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.”

Michael R. Zile, MD
Cardiology/Medicine
Medical University of South Carolina
135 Rutledge Avenue
Suite 816
P.O. Box 250592
Charleston, SC 29425
E-mail: zilem@musc.edu
doi:10.1016/S0735-1097(03)00998-7

REFERENCES

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 transmission 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.

Steven R. Steinhibl
University of North Carolina
Division of Cardiology
CB #7075
Chapel Hill, NC 27599
E-mail: steven_steinhubl@med.unc.edu

Jay S. Varinasi, MD
Lee Goldberg, MD

doi:10.1016/S0735-1097(03)00999-9

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.

REFERENCES