Weight-Reducing Diets and Sodium Intake

To the Editor:

I have the following comments to the recent article by Fagerberg et al.1 In this stimulating article, the authors reported the effects of caloric restriction with or without concomitant reduction of sodium intake on the blood pressure (BP) and sympathetic nervous system activity of obese hypertensive men (see also Reference 2).

Based on their results, Fagerberg et al. concluded that hemodynamic adjustment and BP reduction were associated with weight loss only when dietary sodium was restricted, although a decrease in sympathetic tone seemed to occur both in men on the low energy–unchanged sodium regimen (Group I) and in those on the low energy–low sodium diet (Group II). In this regard, I should like to observe that there was actually some BP reduction not only in Group II but also in Group I: average BP fall in Group I was 4.7/5.0 mm Hg in the first report (n = 13) and 7.5/6.1 mm Hg in the second report (n = 10).2 Despite the small sample size, this result cannot be overlooked, at least from an epidemiological point of view.

My second observation is that, judging from the reduction in urinary norepinephrine (NE) excretion, a more marked decrease in sympathetic discharge seemed to occur in the low energy–unchanged sodium group (Group I), a finding also supported by the concomitant significant decrease in plasma renin activity in this group. Based on the findings of the NE infusion test, the authors concluded that this effect of the low energy diet was counteracted by an increase in the vascular sensitivity to NE, probably due to up-regulation of adrenergic receptors, whereas such unfavorable adjustment would have been prevented in Group II by the concomitant reduction of sodium intake. Accordingly, in their interpretation, BP fell only (or to a greater extent) in Group II.

It has been repeatedly suggested that sodium restriction per se may enhance the sympathetic discharge,14 if this is true, then the depressing effect of low energy intake on sympathetic tone might have been balanced at least partly in Group II by the contrasting effect of reduced sodium intake. Thus, I wonder whether the unchanged reactivity to exogenous NE in Group II might simply reflect a lesser reduction in sympathetic discharge compared with that in Group I. If so, other explanations must be sought for the greater BP fall in this group. (In fact no statistical relationship was found between BP changes and changes in reactivity to NE infusion.) An alternative interpretation of the data is that a volume factor related to sodium restriction adds to the effect of caloric restriction alone in Group II, resulting in a more pronounced BP fall in this group. I believe that the lack of a demonstrable difference in blood volume in the two groups does not rule out this possibility. This view is supported by the finding that the BP reduction was traced to reduced cardiac output (and cardiac index) in Group II, although total peripheral resistance was unchanged (a finding difficult to reconcile with the interpretation proposed by the authors).

What also puzzles me in this context is the role of the carbohydrate (CHO) content of weight-reducing diets with respect to their hemodynamic effects. In another recent article1 they reported on the lack of effect on BP and sympathetic nervous system activity of a diet identical to the one adopted in this study (i.e., >50% of total intake supplied as carbohydrates). They found instead that an isocaloric diet with much lower CHO content (CHO < 30% of total intake) reduced BP through a reduction of sympathetic tone (and with no need for sodium restriction). Thus, they concluded that a relatively high CHO content prevented the BP lowering effect of caloric restriction, while reduction of CHO to less than 30% of total intake significantly reduced sympathetic tone and BP. I wonder why in this later study they used a diet with 50 to 60% of total intake from carbohydrates, which was likely to be ineffective according to their own previous experience. It seems to me that they could have obtained more definite information had they tested the additive effect of sodium restriction with respect to a low calorie and low CHO diet.

References
The second question is whether the reduced response to exogenous NE in the group with energy and sodium restriction might simply reflect a lesser reduction in their sympathetic discharge. It is important to remember that a growing body of data indicates that the sympathetic nervous outflow is intermittent by nature as well as differentiated in terms of different vascular beds. We measured NE in three separate ways and tried to interpret the results in accordance with the new concept that each sampling point yields NE concentrations that are determined by the sympathetic outflow in the examined vascular bed. In Group I (low energy–normal salt diet), urinary NE excretion and PRA diminished, indicating a lowered renal sympathetic discharge. In Group II (low energy–low salt diet), on the other hand, arterial plasma NE concentration showed a highly significant decrease, which indicates a reduced cardiac sympathetic activity supported by a substantial fall in heart rate. Taken together, these results suggest that the two diets were associated with different responses in the sympathetic nervous system. We find it difficult to determine that one group showed a lesser decrease in sympathetic outflow than the other owing to this differentiated response.

The third question (raised as an alternative explanation) is whether sodium restriction induced a reduction in extracellular fluid volume and thereby caused a lowered blood pressure by reducing cardiac output. This suggestion is refuted by several observations. In a previous report we have shown that the diet in Group II is not associated with any decrease of extracellular volume. Furthermore, there was no contraction of blood volume in the present report. Finally, the reduction in sympathetic nervous activity satisfactorily explains the diminished cardiac output, which was caused by a lowered heart rate while stroke volume remained unchanged.

The final question was why blood pressure fell in the present study, which used a diet with the same carbohydrate content as that in a previous study. We found in the previous study that a high content of carbohydrates (59%) in a reduced-calorie diet may prevent the expected reduction in blood pressure and sympathetic discharge. However, there is an important difference between the two diets. In the first reported experiment more than 50% of the carbohydrate was pure sugar. It has been suggested that monosaccharides and disaccharides in excess have properties that are capable of stimulating the sympathetic nervous system, which is a possible explanation for the different responses to the two dissimilar diets. Finally, it should be mentioned that the previous study on the effect of a high sugar–low energy diet (1400 kcal) also was associated with a 59 mmol/day decrease of urinary sodium output, which was obtained without any advice on sodium intake. This finding nicely shows that a weight-reducing diet is associated with a lowered sodium intake.

References
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