Dietary Sodium Restriction in Heart Failure: A Recommendation Worth its Salt?*

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Dietary sodium “indiscretion” is considered a common and potentially modifiable precipitant of heart failure (HF) decompensation. As a result, dietary sodium restriction is the most commonly recommended self-care behavior suggested to patients with HF (1). On average, Americans consume between 3,400 and 3,700 mg/day of sodium (2). Current guidelines advise that patients with symptomatic HF should restrict sodium intake to 2,000 to 3,000 mg/d or less (3,4). Illustrating the paucity of data in this area, these thresholds for “restriction” are actually higher than the 1,500 mg/d maximum amount recommended for all Americans by the American Heart Association’s Presidential Advisory panel (5).

In this issue of JACC: Heart Failure, Doukky et al. (6) provide new information for this debate and challenge conventional wisdom. The authors analyzed data from the Heart Failure Adherence and Retention Trial, which assessed the efficacy of self-management counseling in 902 patients with HF followed up for a median of 36 months. Sodium intake was assessed by using a food frequency questionnaire; subjects were categorized as sodium restricted (defined as <2,500 mg/d) and sodium unrestricted (≥2,500 mg/d). Adjusting for many potential confounders, 130 subjects in each group underwent propensity score matching. Matched patients reporting sodium restriction had a significantly higher risk for the combined primary endpoint of death or HF hospitalization (42.3% vs. 26.2%; hazard ratio: 1.83; 95% CI: 1.21 to 2.84; p = 0.004), driven primarily by an increased risk of HF hospitalization (32.3% vs. 20.0%; hazard ratio: 1.82; 95% CI: 1.11 to 2.96; p = 0.015). These propensity-matched observations were confirmed across the entire cohort and after adjustment for residual differences between groups.

These seemingly counterintuitive findings are supported by several lines of evidence. Particularly in the setting of fluid restriction and diuretic therapy, sodium restriction may increase sympathetic and renal-angiotensin-aldosterone system activation by contributing to intravascular volume depletion (7). Few randomized studies of dietary sodium restriction in patients with HF have been performed, but the findings are generally aligned with those of the present report (6). In a small inpatient study, aggressive sodium and fluid restriction (800 mg/d and 800 ml/d, respectively) did not produce clinical improvement or reduce length of stay (8). In a large, randomized Italian trial examining the acute effects of hypertonic saline and furosemide in inpatients with HF and reduced ejection fraction, patients assigned to low versus moderate sodium intake (i.e., 1,840 vs. 2,760 mg/d) had reduced diuresis (9). This study (as well as 2 smaller trials by the same group) noted more HF readmissions, poorer renal function, and a trend toward increased mortality in patients continuing on low-sodium intake post-discharge. The generalizability of these results is unknown, as subjects received strict fluid restriction (1,000 ml/d) and high diuretic doses (up to 100 to 1,000 mg/d of furosemide) that were not adjusted based on clinical status; moreover, many participants were not receiving optimal neurohormonal blockade.

We agree with the conclusion of Doukky et al. (6) that randomized trials evaluating sodium restriction in patients with HF receiving standard of care treatment are sorely needed. Until then, a recent

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American Heart Association Science Advisory provides a framework by which observational studies relating sodium intake to cardiovascular outcomes can be analyzed (10). Errors or misclassification in sodium intake represent the largest issue to overcome. Although many studies have used food frequency questionnaires and food diaries, these measures are subject to incomplete recording and recall bias. Averaging multiple 24-h urinary sodium collections provides the most accurate characterization of usual sodium intake. However, even this gold standard presents substantial difficulties, including patient burden, confounding by variable diuretic use, and the potential for error introduced by incomplete collections.

A notable aspect of this study (6) is that it averaged estimated sodium intake from multiple time points (baseline and 1, 2, and 3 years). A food frequency questionnaire assessed intake of 57 common food items over the preceding week. Daily sodium intake was then estimated by adding the sodium content of these items to an assumed baseline sodium consumption of 1,250 mg/d derived from essential food items in the American diet. As with other questionnaires, this tool is subject to recall inaccuracies and bias; it is also unclear how much measurement error is introduced by assuming a “floor” for sodium consumption. The food frequency questionnaire used was originally developed to assess cholesterol and fat intake (11), and its accuracy for sodium intake has not been reported. Although the mean estimated sodium intake in this study was similar to other cohorts with HF (12), comparison with 24-h urinary sodium excretion or other validated questionnaires would have strengthened the generalizability of the findings.

Reverse causality is another issue that could alter the association between sodium intake and outcomes (10). In other words, higher risk individuals might consume less sodium either because they have been instructed to do so or because of the severity of the illness itself. Indeed, sodium-restricted patients in the study by Doukky et al. (6) more frequently had a history of chronic kidney disease or cerebrovascular accident and were taking higher doses of loop diuretics. In addition, sodium restriction seemed most harmful in patients not taking angiotensin-converting enzyme inhibitors or angiotensin receptor antagonists. Although this finding could point to “unblocked” neurohormonal activation during sodium restriction, patients with more advanced HF are also more likely to have intolerance to these medications. The authors acknowledge the potential for reverse causality and achieved well-balanced groups for comorbidities, medication use, and HF severity through propensity matching.

Despite this approach, several potentially important unmeasured confounders are worth mentioning. First, previous hospitalization is the strongest predictor of subsequent admissions (13). Because HF hospitalization drove the primary outcome, adjustment of the propensity score history for prior hospitalizations would have been of great interest. This factor is potentially also important because patients often receive instructions at hospital discharge for reducing sodium intake (14). It seems plausible that patients with multiple prior HF admissions might have had lower baseline and subsequent sodium intake, as well as high risk for readmission.

Second, overall nutritional status and caloric intake are important issues to consider. Weight loss and protein calorie malnutrition strongly predict adverse outcomes in HF with reduced ejection fraction. Body mass index and serum albumin, although well matched by propensity scoring in the study by Doukky et al. (6), are not strong surrogates for nutritional status in HF (15). Moreover, baseline measurements do not provide the trajectory of a patient’s body composition or nutritional status over time. Sodium intake is closely correlated with caloric intake in the United States (16), and calorie indexing of dietary sodium content could help refute reverse causality due to overall low-energy intake in this longitudinal study.

The results of this study (6) highlight the need for further research investigating the effect of sodium restriction on HF physiology and outcomes. It is notable that only 145 of the 902 study patients were restricting their sodium intake to <2,500 mg/d. Considering the challenge this restriction poses for patients, it is even more important to clarify whether sodium restriction is beneficial at all. Furthermore, studies need to determine if variables such as HF subtype and severity, comorbidities, medical therapies, or “salt sensitivity” (a specific vulnerability to adverse effects during high-sodium diet) should affect a patient’s recommended daily sodium intake (17). Ongoing randomized trials in outpatients with stable HF (NCT02012179) and patients with HF after their hospital discharge (NCT02148679 and NCT02467296) may provide some answers. Until more data are available, however, guidelines regarding sodium intake in patients with HF should be taken with a grain of salt.

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