METHODS, OUTCOMES, AND RELATIONSHIPS

To the Editor:

Methods that quantify hemodynamic outcomes are essential after ventricular shape rebuilding. The December 2010 issue contains 2 thoughtful articles that evaluate surgical ventricular restoration (SVR) by using pressure–volume loops to address diastolic function¹ and echocardiographic differences in end-systolic and end-diastolic volume to determine cardiac output.² New York Heart Association (NYHA) classification improved in both reports (from 3.3 to 1.4¹ and 2.6 to 1.6²), yet the results imply passive diastolic stiffness increases, and similar diastolic dysfunction outcomes are observed as in patients in heart failure with preserved systolic function³ while stroke volume decreases in 71%.² Conversely, respective 6-month and 4-year follow-up studies demonstrate improvement with exercise to imply clinical diastolic dysfunction does not seem to occur and stroke volume must increase to improve NYHA status. These relationships of patient data during exercise suggest that these quantifiably impaired hemodynamic outcomes after SVR might be somewhat misleading.

Pressure–volume loops to evaluate diastolic dysfunction use only heart rate to evaluate a variable that is not clinically separate during exercise, as suggested within the heart rate discussion.¹ Heart rate might separate the effect of other functional determinants if

these are constant, but exercise also varies contractility, preload, and afterload, perhaps showing why SVR improves NYHA criteria from 3.3 to 1.4 after exertion. Westermann and colleagues⁴ studied patients not undergoing operations and described this vital difference between exercise and pressure–volume loops by showing that pacing alone caused findings that mirrored the pressure–volume loops findings previously reported by Tulner and associates⁵ after SVR. Conversely, adding supplemental hand-grip exercise excluded changes in ventricular stiffness, increased stroke volume, and allowed a wider chamber size. Results during exercise provide the true functional end point, and therefore the validity of theoretic calculations at rest to predict clinical outcomes needs testing during exertion.

Stroke volume increased at 80 beats/min in this recent pressure–volume loop study,¹ whereas Di Donato and coworkers² reported a 71% reduction when only echocardiography was used to measure stroke volume. Moreover, 90% 4-year survival was reported² without correlation of clinical improvement in patients whose stroke volume increased or decreased. This discrepancy may be clarified by following the authors' suggestion to do magnetic resonance imaging studies to determine 3-dimensional volume rather than echocardiography to determine 2-dimensional volume. Potential application of these stroke volume findings has important implications because the suggestion that SVR has neutral outcomes in akinetic ventricles would imply that the vast majority of patients who retain this anatomy after only revascularization might not have a good result. This suggestion contradicts the literature,⁶ except for the STICH trial, in which the post-SVR left ventricular end-systolic volume index decreased only 19% compared with the 40% reduction in registry reports of more than 1500 patients. Consequently, methods should correlate with clinical outcomes to have valid

relationships because measurement consistency should not be the only barometer of clinical validity.

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References

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Reply to the Editor:

In his letter, Dr Buckberg refers to our article¹ on stroke volume (SV) changes after surgical ventricular reconstruction (SVR) and to the article by ten Brinke and colleagues² that appeared in the same issue of the *Journal* in December 2010. Dr Buckberg commented on the contradiction between hemodynamic parameters worsening and clinical improvement at follow-up shown in the 2 studies and concludes that measurement modalities must correlate to clinical findings to be effective; otherwise, they may be misleading. In other words, Dr Buckberg questions the appropriateness,

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