causes of death in the United States, and for stroke.

which is the third most frequent cause of death. All of the cardiovascular diseases are highly prevalent in this country (Table 3-2). A high proportion of end-stage renal failure is due to hypertension. The magnitude of the high blood pressure problem is demonstrated by the fact that in 1980 and 1981, the estimated annual number of visits to physicians' offices by patients with cardiovascular disease was over 56 million. The largest subgroup was for hypertension, a total of 29 million visits (Ezzati and McLemore 1980; NCHS 1984).

Scientific Background

Regulation of Blood Pressure

Blood pressure is regulated by a complex process involving the interactions of multiple factors that are not completely understood. Ultimately, the regulation of blood pressure reflects the interaction of cardiac output (the amount of blood the heart pumps per unit of time) and total peripheral resistance (the resistance to flow that blood encounters in the arteries and arterioles). Therefore, hypertension is an imbalance of the mechanisms that affect either output or resistance, or both. Some of the many mechanisms that affect these functions and, therefore, blood pressure are listed in Table 3-3. These include alterations in hemodynamic factors (plasma volume, cardiac output, and arterial pressure); central nervous system mechanisms that influence the hemodynamic mechanisms; interactions of the renin-angiotensin and adrenergic system that elevate arterial pressure; adrenal hormone secretion and maintenance of water, sodium, and other electrolyte balance; and various other hormonal influences.

In over 95 percent of individuals with high blood pressure, the specific cause cannot be determined, and the condition is referred to as primary or essential hypertension. Primary hypertension may sometimes represent nonspecific disturbances in blood pressure regulation, and the specific mechanism is not always identifiable.

Methodological Issues

Each approach to investigating the effects of diet on blood pressure has limitations. Animal experimentation provides optimal control over intake of specific nutrients, and hypotheses generated from animal models have been useful in the study of human primary hypertension. Different mechanisms, however, may be important in humans. Epidemiologic studies that compare customary diets and average blood pressure levels among populations or among smaller groups within populations can lead to inferences about the relative importance of various nutritional factors, particularly if

Table 3-3
Control Mechanisms for Arterial Pressure

Mechanical (posture, etc.)
Hemodynamic
Autonomic nervous system
Central nervous system
Gastrointestinal system (absorption of fluids and electrolytes)
Renal parenchymal function
Maintenance of sodium and fluid balance
Regulation of excretion of other electrolytes
Renin-angiotensin-aldosterone system
Other hormonal factors
Adrenal cortical hormones
Vasopressin
Growth hormone
Parathormone
Thyroid hormone
Kallikrein-kinin system
Prostaglandin system
Histaminergic mechanisms

Source: Adapted from Frohlich 1983.

data are collected longitudinally. Cross-sectional studies have two key drawbacks: (1) only a few observations may be used to characterize factors that often have large individual fluctuations (e.g., diet, blood pressure), and (2) they do not consider whether the observed dietary intake is typical of long-term habits, reflects an aberration in usual intake, or reflects a dietary pattern that has been recently modified in response to health concerns.

Observational studies cannot distinguish between the effects of highly correlated dietary constituents. For example, in an observational study, both a low intake of potassium and a high intake of sodium may be associated with high blood pressure. A well-designed human intervention study can yield information on the independence or interaction of the nutrients. Such studies require reliable dietary assessment and use of objective measures whenever feasible as well as standardized blinded blood pressure measurements, adequate length of followup, and control of potentially confounding variables such as other nutrients or weight change.

Nutritional Correlates

Research on diet and hypertension is complicated by the need to assess the relative importance of about 50 essential nutrients, dietary fiber, and nutrition-related factors such as obesity in the face of serious methodological problems inherent in studies of the role of diet in chronic disease (Reed et al. 1985). Although certain individual nutrients are implicated in hypertension, future nutrition research may need to focus more on the interrelationships among dietary factors than to consider each separately (Hegsted 1985).

Several symposia have reviewed the role of nutrition in blood pressure regulation as well as the mechanisms by which dietary factors are known or thought to influence blood pressure regulation (McCarron, Filer, and Van Itallie 1982; Horan et al. 1985). The major dietary factors thought to influence blood pressure and the mechanisms by which they may do so are summarized in Table 3-4. The complexity of these interactions explains why “. . . even where there is general agreement about the importance of a specific nutrient's effect on blood pressure, there is not necessarily consensus on the mechanisms involved” (McCarron, Filer, and Van Itallie 1982).

Key Scientific Issues

- Role of Obesity in Hypertension
- Role of Sodium in Hypertension
- Role of Alcohol in Hypertension
- Role of Other Minerals in Hypertension
- Role of Macronutrients in Hypertension
- Role of Caffeine in Hypertension

Of the many dietary factors listed in Table 3-4 that affect blood pressure, three—obesity, sodium, and alcohol—have a role that is well supported by scientific evidence (JNC IV 1988). At present, research on the effects of other dietary factors is suggestive but not conclusive.

Role of Obesity in Hypertension

As was noted in a special supplement on nutrition and blood pressure control (Dustan 1983; Havlik et al. 1983), early studies on the association between body weight and blood pressure have been confirmed in epidemiologic studies of primitive as well as developed populations. In many

Table 3-4
Major Nutrients and Possible Mechanisms
for Influencing Blood Pressure

<u>Calories and Macronutrients</u>	<u>Possible Mechanisms</u>
Total calories	Obesity Energy generation
Carbohydrates (and alcohol)	Energy metabolism Membrane synthesis Insulin regulation—sodium excretion Catecholamine regulation—vascular tone
Proteins	Protein/peptide synthesis Control of cellular function Membrane transport systems
Lipids	Energy source Cell membrane components Prostaglandin synthesis
<u>Electrolytes and Minerals</u>	
Sodium	Intravascular volume Hormone regulation Membrane potential
Potassium	Vascular tone Hormone regulation Cation transport
Calcium	Receptor-ligand binding Hormone synthesis/release Vascular tone Contractile protein interactions
Magnesium	Regulation of calcium channels ATP production Contractile protein interaction
Phosphorus	Membrane structure ATP-energy metabolism cAMP component
Trace elements	Copper-vascular integrity Manganese-energy metabolism Chromium-lipid metabolism Vanadium-sodium/potassium ATPase

Source: Adapted from McCarron, Henry, and Morris 1982.

populations where body weight does not increase with age, neither does blood pressure. Further connection between hypertension and obesity has been demonstrated in the Hypertension Detection and Follow-Up Program, which reported that 60 percent of the participants with hypertension were more than 20 percent overweight (Hypertension Detection and Follow-Up Program Cooperative Group 1977).

Evidence for the effect of weight reduction on blood pressure began to accumulate in the early part of this century. This association has now been investigated in many epidemiologic studies and several clinical trials (as reviewed by MacMahon et al. 1987). Although a few studies have reported that treatment of hypertension with weight loss did not result in lower blood pressure, many investigators have reported significant reduction of elevated blood pressure by weight loss (Table 3-5).

Complicating the relationship of obesity to blood pressure is the role of sodium. An early hypothesis was that obese individuals with hypertension are sensitive to the blood pressure-raising effects of a high sodium intake associated with long-term calorie excess (Dahl, Silver, and Christie 1958). However, other investigators have dissociated the two factors and demonstrated that weight loss is effective in lowering blood pressure even in the absence of sodium restriction (Reisen et al. 1981; Maxwell et al. 1984).

In conclusion, increased body weight is related to increased blood pressure. Furthermore, a fall in blood pressure can be expected with weight reduction. Further studies may help to define other factors such as the distribution of body fat (Kalkhoff et al. 1983; Krotkiewski et al. 1983; Berchtold 1985) or specific mechanisms by which obesity might be involved in the development of hypertension.

Weight loss is recommended for all overweight persons, particularly for those with hypertension. It has been suggested that control of obesity would eliminate hypertension in 48 percent of whites and 28 percent of blacks (Tyroler, Heyden, and Hames 1975). Even when weight loss does not reduce blood pressure to normal, health risks may be reduced, and smaller doses of antihypertensive medication may be needed as a result.

Role of Sodium in Hypertension

Definitions

In most human studies, sodium intake is estimated from reported dietary intake of salt (sodium chloride) among the study participants. A more

Table 3-5
Changes in Weight and Blood Pressure (Baseline to Followup) in Treatment (Rx)
and Control Groups of Five Randomized Controlled Trials

	N		Followup (months)	Weight Change (kg)		Systolic BP Change		Diastolic BP Change	
	Rx	Control		Rx	Control	Rx	Control	Rx	Control
Diet trials									
Reisin et al.	57	26	4	-14.9	-1.2	-37.4	-6.9	-23.3	-2.5
Heyden et al.	63	64	12	-8.1	-1.9	-18.0	-12.0	-13.0	-8.0
Ramsay et al.	15	34	12	-5.1	-2.4	-11.9	-8.9	-6.9	-4.4
Haynes et al.	30	30	6	-4.1	-0.8	+4.8	-0.2	+1.4	-0.1
MacMahon et al.	20	18	5	-7.4	+0.5	-13.3	-7.4	-9.8	-3.1
Pooled estimates ^a (Rx vs. control)									
Diet trials	185	172		-9.2		-6.3		-3.1	
(95% confidence limits)				(-8.2, -10.2)		(-3.3, -9.4)		(-1.5, -4.7)	
Pooled estimates ^a (Rx vs. control)									
All trials	336	254		-8.7		-5.3		-3.3	
(95% confidence limits)				(-7.9, -9.5)		(-3.4, -7.3)		(-1.8, -4.7)	

^aSee MacMahon et al. 1987 for methods.

Source: Adapted from MacMahon et al. 1987.

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precise measure of sodium intake is 24-hour urinary sodium output, considered to be an accurate reflection of recent dietary sodium intake in persons with normal kidney function. An average urinary sodium output can be estimated to be about 150 mEq/24 hours. Dietary sodium intakes are commonly reported in mEq, mM, or g. One mEq or one mM of sodium equals 23 mg, and 1 g of sodium equals 44 mEq or mM and is equivalent to 2.5 g of sodium chloride (table salt). The average daily sodium intake of adults is about 4 to 6 g (175 to 265 mEq).

Research Evidence

Epidemiology. A relationship of sodium to hypertension is supported by several lines of evidence. In non-Western populations with low salt consumption, blood pressure does not rise with age. Populations with low blood pressure generally do not consume much salt. These associations have been reported among numerous populations such as the Bushmen of Kalahari, Indian tribes in Brazil, and other groups in New Guinea, Malaysia, the Polynesian Islands, and Solomon Islands.

Salt intake is positively correlated with average systolic and diastolic blood pressures in samples of men and women from 25 diverse populations, regardless of methods of measurement (Gleibermann 1973; McCarron, Henry, and Morris 1982); significant positive associations have not been observed in other populations (see review, Subcommittee 1986). Associations among individuals within a population have been less consistent, perhaps for methodological reasons (Liu et al. 1979).

Clinical Studies. Many studies have examined the effect of moderate restriction of sodium consumption on blood pressure in adults. One non-randomized crossover study, for example, found that a diet considered to be moderate in sodium restriction—that is, “avoiding all foods which had sodium added during preparation”—reduced sodium excretion from 191 to 93 mEq and was accompanied by reductions in systolic and diastolic blood pressure that averaged 7.7 and 4.4 mm Hg, respectively (Parijs et al. 1973).

A randomized study of 37 hypertensive patients with initial diastolic blood pressures of 90 to 110 mm Hg reported that dietary restriction to 50 mEq of sodium/day for 15 to 21 months produced reductions in systolic and diastolic blood pressure that were similar to those achieved by drug treatment (Magnani et al. 1976). Long-term studies have shown that 39 percent of hypertensive patients could control blood pressure with sodium intakes below 50 mEq/day and close supervision by a dietitian and physician, and about one-third of patients with mild hypertension could control blood pressure with a sodium intake of 75 mEq/day or less (Hunt 1977).