Reduction of Dietary Sodium in Western Society
Benefit or Risk?

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THERE is widespread interest in relationships between dietary electrolyte intake and arterial pressure. Attention has been directed to potassium, calcium, and magnesium, but the predominant discussion has been of the possible role of dietary sodium in determining the level of blood pressure in Western society. Experts viewing information from animal research, from intra- and interpopulation studies, and from manipulations of sodium intake in hypertensive and normotensive individuals have joined two opposing camps. Dialogue between the two has often been heated. In one camp are the "enthusiasts" who consider the evidence sufficient for encouraging Western society as a whole to restrict dietary sodium intake. Such dietary change would, they feel, lower arterial pressure overall, prevent the onset of hypertension in many, and most likely reduce the complications of high blood pressure. Unlike antihypertensive drug therapy, diet modification would have few if any undesirable effects; thus, for those in whom no benefit was seen at least no harm would result. The contrary camp houses the "skeptics." They claim the evidence is insufficient to justify attempts at lowering dietary sodium for the whole of Western society or even for all mild hypertensive patients. Skeptics emphasize that reports are conflicting on relationships between the level of sodium intake and blood pressure in both animals and humans; that the effects of short-term reductions in dietary sodium on arterial pressure in humans are disputed; that there is little information on the effects of sodium restriction over prolonged periods; and that there is no evidence that the complications of hypertension can be avoided by diet modification. Further, the skeptics are unwilling to accept that dietary changes may necessarily be harmless.

My aim is to look at current evidence together with the views of enthusiasts and skeptics and to reach a final, but personal, conclusion as to whether we are sufficiently informed to advise a reduction in dietary sodium of a realistic nature (to around 80 mmol/day) for Western society. Before pursuing this course, however, I should make some observations. First, the literature is vast, and I shall not attempt here to quote all relevant studies. Second, there are special circumstances where moderate restriction of dietary sodium is of accepted benefit, in particular during drug treatment for hypertension.1 My present discussion addresses the advisability of limiting dietary sodium in whole populations. Third, the design of many studies relating sodium to blood pressure, especially those in humans, is open to criticism, and in some cases, no useful conclusion can be reached. To ignore the effects on blood pressure of factors such as season of the year, ambient temperature, sequence of dietary interventions, time of day, and so forth is to place a trial, no matter how expensive, in jeopardy. This is especially so since these and other confounding factors can have large effects on blood pressure, whereas changes due to minor manipulations of sodium intake may be relatively small. Even more surprising are the poor standards observed in techniques of blood pressure measurement. Few workers have made use of objective, accurate measuring devices; it is frequently unclear whether one or many in the research team have recorded pressures in the same patient; and some papers omit any mention whatsoever of measurement technique. Such basic, vital omissions make interpretation of results difficult or impossible.
Animal Models

Dahl et al.\(^5\) developed by selective inbreeding two strains of Sprague-Dawley rat, a salt-sensitive S strain that regularly became hypertensive on a high sodium intake and a salt-resistant R strain that remained normotensive. The blood pressure response characteristic could be transferred from the R to the S strain, or vice versa, by renal transplantation.\(^6\) These observations led to the concept that hypertension in these rats, and perhaps also in humans, required a genetic substrate transferable by the kidney and an environmental factor, such as a high sodium intake.

Can experimental information from a particular rat strain be extrapolated to humans? Some enthusiasts are convinced that it can be and that sodium restrictions in humans will, as in some rats, prevent hypertension and its complications. "Have faith in the animal model."\(^7\) Skeptics respond by asking "which animal model"? They point to randomly bred rats that show little or no change in blood pressure on high sodium diets.\(^8\) The New Zealand strain of genetically hypertensive rat is resistant to salt.\(^9\) Other rat models show increased pressure when placed on a low sodium intake\(^10\) or temporarily decreased pressure when intake of sodium is increased.\(^10\) Skeptics may also point out that the amount of sodium used by Dahl et al. was colossal and outside the circle of relevance to humans,\(^9\) that the degree of sodium restriction necessary to limit hypertension in some rats will simultaneously reduce the growth rate,\(^12\)\(^13\) and that chronic sodium restriction may not be without hazard, as, for example, during hemorrhage.\(^14\) Whether one is an enthusiast or a skeptic, it is clear that evidence from rats alone is an insufficient basis for altering the sodium content of food in Western society.

Interpopulation Studies in Humans

Although the methods used in assessing the intake of sodium and in measuring blood pressure might be questioned, there is little doubt that hypertension is absent or rare in societies with a lifelong low sodium intake, but that hypertension is common in those with a very high intake.\(^1\) A major point of uncertainty and disagreement between enthusiasts and skeptics here is whether dietary sodium, exclusive of factors such as obesity, race, physical fitness, "stress," and dietary factors other than sodium, can explain the differences in blood pressure. Further, if dietary sodium is an important determinant of blood pressure across populations, what is the level to which it must be reduced in Western society to achieve a useful lowering of pressure? Data from studies of different populations throughout the world support the cause of the enthusiasts somewhat, but do not help us to decide whether Western society as a whole should lower its sodium intake to a modest degree.

Intrapopulation Studies

Accurate assessment of dietary sodium intake is particularly difficult.\(^9\) With this limitation in mind, most workers have been unable to find, within populations, any relationship between sodium intake and blood pressure. Many reports can be quoted, but some are of particular note since they focus on subjects taking extremes of dietary sodium or those with extremes of arterial pressure. A study of 50-year-old men in Göteborg looked at relationships between blood pressure and urine sodium excretion in 19 untreated hypertensive patients and 19 normotensive subjects.\(^16\) In the former group, there was an inverse correlation between the 24-hour urine sodium excretion and diastolic pressure, whereas the relationship was positive in normotensive subjects. Overall, the association of urine sodium and blood pressure looked inverse. The Connecticut Blood Pressure Survey\(^17\) compared blood pressures between two groups of healthy adults — those whose dietary sodium intake (on questionnaire) was at or above the 90th percentile, and those at or below the 10th percentile. Overall, there was no difference in arterial pressure between the groups. Watt et al.\(^18\) measured blood pressure, 24-hour urine electrolyte excretion, and plasma renin activity (PRA) in the offspring of two groups of parents in a Welsh village — those in the top third of the distribution of blood pressure for age, and those in the bottom third. Although arterial pressure was higher in offspring of the former group, no difference existed in urine sodium excretion or in PRA between the groups. Enthusiasts can point to intrapopulation studies that report positive relationships between dietary sodium intake and arterial pressure.\(^19\)\(^–\)\(^21\) Skeptics may counter with data showing inverse correlations.\(^22\)\(^–\)\(^24\) Over-
all, then, most found no association between the level of sodium intake and blood pressure within populations. Had a positive relationship been observed in most instances, the case for the enthusiasts would have been strengthened considerably. Its absence, however, by no means defeats their case since confounding factors can be enlisted. For example, there may be wide individual variability in susceptibility to sodium; there could be a threshold of sodium intake above which little further effect from salt on blood pressure is apparent; the sodium-blood pressure association might exist only in infancy; since day-to-day variation of sodium excretion in an individual is high, a relationship with arterial pressure should not be expected. Nevertheless, intrapopulation studies alone are insufficient basis to advise change in the eating habits of Western society.

Effects of Major Change in Sodium Intake

No one doubts that extreme restriction of sodium intake (to less than 15 mmol/day) can lower blood pressure in many patients with severe hypertension and can reverse ECG abnormalities, retinopathy, proteinuria, and cardiomegaly in some. That sodium chloride is central to the hypotensive action of such Spartan diets was confirmed when hypertension redeveloped as NaCl was restored. From these earlier studies it was clear that sodium intake must be reduced to very low levels to achieve clearcut drops in blood pressure, and even then some patients remained severely hypertensive. Further, few patients could tolerate the diets for long, and they were impractical in the outpatient setting.

At the other end of the spectrum, gross sodium overloading increased blood pressure in normotensive volunteers, although the pressure rise was very small, at least in the short term for those without a family history of hypertension. Skeptics can point to studies of normotensive subjects that failed to report a rise in blood pressure during a high sodium intake.

Effects of Minor Change in Sodium Intake

Minor reductions in sodium intake (from around 180 to 80 mmol/day) might be acceptable to Western society if it can be shown clearly that benefits will result. The first step is to define whether blood pressure is indeed altered by such minor manipulations. Apart from the fact that deficiencies in protocol design and in techniques for measuring blood pressure are common, the most obvious void here is in long-term studies. Almost all information comes from brief periods of diet manipulation, and data can be quoted to favor arguments of enthusiasts or skeptics. For example, some workers report definite decreases in blood pressure in normotensive volunteers, but others have found little or no such effect. Those who have observed clearcut decreases in blood pressure studied more severe hypertension. An almost uniform finding has been that individuals exhibit very different responses to restriction of sodium intake. Whereas some patients have a sizable drop in pressure, others show no change, and a minority show a rise in blood pressure. The favored explanation for these different responses is that variable activation of the sympathetic nervous system or the renin-angiotension system occurs with sodium restriction and contributes to the final change in blood pressure.

If sodium restriction were to be effective in preventing hypertension, the appropriate diet would need, presumably, to be introduced early in life. Short-term studies have been carried out in infants and children, but the results are conflicting. For example, a careful double-blind, randomized study of newborn infants in Holland reported lower systolic pressures at 6 months in those who received a sodium intake that was one-third that of control infants. Other workers found little or no effect from altering sodium intake. Maybe diet modification must extend from infancy into adulthood before clearcut effects on blood pressure are seen. At the other end of the age scale, older subjects, either normotensive or hypertensive, apparently show a more uniform response to manipulations in dietary sodium. On this information alone, one might hope to find a useful blood-pressure-lowering effect of moderate sodium restriction in middle-aged and elderly hypertensive patients. There is little encouragement, however, to advise dietary change in younger subjects, with or without hypertension.
Risk of Restricting Dietary Sodium

Are there risks involved in restricting sodium intake to around 80 mmol/day in Western society? Enthusiasts claim it is most unlikely, and none have yet been shown convincingly. Skeptics point to animal studies which report that chronic sodium restriction interferes with neurohumoral defenses against accidental fluid loss. Some animal models actually increase (rather than decrease) blood pressure. Further, they may restate that modest sodium restriction actually increases arterial pressure in a minority of subjects and can adversely affect renal function. The most telling argument, say the skeptics, is that no one has, in a systemic objective prospective manner, looked for adverse effects from modest sodium restriction. Hence, it is possible that some subgroup of the population will be placed at risk by reduction of its sodium intake. The skeptics do not feel compelled to state what these disadvantages might be or who is likely to be affected. This, they claim, is a burden the enthusiasts must bear — to demonstrate lack of risk now or stand accused in the future when the dangers become apparent and counterbalance, or even outweigh, any benefits. The analogy with diuretic treatment for mild hypertension (benefit vs risk) is obvious.

Summary of the Evidence

There are inbred strains of rats with blood pressures that are sensitive to sodium intake, but other strains that show no response. Comparison of peoples from different societies shows a relationship between extremes of sodium intake and frequency of hypertension, but cause and effect are not proven. Intrapopulation studies have, in general, failed to find any association between sodium intake and blood pressure. Whereas massive reductions in dietary sodium can alter the level of arterial pressure in many severe hypertensive patients (and reverse complications in some), short-term modest diet changes have small and variable effects. The blood pressure of older patients responds more uniformly to sodium restriction than that of younger subjects. It remains to be shown whether long-term minor restrictions in dietary sodium lower blood pressure or prevent the development of hypertension. No studies have determined whether modest reductions in sodium intake lessen complications of hypertension. Although no definite disadvantages from long-term mild sodium restriction have been observed, none has been looked for in a systematic manner.

Confounding Factors and Unknowns

What level of sodium restriction is necessary for benefit to be observed? Is this level of salt intake feasible for all or most of Western society? Feasibility has been confirmed under a few circumstances and in some countries. Is it necessary to only limit the addition of salt to cooking and at the dinner table, or must our eating habits otherwise be changed to achieve the desired level of sodium? If the latter applies, we would be altering the intake of other food components such as calcium, potassium, magnesium, vitamins, fats, and fiber, all of which could have an effect on blood pressure. Is it perhaps more important to limit chloride rather than sodium? Since racial groups within Western society have different metabolic and hemodynamic characteristics and respond in different ways to electrolyte manipulations, should dietary advice be the same for all people? Older subjects are apparently more sodium-sensitive than youngsters, and body sodium content correlates with arterial pressure best in those with moderate hypertension or in older age groups. Should dietary modification therefore be limited only to middle-aged and older groups with moderate hypertension, or, with current knowledge, should it be initiated in infancy? Will the incidence of other disorders, such as gastrointestinal neoplasia, be altered by changes in diet? These questions are but an aliquot from a large reservoir of unknowns.

Personal Overview

There is every reason to limit sodium intake in most patients taking antihypertensive medications. A trial period of sodium restriction seems worthwhile for individuals with mild or moderate hypertension since they may be salt-sensitive and could thus avoid drug therapy. More research is needed to find methods of detecting salt-sensitive individuals.

But what should be the advice for Western society at large? It is an attractive concept that lifelong dietary sodium restriction will lower blood pressure, and the benefits from
such a "massive approach, in which everybody receives a small benefit, may be unexpectedly large."63 Available evidence, however, is insufficient to conclude that blood pressure overall will decline with long-term modest sodium restriction and that complications of hypertension will be reduced. Adverse effects from dietary modifications have not been sought — they could conceivably arise from limitation of sodium per se or from concurrent change in other dietary components. Quite simply, we know neither the benefits nor the dangers.

On the continuing and heated debate between enthusiasts and skeptics, Claude Bernard's thoughts might well be pondered. "When two physiologists or two doctors quarral, each to maintain his own ideas or theories, in the midst of their contradictory arguments, only one thing is absolutely certain: that both theories are insufficient and neither of them corresponds to the truth."64 Theory, in the absence of facts, can be sufficient basis for a particular form of treatment under special circumstances when risk to the individual is high. But this approach is hard to justify for the circumstances under discussion. My own view is that long-term studies of diet sodium manipulation in Western society will be hard pressed to demonstrate benefit over risk. Until we have this information, however, we simply continue a time-wasting controversy.

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