Dietary Electrolytes and Blood Pressure
A Statement for Healthcare Professionals From the American Heart Association Nutrition Committee

Theodore A. Kotchen, MD; David A. McCarron, MD; for the Nutrition Committee

Blood pressure–associated risks ensue incrementally over a wide range of blood pressure levels, and even among nonhypertensive persons, blood pressure levels are predictive of morbidity and mortality from stroke, heart disease, and end-stage renal disease.1-3 On a population basis, it has been estimated that a reduction in diastolic blood pressure of 2 mm Hg would result in a 15% reduction in risk of stroke and transient ischemic attacks and a 6% reduction in risk of coronary heart disease.4

Between 1971 and 1991, national health examination surveys5 documented a downward trend in blood pressure levels and the prevalence of hypertension in the United States. Adoption of a healthier lifestyle may have contributed to this favorable trend. Not all subgroups have benefited equally, however, particularly African Americans. According to the Third National Health and Nutrition Examination Survey (NHANES III)6 (1988 to 1991), 24% of the US population was classified as having hypertension. Blood pressure level and hypertension prevalence increase with age: high-normal and high blood pressure continue to be major contributors to cardiovascular disease.7

The relationship between dietary electrolyte consumption and blood pressure is the focus of this brief review. Evidence for a positive association between sodium chloride (NaCl) intake and blood pressure is discussed. Increasing evidence also suggests that dietary patterns associated with low intakes of potassium, calcium, and possibly magnesium also contribute to higher levels of blood pressure. An understanding of these associations has important implications not only for the prevention and treatment of hypertension but also for developing population-based strategies to decrease cardiovascular disease risk by shifting the overall blood pressure distribution toward lower levels.

Sodium Chloride
A high NaCl intake convincingly contributes to elevated arterial pressure in a number of animal models of genetic and acquired hypertension. The chimpanzee is phylogenetically close to the human, and it has recently been demonstrated that the addition of NaCl (5, 10, and then 15 g/d) to the chimpanzee’s usual diet (a fruit-and-vegetable diet low in NaCl and high in potassium) over 20 months results in significant and progressive elevations of blood pressure.8 After 84 weeks of added NaCl, relative to both baseline values and a control group, mean systolic and diastolic blood pressures increased by 33 mm Hg and 10 mm Hg, respectively. This increase was completely reversed within 6 months of cessation of the high NaCl intake. Animal studies (as well as limited epidemiological and clinical observations) suggest that diets high in NaCl may also have deleterious cardiovascular consequences independent of blood pressure, eg, cerebral arterial disease and stroke, left ventricular hypertrophy, renal vascular disease, and glomerular injury.9

In humans, evidence for an association between NaCl consumption and blood pressure is based on anthropological research, observational epidemiological studies (cross-population and within-population), and intervention trials.10-13 The strength of the association of NaCl intake with blood pressure increases with age, blood pressure level, and among individuals with a family history of hypertension.14 In addition, the full expression of NaCl-sensitive hypertension depends on the concomitant intake of both sodium and chloride.15 In both experimental models and humans, blood pressure is not increased by a high dietary sodium intake provided as nonchloride salts of sodium. In the usual diet, however, most sodium is consumed as NaCl,16 largely from its use as an additive in commercial food processing.17,18

Across populations, level of blood pressure, increment in blood pressure level with age, and prevalence of hypertension are related to NaCl intake. The International Study of Salt and Blood Pressure (INTERSALT)10,19,20 is a cross-sectional study designed to evaluate both within-population and cross-population hypotheses on the relationship between blood pressure and sodium excretion in >10 000 adults (aged 20 to 59 years) at 52 centers around the world. Across the 52 populations, 24-hour sodium excretion was significantly related to median systolic and diastolic blood pressure, the upward slope of systolic and diastolic blood pressure with age, and the prevalence of high blood pressure. For individuals, the within-population analyses demonstrated that the relationship between sodium excretion and blood pressure was similar for nonhypertensive and all participants, indicating that varying degrees of salt sensitivity of blood pressure occur throughout the population. Overall the principal observations in INTERSALT are that (1) for individuals, a difference of 100 mEq (equivalent to 5.9 g NaCl) per day in sodium
intake is associated on average with a difference of 3 to 6 mm Hg in systolic blood pressure; and (2) for populations, a 100 mEq/d lower sodium intake is associated with attenuation of the rise in systolic blood pressure by 10 mm Hg in persons aged 25 to 55 years. As with observations in several other isolated, preliterate populations, in 4 remote INTER-SALT samples both sodium excretion and blood pressure were low, there was little or no upward slope of blood pressure with age, and there was little or no hypertension.

A recent review20 of a limited number of observational and intervention studies suggests that there also is an association between NaCl consumption and blood pressure level in children and adolescents. In a randomized, double-blind trial conducted in 1980 among 476 newborn Dutch infants, blood pressure was lower in infants fed a diet low in NaCl than in infants fed a diet with normal NaCl intake during the first 6 months of life.22 In a cohort of these same subjects at 15-year follow-up, adjusted systolic blood pressure was lower in children who had been assigned in infancy to the low-NaCl group compared with the control group.23

On the basis of results of acute NaCl depletion or loading protocols, depending on arbitrary definitions of NaCl sensitivity, it has been estimated that approximately 30% to 50% of hypertensive persons and a smaller percentage of nonhypertensive persons are NaCl sensitive, ie, arterial pressure is decreased by NaCl depletion and/or increased by NaCl loading.24,25 Blood pressure responses to acute protocols are reproducible.26 In 40 nonhypertensive and hypertensive subjects, it has been reported that blood pressure response to an acute volume expansion–volume contraction protocol correlates (r = .40, P < .01) with the change in blood pressure in response to a low-sodium diet.27 Nevertheless, the arbitrary designation of salt sensitivity based on blood pressure responses to acute protocols in a limited number of subjects may not reflect long-term blood pressure responses to dietary NaCl in a population. Such a designation of salt sensitivity, however, does highlight the heterogeneity of response to acute changes in NaCl balance, although intervention studies have not identified a bimodal blood pressure response to NaCl reduction.28 On a population basis, the average change in blood pressure in response to NaCl reduction may be more meaningful than identification of “salt-sensitive” individuals.

Randomized, controlled trials provide compelling evidence for a causal relationship between dietary NaCl and blood pressure. Despite reservations about the limitations of meta-analyses (including criteria for inclusion and exclusion of trials, different study designs and approaches to data analysis among individual trials, and variable documentation of adherence to study diets), 2 recent meta-analyses document consistent reductions in blood pressure in response to lowered intake of NaCl.29,29 In 1 meta-analysis of 32 trials, estimated median reductions of 24-hour urine sodium excretion across trials were 76 mmol (range 56 to 105) and 106 mmol (range 60 to 210) in hypertensive subjects and nonhypertensive subjects, respectively; overall reductions of systolic and diastolic blood pressure were –1.9/–1.1 mm Hg in nonhypertensive subjects and –4.8/–2.5 mm Hg in hypertensive subjects.26 In a second meta-analysis of 52 trials, estimated median reductions of sodium excretion across trials were 79 mmol (range 71 to 119) for hypertensive subjects and 133 mmol (range 95 to 156) for nonhypertensive subjects; overall reductions of blood pressure were –1.6/–0.5 mm Hg in nonhypertensive subjects and –5.9/–3.8 mm Hg in hypertensive subjects.28 The reductions in blood pressure by a diet lower in NaCl are more prominent in hypertensive than in nonhypertensive individuals, and greater reductions of blood pressure have been observed in trials lasting ≥5 weeks than in shorter trials.29 Although the blood pressure reductions in these short-term trials are relatively modest for individuals, it is estimated that lowering a population’s blood pressure to this degree would translate into a significant reduction in cardiovascular disease mortality.

The Trials of Hypertension Prevention (TOHP) evaluated the effects of nonpharmacological therapy on blood pressure in adults with high-normal blood pressure. In phase I, systolic and diastolic blood pressure were significantly reduced in separate groups treated with weight loss or reduction of NaCl intake over 18 months but not by dietary supplementation with calcium, magnesium, or potassium for 6 months.30,31 The results of phase I were the basis for phase II of TOHP, which more extensively evaluated the effects of weight loss and reduced NaCl intake, alone and in combination, on blood pressure over 3 to 4 years in overweight adults with high-normal blood pressure.32 Compared with blood pressures in a usual-care control group, at 6 months, systolic and diastolic blood pressure were both significantly reduced by weight loss alone (–6.0/–5.5 mm Hg) and lowered NaCl intake alone (–5.1/–4.4 mm Hg), although the effects of the 2 interventions on average blood pressure reduction were not additive. At 6 months, hypertension incidence was 7.3% in the usual-care group, 4.2% in the weight loss group, 4.5% in the reduced-NaCl group, and 2.7% in the combined weight loss–reduced-NaCl group. Beyond 6 months, the interventions were less effective for maintaining both weight loss and lowered NaCl intake, and impact on blood pressure was lessened. At termination of the study there were small but significant reductions in systolic blood pressure in the weight loss and low-NaCl group, whereas reduction of systolic blood pressure in the combined-intervention group (weight loss and low NaCl) did not achieve statistical significance. Reduction of diastolic blood pressure was significant only in the weight loss group. The incidence of hypertension during the entire course of the study was significantly lower in each of the 3 intervention groups (approximately 38%) than in the usual-care group (44%).

Experimental models of hypertension and increasing information on humans provide convincing evidence for genetic susceptibility and resistance to the effects of dietary NaCl on arterial pressure.33,34 In the United States, a larger proportion of both nonhypertensive and hypertensive African Americans are NaCl sensitive compared with non-Hispanic whites.35 In response to acute NaCl loads, African Americans excrete sodium less efficiently than whites, and it has been estimated that >50% of hypertensive African Americans in the United States are NaCl sensitive. The prevalence of diuretic-sensitive (and presumably NaCl-sensitive) blood pressures in African Americans approaches 75%.36 Clinically there is evidence for heritability of sodium excretion, levels of hormones that
Potassium, Calcium, and Magnesium

Observational studies document inverse associations of blood pressure with dietary potassium, calcium, and magnesium consumption. On the basis of these observations, however, it is difficult to relate blood pressure levels to specific nutrients because of strong correlations among dietary intakes of potassium, magnesium, fiber, and, to a lesser extent, calcium. Furthermore, an inverse relationship between calcium intake and blood pressure has not been observed in all studies, and a recent critical review has highlighted methodological issues that complicate interpretation across these studies. The inverse relationship between calcium intake and blood pressure is more convincing at low levels of calcium consumption, ie, 300 to 600 mg/d.

The impact of dietary NaCl on blood pressure may be affected by consumption of potassium or calcium. The urine sodium-potassium ratio is a stronger correlate of blood pressure than either sodium or potassium alone. In addition, high sodium intake is associated with higher blood pressure levels among persons consuming low-calcium diets.

Results of 2 recent meta-analyses of clinical trials support the conclusion that oral potassium supplements (60 to 120 mEq/d) lower blood pressure. The magnitude of the blood pressure–lowering effect is greater in hypertensive than in nonhypertensive persons and more pronounced in persons consuming a diet high in NaCl. In an analysis of 19 trials of hypertensive subjects, blood pressure was lowered on average 2.8/−4.5 mm Hg. In a separate analysis of 33 trials, blood pressure was lowered on average by 1.8/−1.0 mm Hg in nonhypertensive subjects and −4.4/−2.5 mm Hg in hypertensive subjects.

Two meta-analyses of controlled clinical trials have shown that calcium supplementation (1000 to 2000 mg/d) results in small but significant reductions of systolic (−4.3 and −1.7 mm Hg, respectively) but not diastolic blood pressure, and only in hypertensive individuals. Calcium supplementation may preferentially lower blood pressure in patients with NaCl-sensitive hypertension. Contrast to earlier evidence of a benefit of calcium supplementation in high-risk pregnancy, results of a recent randomized multicenter trial indicate that calcium supplementation during pregnancy does not prevent preeclampsia or pregnancy-associated hypertension in healthy nulliparous women.

The overall antihypertensive response to magnesium supplementation among hypertensive individuals is small, and several trials have failed to show a significant effect of magnesium supplementation on blood pressure. One placebo-controlled, 6-month trial in hypertensive women found that magnesium supplementation (20 mmol/d) significantly lowered diastolic (−3.4 mm Hg) but not systolic blood pressure.

Dietary Approaches to Stop Hypertension Trial

Persons consuming vegetarian diets tend to have lower blood pressure than nonvegetarians. For Americans, fruits and vegetables are a main source of potassium, magnesium, and fiber, and dairy products are a main source of calcium. In a randomized, multicenter study, the Dietary Approaches to Stop Hypertension (DASH) trial evaluated the effects on blood pressure of 3 dietary patterns over 8 weeks in 459 adults with high-normal blood pressure or mild hypertension. The dietary interventions were (1) a control diet with potassium, calcium, and magnesium levels close to the 25th percentile of US consumption; (2) a diet rich in fruits and vegetables; and (3) a “combination” diet rich in fruits, vegetables, and fat-free or low-fat dairy products.

The study diets were relatively high in potassium, calcium, and magnesium content. Compared with the control diet, the study diets also were higher in fiber, protein, carotenoid, and folate, and lower in total fat, saturated fat, and cholesterol. NaCl content was equivalent in all 3 diets (7.5 g/d). Caloric intake was adjusted so that weight change was avoided. Persons consuming >14 alcoholic beverages per week were excluded from the study.

Systolic and diastolic blood pressure were significantly reduced by the diet enriched with fruits and vegetables (−2.8/−1.1 mm Hg) and, compared with the control diet, were reduced to an even greater extent by the combination diet (−5.5/−3.0 mm Hg). The greater effect of the combination diet was manifest in both hypertensive (−11.4/−5.5 mm Hg) and nonhypertensive (−3.5/−2.1 mm Hg) persons, although reductions in blood pressure were more pronounced in hypertensive persons. Blood pressure reductions were similar in men and women and across ethnic groups.

Although not designed to identify the effective nutrients of the diets, the DASH trial convincingly reaffirms the importance of multiple factors in the diet for blood pressure control.

Conclusion

The beneficial effects of diet on blood pressure can be maximized by avoiding high intake of NaCl and ensuring adequate intake of fruits, vegetables, and fat-free and low-fat dairy products. Such diets are rich in potassium, calcium, magnesium, and protein and low in total fat, saturated fat, and cholesterol. Although not discussed here, additional nutritional strategies for optimizing the effect of diet on blood pressure are prevention and treatment of obesity, and for people who drink alcohol, avoiding consumption of >2 drinks per day.

Any population-based guideline for an upper limit of NaCl is arbitrary and should represent a reduction that is acceptable and safe. For the general population, the AHA recommends that the average daily consumption of NaCl by adults not exceed 6 g. This recommendation is consistent with the guidelines of a number of other agencies in both the United
States and abroad. 20,25 Although total cholesterol and LDL cholesterol may be slightly increased by short-term “excessive sodium reduction,” 28 there is no evidence that limiting NaCl consumption to 6 g per day poses any health risk. A lower NaCl intake may be recommended for hypertensive persons. Although there is insufficient evidence to recommend high intake of calcium for prevention or treatment of hypertension, calcium deficiency should be avoided. Primarily for prevention of osteoporosis, the National Academy of Sciences recently recommended the following calcium intakes as adequate: 1300 mg/d for adolescents (9 to 18 years); 1000 mg/d for persons aged 19 to 50 years; and 1200 mg/d for persons older than 50. It remains to be determined whether lowering blood pressure–lowering capacity of fruits, vegetables, and low-fat dairy products can be explained entirely by their electrolyte content.

References


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