colleagues remain concerned that we have underestimated the importance of PMI. We agree with many of the suppositions in their communication (as clearly stated throughout our report [1]), namely that “even small periprocedural troponin rises reflect new areas of myocardial necrosis on MRI imaging,” and “there is little doubt about the influence of large periprocedural enzyme rises . . . .” We disagree, however, that “debate persists about the influence on prognosis of these small/moderate enzyme rises . . . .” Whereas the literature is replete with prior studies examining the influence of PMI, the largest studies in the stent era have shown that only truly large PMI as evidenced by elevation of peak creatine kinase-myocardial band to >8× or >10× normal or the development of new Q-waves significantly influences subsequent survival (2,3). Other studies that have used a low threshold for PMI have found that such infarctions have no long-term prognostic significance (4). Smaller enzyme elevations are a marker for more diffuse atherosclerosis (5), but do not have per se a great enough influence on left ventricular function or arhythmmogenesiss to directly impair coronary survival. Other studies have suggested that biomarker elevations after percutaneous coronary intervention are of no clinical relevance unless associated with a failed procedure or angiographic complications (6). We also agree with Dr. Lim and colleagues that the prognostic implications of troponin elevations after percutaneous coronary intervention are uncertain, with questionable clinical relevance (7).

The data from the ACUITY (Acute Catheterization and Urgent Intervention Triage Strategy) trial are consistent with these previous studies; when considering all PMI (defined as creatine kinase-myocardial band >3× normal, the most widely used definition) in a time-updated, covariate-adjusted multivariable model, PMI was not an independent correlate of 1-year survival. However, large PMI (such as Q-wave myocardial infarction), was a predictor. Our study was not meant to minimize the role of large PMI, the largest studies in the stent era have shown that only truly large PMI as evidenced by elevation of peak creatine kinase-myocardial band to >8× or >10× normal or the development of new Q-waves significantly influences subsequent survival (2,3). Other studies that have used a low threshold for PMI have found that such infarctions have no long-term prognostic significance (4). Smaller enzyme elevations are a marker for more diffuse atherosclerosis (5), but do not have per se a great enough influence on left ventricular function or arhythmmogenesiss to directly impair coronary survival. Other studies have suggested that biomarker elevations after percutaneous coronary intervention are of no clinical relevance unless associated with a failed procedure or angiographic complications (6). We also agree with Dr. Lim and colleagues that the prognostic implications of troponin elevations after percutaneous coronary intervention are uncertain, with questionable clinical relevance (7).

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The paper by Boonyasirinant et al. (1) in a recent issue of the Journal of the American College of Cardiology (JACC) is important. In their report, they present data from the ACUITY trial showing that periprocedural enzyme rises are associated with worse outcomes. They conclude that these rises are “clinically significant.” However, these conclusions are based on a low threshold for PMI, which is not supported by current evidence. A more appropriate threshold for PMI should be used in future research.

Aortic Stiffness in Hypertrophic Cardiomyopathy

In the paper by Boonyasirinant et al. (1) in a recent issue of the Journal of the American College of Cardiology (JACC), there are serious problems in the use of pulse wave velocity (PWV) data as an index of aortic stiffness in hypertrophic cardiomyopathy (HCM).

The major technical problem is that whereas magnetic resonance imaging (MRI) can measure dimensions accurately, it can only measure flow at intervals of around 30 ms (Fig. 1 from Boonyasirinant et al. [1]), so that PWV, as distance travelled divided by time between wave feet, is likely to be less accurate, especially over very short distances (typically 12 to 13 cm in this report [1]) than when measured invasively or noninvasively by high fidelity manometry (2,3). This may account for unusually high variability of PWV measurements, especially in the HCM groups.

The most curious issue in this MRI report (1) is that normal subjects had lower values of aortic PWV (3.7 m/s, SD 0.9 m/s) than previously published for any normal group using invasive or noninvasive techniques (2,3). A previous MRI study (4) in normal (but obese) subjects of similar age gave PWV values of 6.8 m/s (SD 2.2 m/s), which is similar to that reported for HCM patients in the recent MRI report (1). Further, normal subjects (Fig. 5 from Boonyasirinant et al. [1]) had a blunted wave foot for the distal aortic wave, which is quite different from normal flow and pressure waves, from which PWV is usually calculated. High variability of PWV in HCM (mean 9.66 m/s, SD 6.43 m/s in 1 group and 6.51 m/s, SD 3.2 m/s in another) is not correctly represented in Figure 2 of Boonyasirinant et al. (1), nor are the confidence intervals, whose long “whiskers” do not appear at all. Data in the text and legend of Figure 2 (1) do not correspond to that shown in the figure.

These issues ought to be considered before the confident assertions of Boonyasirinant et al. (1) or Kuhl (5) are accepted that the MRI technique reveals “novel insights in vascular function from MRI” (1) or “an unidentified association unraveled by MRI” (5).
Increased Aortic Stiffness in Hypertrophic Cardiomyopathy
Different Methods, Same Conclusions?

We read the report by Boonyasirinant et al. (1) with great interest. The major finding of this study was that increased aortic stiffness, as indicated by increased magnetic resonance imaging (MRI)-derived pulse wave velocity (PWV), is evident in hypertrophic cardiomyopathy (HCM) patients and is more pronounced in those with myocardial fibrosis. The results are impressive, but we feel that a few additional comments are necessary.

Boonyasirinant et al. (1) used MRI-PWV analysis to quantify aortic stiffness in their clinical study. Measurement of PWV by different tonometric, piezoelectronic, oscillometric, and MRI methods are widely used scientific tools. However, there is another way to evaluate aortic stiffness, for which 2 important variables should be noted: 1) the change in volume due to blood injection into the aorta; and 2) the pressure change caused by this volume change (2). Together with measurement of forearm systolic blood pressure (SBP) and diastolic blood pressure (DBP) changes, aortic systolic diameter (SD) and diastolic diameter (DD) or cross-sectional areas at different levels of aorta can be measured with echocardiography, computed tomography, or MRI. Using these parameters, indexes or moduli can be calculated characterizing aortic elasticity (3). The most important parameters are listed here:

- Aortic strain = (SD – DD)/DD
- Aortic stiffness index (beta) = ln (SBP/DBP)/[(SD – DD)/DD], where SBP and DBP are the systolic and diastolic blood pressures, and ln is the natural logarithm
- Aortic distensibility = 2 × (SD – DD)/[(SBP – DBP) × DD]
- Aortic elastic modulus E(p) = (SBP – DBP)/[(SD – DD)/DD]
- Young’s circumferential static elastic modulus E(s) = E(p) × DD/2h, where h indicates diastolic intima-media thickness

Boonyasirinant et al. (1) were the first to demonstrate alterations in aortic distensibility in HCM. However, further investigations are warranted to examine the previously mentioned parameters in HCM, especially with versus without left ventricular outflow gradients. Moreover, correlations between PWV and echocardiography-derived parameters should be confirmed in HCM as well.

REFERENCES


We thank Dr. O’Rourke and colleagues and Dr. Nemes and colleagues for the interest in our study (1). A common thread is that each promotes alternative methods for computing aortic stiffness to the method we chose to employ, namely pulse wave velocity (PWV) computed by magnetic resonance imaging (MRI).

We are in agreement with Dr. Nemes and colleagues that more work is needed between and among these various approaches. We also recognize that many techniques exist to quantify aortic stiffness, and each is subject to its own characteristic strengths and weaknesses. Reliable quantification of PWV is dependent on accurate measurement of both the aortic flow (or pressure) wave at 2 measurement sites as well as the distance between these 2 sites. Previously, in numerous studies, MRI has been shown as a reliable technique for quantifying the aortic flow wave; its longitudinal dimensional accuracy is unparalleled, although it does suffer from a relatively low temporal resolution when compared with alternate techniques.