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Should We Restrict Sodium Intake in Compensated Heart Failure?

Ileana CORLAN, MD

Cardiology and Internal Medicine Department, Emergency University Hospital, Bucharest, Romania

One of the main dietary recommendations in patients with congestive heart failure is sodium intake restriction, based on the well known kidney inability to excrete excess sodium in untreated heart failure patients. Recent data, however, suggest that in patients with compensated heart failure treated with beta-blockers and inhibitors of the rennin-angiotensin-aldosterone system (RAAS), volume expansion produces hemodynamic, neuroendocrine and renal responses similar to that of controls, in which another study demonstrated a decrease in vascular resistance, increase in cardiac performance and suppression of vasoconstrictive hormones, with high sodium intake.

This study performed by Dr. Damgaard and col. from several centers in Copenhagen, Denmark assessed hemodynamic, neuroendocrine and renal responses to a change in sodium intake in 12 controls and 12 medically treated patients with stable, compensated heart failure, with EF < 40%, in sinus rhythm, without recent history of cardiac ischemic event, with normal creatinine levels and under a treatment unchanged 2 weeks prior to the study, that included in all patients a RAAS inhibitor and a beta-blocker.

The protocol consisted in 2 consecutive one-week periods between which the patients shifted from a low (70mmol/day) to a high (250mmol/day) sodium diet or vice-versa in a randomized fashion, with a free water intake, with resting (seated position) and exercise (bicycle until dyspnea or fatigue) measurements at the end of each dietary period.

With high sodium intake, in the seated position and during exercise cardiac index and stroke volume index increased significantly in both patients and controls, while mean arterial pressure remained unchanged; plasma norepinephrine and plasma angiotensin II concentrations decreased significantly and plasma pro-BNP did not change at rest or exercise.

With all the limitations of the study, such as the use of diuretic in 10 of the 12 patients, the short duration of the study and the noninvasive techniques for the assessment of hemodynamic variables, the authors succed to draw attention to the dogma that low sodium intake per se is favorable in heart failure.

Comment on the paper:
Morten Damgaard, Peter Norsk, Finn Gustafsson, et al – Hemodynamic and Neuroendocrine responses to Changes in Sodium Intake in Compensated Heart Failure. In: "American Journal of Physiology – Regulatory", *Integrative and Comparative Physiology* 2006; 290:R1294-R1301