Impact of Diastolic Dysfunction on Heart Failure-Related Hospitalizations

Fang et al. (1), using National Hospital Discharge Survey data, showed a steady increase in heart failure hospitalizations from 1979 to 2004. More than 80% were elderly (age >65 years). The median hospital stay and proportion of in-hospital deaths declined during this period. This declining mortality was related to improvement in medical therapy and widespread availability of revascularization procedures.

Over the last 3 decades, a novel syndrome, heart failure with preserved systolic function (diastolic dysfunction), has been increasingly recognized. This predominantly involves the elderly and is responsible for approximately 50% of heart failures in this group (2,3). Fang et al. (1) did not mention left ventricular function in their report, and the impact of diastolic heart failure in their conclusions is speculation.

Recent epidemiologic data have confirmed the increasing prevalence and hospitalization of patients with diastolic dysfunction (3). Over the last 2 decades, there have been significant advances in the management of heart failure, but these have almost exclusively focused on a population with systolic dysfunction. Survival among patients with preserved ejection fraction has been shown to be better than that among those with reduced ejection fractions (4,5).

In their report, the investigators highlighted an increase in the hospitalization of women with heart failure, relative to men. Given the generally older female U.S. population, patients with diastolic dysfunction have consistently been shown to more likely be female (5,6).

Therefore, the increase in the prevalence of diastolic heart failure likely has a major contribution to the findings of Fang et al. (1) and may entirely account for the increase in hospitalization, the aged population, the gender discrepancy, as well as the demonstrated improved survival.

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Reply

We would like to thank Dr. Sorrell and colleagues for their interest in our work (1). The primary objective of our study was to use national surveillance data to examine annual hospitalization rates and trends in characteristics for patients hospitalized with heart failure as either the first-listed diagnosis or additional (second to seventh) diagnosis, as well as to assess the trends in other clinical conditions that lead to hospitalization among patients with any mention of heart failure. Thus, we did not present data on left ventricular ejection fraction or speculate on the impact of systolic or diastolic ventricular function in our conclusions. In fact, the National Hospital Discharge Survey contains no information for any measurement of left ventricular function.

We agree with Dr. Sorrell and colleagues that a substantial proportion of patients have heart failure with preserved systolic function and that this proportion has increased over time (2). We would like to use International Classification of Diseases–Ninth Revision–Clinical Modification (ICD-9-CM) codes to identify heart failure with systolic heart failure (ICD-9-CM 428.2) and diastolic heart failure (ICD-9-CM 428.3). Unfortunately, these codes were not in effect until 2003. Previously, both conditions were coded as 428.0. The data showed that the percentages of recorded isolated systolic heart failure were 1.2%, 1.7%, and 1.8% for 2003, 2004, and 2005, respectively, and the corresponding percentages for isolated diastolic heart failure were 3.0%, 4.3%, and 4.5%, respectively. Although the percentages for diastolic heart failure were more than twice those of systolic heart failure, neither contributed a significant proportion to total heart failure. However, as pointed out by Dr. Sorrell and colleagues, women had a higher proportion of isolated diastolic heart failure than men (4.7% vs. 3.0%, p < 0.001) and had a lower proportion of isolated systolic heart failure (1.2% vs. 2.0%, p < 0.001). Because only 3 years of hospitalization data are available for identifying systolic heart failure and diastolic heart failure separately, we are unable to present 25-year trends.
Therefore, with the limitations of the National Hospital Discharge Survey, we are unable to conclude that the increase in the prevalence of diastolic heart failure contributed to the finding of increasing heart failure hospitalization over the past 25 years.

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Reply

We thank Dr. van Gestel and colleagues for their interest in our work (1). They wonder whether the presence of underlying chronic obstructive pulmonary disease (COPD) might have contributed to the strong association between N-terminal pro-B-type natriuretic peptide (NT-proBNP) and outcomes in the Rancho Bernardo Study. We did not find this to be the case.

Dr. Van Gestel and colleagues stated that COPD affects nearly 35% of the elderly population; however, the article that they reference actually found this incidence only in current smokers (2). The cumulative incidence of COPD was only 14% in ex-smokers and 12% in never-smokers.

Elevated N-Terminal Pro-B-Type Natriuretic Peptide Levels:  
The Effect of Chronic Obstructive Pulmonary Disease

We read with great interest the recent article by Daniels et al. (1) in which the investigators reported that detectable cardiac troponin T and N-terminal pro-B-type natriuretic peptide (NT-proBNP) were both associated with increased all-cause and cardiovascular death in healthy older adults.

NT-proBNP was previously identified as a prognostic cardiac risk marker associated with increased mortality. NT-proBNP is released by cardiac myocytes in response to wall stress in conditions associated with volume overload as in heart failure and chronic kidney disease, pressure overload as in patients with heart valve abnormalities, and ischemia owing to coronary disease. In the present study, the association remained even after participants with baseline coronary heart disease were excluded, which was 30% of the subjects with elevated (≥450 pg/ml) NT-proBNP levels.

However, the authors might have overlooked the effect of chronic obstructive pulmonary disease (COPD) on NT-proBNP levels. COPD is associated with cardiovascular disease and is an independent risk factor for cardiovascular morbidity and mortality (2). We recently investigated the relationship between COPD, both the presence and severity, and NT-proBNP levels in 376 patients. To mitigate the influence of heart failure, chronic kidney disease, and myocardial ischemia, we adjusted for history of angina pectoris, myocardial infarction, heart failure, and renal function. In addition, all patients had resting left ventricular function of more than 40% using echocardiography. The severity of COPD was assessed using pulmonary function tests with the GOLD (Global Initiative for Chronic Obstructive Lung Disease) classification. We found COPD to be an independent risk factor for increased NT-proBNP levels, and the levels increased with the severity of COPD.

The underlying mechanism is likely to be pulmonary hypertension and right ventricular dysfunction caused by pulmonary arterial pressure overload (3). COPD may induce wall stretching, ventricular dilation, and/or increased vascular pressures, which may promote the secretion of the neurohormone NT-proBNP. Because COPD is common in the elderly population (affecting nearly 35% of this population) (4), the addition of spirometric data to the analysis by Daniels et al. (1) would have been very interesting and informative. The less severe forms of COPD are often asymptomatic and therefore frequently underestimated, especially in the elderly. Thus, the presence of underlying COPD might have contributed to the observed correlation between NT-proBNP and outcome in this elderly population.

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