Salt Intake and Sympathetic Activity

To the Editor:

Grassi et al1 studied the effect of modest salt reduction on sympathetic activity in 27 hypertensive patients and concluded that this triggered a sympathetic activation and baroreflex impairment. These results need to be interpreted with caution. Firstly, the study was not randomized or double-blinded. Furthermore, 60% of patients (16 out of 27) originally recruited into the study were excluded from the analysis, a few because of unsatisfactory muscle sympathetic nerve traffic recording, but most because of poor compliance. However, no information was given about these patients. How far were they compliant? Was this based on urinary sodium? Additionally, no information whatsoever was given on how the reduction in salt intake was carried out. The decrease in body weight (2.3 kg) is much more than would be seen with a modest salt reduction. Therefore, there must have been other dietary changes, presumably calorie restriction. In addition, no account is taken of the adequacy of urine collections eg, 24-hour creatinine excretion, an essential component of a study on salt restriction.

What is needed is a properly controlled, double-blinded randomized study of modest salt reduction looking at sympathetic tone. Even if there was an increase in sympathetic tone, this may be part of the normal physiological response, as is the increase in plasma renin activity. The authors’ lack of scientific rigor is also evident in their quoting of Alderman’s review on salt intake and cardiovascular disease. His studies have been severely criticized.2 Grassi et al1 do not seem to be aware of a more in-depth analysis of the data from the first National Health and Nutrition Examination Survey showing that a high salt intake was significantly associated with an increased risk of cardiovascular disease and all-cause mortality in overweight persons, and more recently, of heart failure.3 A study using a random sample of the Finnish adult population showed that a high salt intake was directly related to increased cardiovascular and total mortality.4 The authors do not quote any other papers showing that a long-term modest salt reduction does not have adverse effects. For instance, Beckmann et al5 demonstrated that a modest reduction in salt intake, along with a reduction in body weight and saturated fat for 1 year, significantly reduced arterial plasma noradrenaline and adrenaline in hypertensive subjects.

Furthermore, salt reduction lowers blood pressure in a similar mechanism to that of thiazide diuretics. Both stimulate the renin-angiotensin system and in the short-term the sympathetic nervous system. However, outcome trials have repeatedly demonstrated that long-term treatment with thiazide diuretics significantly reduces cardiovascular morbidity and mortality in hypertensive patients.

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Response

Our study1 aimed to provide evidence on whether the acute sympathoexcititation we found when hypertensive patients were exposed to a short-term drastic reduction in salt intake2 could also be seen when salt intake was moderately reduced and maintained for a longer time. In the patients in whom the study requirements were met, this was clearly the case. It is worth noting that (1) we used the most sensitive and reproducible available technique to quantify sympathetic activity, (2) the protocol was extremely demanding because it required 4 good quality nerve recordings in each patient, and (3) although the study was not double-blind, bias was avoided because nerve traffic was quantified by an investigator unaware of the experimental design.

Furthermore, adherence to diet was assessed by 24h urinary sodium excretion before, during the dietary intervention (4 times), and after restoration of regular sodium diet. We cannot exclude that, despite instructions, some patients reduced caloric intake. However, a reduction in caloric intake and body weight leads to sympathetic deactivation.3 Thus, its occurrence should have opposed rather than favored the sympathetic activation. Also, as when caused by antihypertensive drugs, sympathetic activation should be regarded as a non-desirable effect of the blood pressure lowering intervention on the basis of the evidence that increasing sympathetic activity may not only oppose the blood pressure reduction but also contribute to organ damage and metabolic alterations, some of which have been reported for low-sodium diet.

Our study did not aim to show to what extent a low-sodium diet is useful in hypertension, which is the reason why we avoided extensive quotation of the literature. We believe, however, that the issue remains somewhat controversial,4 especially because morbidity and mortality data under low-salt diet are still unavailable. We do not agree that a low-sodium diet can be equated to diuretic treatment, whose mechanisms of action are more complex. Furthermore, this (as well as other treatment) fails to normalize the cardiovascular risk of the treated hypertensive patient, raising the possibility that some undesirable effects minimize the net benefit.

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