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Letters

TO THE EDITOR

Factors That May Affect Body Change During and After Hospitalization for Acute Heart Failure

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We read with great interest the study performed by Ambrosy et al. (1) about body weight changes during and after hospitalization for acute heart failure and their effects on the different outcomes for patients with acute decompensated heart failure. Although the observed results were convincing regarding the benefit of weight loss as a marker of 30- and 180-day mortality decrease, the actual statistical analysis plan was missing one important possible confounding factor: the role of cardiac rehabilitation after discharge and whether those who underwent cardiac rehabilitation were more likely to have higher survival rates. It is known that cardiac rehabilitation is useful in patients with stable heart failure to improve not only functional status but also overall mortality and health-related quality of life (2). Even noncompliance with exercise regimens following admission for heart failure were associated with 1.5 times increased risk of mortality or readmission (3).

We also inquire about the absence of the baseline fluid and sodium restriction between the different groups, especially because fluid restriction is one of the recommended treatment measures in class D heart failure. The amount of fluid restriction and sodium intake may confound the results as patients exposed to high fluid restriction and sodium restriction were found to have less mortality and better survival outcomes than those who did not follow fluid and salt restriction in some studies, even while taking the same medications (4,5).

Adjusting the results by considering these previously mentioned factors would give a more accurate idea about the effects of weight gain or loss while maintaining the same pharmacological and nonpharmacological treatment measures.

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REPLY: Factors That May Affect Body Change During and After Hospitalization for Acute Heart Failure

We appreciate the interest by Dr. Rasla and colleagues in our recent publication (1). The authors comment on the fact that dietary restrictions and referrals for cardiac rehabilitation were not taken into consideration and might potentially have confounded body weight measurements. However, these data were not collected as part of the ASCEND-HF (Acute Study of Clinical Effectiveness of Nesiritide in Decompensated Heart Failure) trial (2).

Regarding dietary restriction, there are limited data addressing the optimal levels of sodium intake, both in ambulatory and hospitalized heart failure populations (3). Not only have there been no randomized investigations testing the safety and efficacy

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of salt restriction for stable heart failure, some studies suggest that low-sodium diets may increase activation of the renin-angiotensin-aldosterone system potentially translating into worse clinical outcomes. The PROHIBIT (Prevent Adverse Outcomes in Heart Failure by Limiting Sodium) study will evaluate adherence to different levels of sodium restriction and provide pilot data on the feasibility and design of a pivotal trial of dietary sodium intake (4).

In addition, although cardiac rehabilitation has been shown to be safe and have a modest effect on quality of life in patients with chronic heart failure, data for cardiac rehabilitation is limited to stable outpatients (5). Patients hospitalized for acute heart failure are phenotypically different than those with ambulatory heart failure and may lack the ability to participate in and/or derive a robust benefit from a structured aerobic exercise program. Thus, the role of cardiac rehabilitation after hospitalization for acute heart failure remains uncertain and is the focus of the ongoing REHAB-HF (A Trial of Rehabilitation Therapy in Older Acute Heart Failure Patients; NCT02196038) trial.

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Amiodarone and Beta-Blockers in Patients With Heart Failure and Atrial Fibrillation

We appreciate the editorial by Piccini and Allen (1), which critically appraises the evidence for and against a survival benefit associated with β-blockers in patients with heart failure with reduced ejection fraction and concomitant atrial fibrillation. The balanced analysis highlights the divergent results obtained in the patient-level meta-analysis by Kotecha et al. (2) and the recently published β-blocker substudy from the AF-CHF (Atrial Fibrillation and Congestive Heart Failure) trial (3). Methodological challenges confronted by these 2 observational studies were discussed. It was insightfully contended that limitations largely stem from the fact that the studies included in the meta-analysis were not designed to assess the population with atrial fibrillation, and that β-blockers were not randomized in the AF-CHF trial.

We deemed it necessary, however, to clarify an important inaccuracy. Piccini and Allen (1) state that the authors of the AF-CHF β-blocker substudy "did not account for the potential impact of β-antagonism from amiodarone." Accounting for amiodarone is, indeed, a critical issue considering that it was the drug of choice for patients randomized to rhythm control therapy and that it has β-antagonism properties. Moreover, prescription bias resulting from withholding β-blockers in patients on amiodarone following myocardial infarction has previously been proposed to explain the absence of all-cause mortality reduction despite fewer arrhythmic deaths (4,5). For these reasons, it was regarded as essential to adjust for amiodarone. As such, amiodarone was a key variable included in generating the propensity score to match patients with and without β-blockers to control for potential selection bias and confounding by indication. Balance in pseudorandomized treated and untreated groups was empirically verified across observable patient characteristics. More specifically, after propensity matching, 42% of patients with and 42% of those without β-blockers received amiodarone (3). The absolute standardized difference of 1.6% was far lower than the 10% cutoff value indicative of residual bias.

Considering the totality of evidence and the lack of a clear biologically plausible mechanism to explain how atrial fibrillation nullifies salutary effects of β-blockers