Is Diastolic Heart Failure Synonymous With Heart Failure With Preserved Ejection Fraction?

In his excellent Editorial Comment, Dr. Zile states that “heart failure with a preserved ejection fraction” and “diastolic heart failure” are synonymous (1). Respectfully, I must disagree. Not all patients with diastolic heart failure have left ventricular hypertrophy. Therefore, the general applicability of the study cited supporting the equivalency of the two terms might be limited because all patients in that study had echocardiographic evidence for left ventricular hypertrophy, and diastolic dysfunction is generally accepted to precede hypertrophy. In our early experience about one-third of patients with heart failure with a preserved ejection fraction had explanations for the signs and symptoms of failure other than diastolic dysfunction, predominately right heart failure due to pulmonary disease and regurgitant valvular heart disease (2).

The nonspecific nature of the symptoms of heart failure and iatrogenic volume overload were also noted. It is unclear to what extent stricter diagnostic criteria for heart failure would affect these findings, and I believe that our initial criteria would still lead most clinicians to the diagnosis of heart failure. Furthermore, a patient with heart failure due to chronic, severe mitral regurgitation with an ejection fraction of 40% or even 50% has predominately systolic, not diastolic, heart failure. Therefore, I believe it is best to conclude that patients with “diastolic heart failure” form a subgroup of patients with “heart failure with a preserved ejection fraction.”

Until a uniformly accepted and therapeutically meaningful measure of diastolic dysfunction is defined, diastolic heart failure is in many ways a diagnosis of exclusion. The value of initially using the term “heart failure with preserved, or normal, ejection fraction” underscores the need to define left ventricular function in virtually all patients with heart failure (3) as well as the need to carefully eliminate other cardiac and noncardiac possibilities from the patient’s signs and symptoms. After eliminating other possibilities, I agree that the term “diastolic heart failure” seems most appropriate, and I hope, as Dr. Zile does, that accepting the term promotes the investigative efforts that are long overdue for these patients.

REFERENCES


REPLY

One of the first, if not the first, study to use the term “diastolic heart failure” was by Dr. Kessler in 1988 (1). His report was truly innovative and showed remarkable insight into a difficult clinical problem. I enthusiastically agree with Dr. Kessler’s point of view and I am grateful to receive his support. In his letter to the editor of the JACC, he raises three important issues: 1) some patients with diastolic heart failure do not have left ventricular (LV) hypertrophy; 2) the diagnosis of diastolic heart failure should exclude patients with noncardiac (such as pulmonary disease) and other cardiac (such as mitral stenosis, regurgitant valve disease) causes of heart failure; and 3) left ventricular (LV) function must be measured in every patient with heart failure.

In the study that Dr. Kessler refers to in his letter (2), only about one-third of the patients had LV hypertrophy defined as LV mass ≥125 g/m². However, all patients had concentric hypertrophic remodeling characterized by a decreased LV end diastolic volume/mass ratio or LV end diastolic dimension/wall thickness ratio or an increased relative wall thickness. I believe that a majority of patients with diastolic heart failure in fact have either concentric remodeling or some other evidence of myocardial or cardiac structural alterations such as an enlarged left atrium. With or without concentric remodeling, if a patient truly has objective signs and symptoms of heart failure and noncardiac and other cardiac causes have been ruled out, then heart failure with a normal ejection fraction (EF) is caused by diastolic dysfunction and the appellation “diastolic heart failure” should be applied.

Dr. Kessler correctly points out that in patients with primary right heart failure (caused by chronic lung disease, pulmonic stenosis, tricuspid regurgitation) or mitral stenosis or left-sided regurgitant valvular disease, this can result in heart failure with a normal EF. I am grateful that Dr. Kessler emphasized this point because our previous publications (2,3) did not make it explicitly clear that we had in fact excluded patients with noncardiac and other cardiac causes of heart failure in this study patient cohort.
It is critical that LV function, both systolic and diastolic, be measured in every patient with heart failure. The advent of tissue Doppler (and other echo-Doppler techniques) has made it easier and more practical to identify abnormalities in diastolic function. Nonetheless, precise and comprehensive assessment of diastolic function requires the use of invasive catheterization techniques. However, if noncardiac and other cardiac causes of heart failure are excluded, the remaining patients with heart failure and a normal EF all have abnormalities of diastolic function making measurement of diastolic function confirmatory rather than a mandatory component of diagnostic criteria.

Finally, I join Dr. Kessler in his enthusiastic use of the term "diastolic heart failure" and renew my editorial plea: "Stop the discrimination against the term diastolic heart failure."

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Determination of the Natural History of Aspirin Resistance Among Stable Patients With Cardiovascular Disease

We read with interest the recent study by Gum et al. (1) regarding aspirin resistance. Their results, in particular the focus on long-term follow-up, offer important information in the confusing but clinically important area of aspirin resistance. They were able to show that, by using standard light-transmittance aggregometry in a population of patients already on aspirin therapy, the response to two different platelet agonists could predict long-term outcome. However, in these investigators’ original study of baseline aspirin responsiveness in this identical patient population, a point-of-care test, the platelet function analyzer (PFA)-100, was also used to determine aspirin responsiveness along with light transmittance aggregometry (2). In the first study, minimal correlation between the two methods was found. It is unclear to us, though, why long-term outcomes based on baseline aspirin responsiveness as determined by the PFA-100 were not also included in their present report. Clearly the routine determination of aspirin responsiveness will depend upon the ability to measure it with a point-of-care device. Therefore, whether or not the PFA-100 results correlated with long-term clinical outcomes would have important implications regarding its utility in that role. The importance of this question is highlighted in the editorial following the Gum et al. (3) study as well as in a recent review of the topic, as both suggest that the PFA-100 may be well suited for the routine determination of aspirin resistance (3,4). However, this would likely not be the case if PFA-100 results were found to not have any clinical relevance in terms of future thrombotic events.

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REPLY

We appreciate and share the interest of Dr. Varinasi and colleagues in aspirin resistance and its clinical relevance. Previously, we documented the profile and prevalence of aspirin resistance in stable patients with cardiovascular disease (1). In this initial study, we used both optical platelet aggregation, which we consider to be the gold standard for the determination of platelet reactivity in the presence of aspirin, and a rapid, whole-blood assay, the platelet function analyzer (PFA)-100, to determine the prevalence of aspirin resistance. The kappa statistic between these two methods was 0.1 (95% confidence interval 0.045 to 0.246), indicating a poor correlation between optical platelet aggregation and the PFA-100 in detection of aspirin resistance.

In our more recently published work (2), we reported an increased risk of death, myocardial infarction (MI), or stroke associated with aspirin resistance as determined by optical platelet aggregation. In analysis, long-term outcomes (death/MI/stroke) were not related to aspirin resistance status as determined by the PFA-100 (12.9% aspirin sensitive vs. 15.1% aspirin resistant, p = 0.4). These findings seem to indicate that the PFA-100 is not as specific a test as compared to optical platelet aggregation for determining clinically relevant aspirin resistance. In fact, this supposition may be supported by the poor kappa statistic between the two tests. However, prior to categorically drawing this conclusion, one must acknowledge the real possibility of a type II error. Although there may be no statistical association between the PFA-100 and clinical outcomes in our investigation, a real association may have been missed by the small sample size of our study.