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

1. ten Brinke EA, Klautz RJ, Tulner SA, Verwey HF, Bax JJ, Schalij MJ, et al. Long-term effects of surgical ventricular restoration with additional restrictive mitral annuloplasty and/or coronary artery bypass grafting on left ventricular function: six-month follow-up by pressure-volume loops. *J Thorac Cardiovasc Surg.* 2010;140:1338-44.
2. Di Donato M, Fantini F, Toso A, Castelvich S, Menicanti L, Anest L, et al. Impact of surgical ventricular reconstruction on stroke volume in patients with ischemic cardiomyopathy. *J Thorac Cardiovasc Surg.* 2010;140:1325-31.
3. Bursi F, Weston SA, Redfield MM, Jacobsen SJ, Pakhomov S, Nkomo VT, et al. Systolic and diastolic heart failure in the community. *JAMA.* 2006;296:2209-16.
4. Westermann D, Kasner M, Steendijk P, Spillmann F, Riad A, Weitmann K, et al. Role of left ventricular stiffness in heart failure with normal ejection fraction. *Circulation.* 2008;117:2051-60.
5. Tulner SA, Steendijk P, Klautz RJ, Bax JJ, Schalij MJ, van der Wall EE, et al. Surgical ventricular restoration in patients with ischemic dilated cardiomyopathy: evaluation of systolic and diastolic ventricular function, wall stress, dyssynchrony, and mechanical efficiency by pressure-volume loops. *J Thorac Cardiovasc Surg.* 2006;132:610-20.
6. Buckberg GD, Athanasuleas CL. The STICH trial: misguided conclusions. *J Thorac Cardiovasc Surg.* 2009;138:1060-4.

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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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validity, and perhaps the relevance of such hemodynamic measurements.

In regard to the effects on diastolic function induced by SVR, he ignores our article published in *Circulation* in 2004,³ in which we demonstrated that SVR induces a mechanical resynchronization through improvement of contraction and relaxation phases leading to early improvement in diastolic function, despite the marked reduction of end-diastolic volume. These data have been confirmed at follow-up in a more recent article.⁴

Diastolic filling depends on several “static and dynamic” variables, and in our opinion it is too simplistic to infer that surgical reduction of end-diastolic volume equates with induction of impaired diastolic function. End-diastolic volume reduction during SVR is the consequence of scar tissue exclusion. Moreover, an increase in stiffness does not necessarily mean a reduction of ventricular filling and worsening of clinical status, given that many dynamic factors can partially compensate for alterations in elastic properties of the ventricular wall. On the other hand, a majority of healthy elderly individuals are free of disease despite the presence of left ventricular diastolic dysfunction as detected by echocardiography. In addition, it is well known that diastolic dysfunction is neither necessary nor sufficient for the development of a heart failure clinical syndrome: Additional complex interactions with metabolic, neurohormonal, and especially renal factors are likely to be required.⁵ At present, we do not think that valid data exist to show that SVR induces changes in diastolic function that are able to turn heart failure with reduced ejection fraction into heart failure with preserved ejection fraction!

With specific regard to our article, Dr Buckberg seems to assume “a priori” that a reduction in SV necessarily implies a worsening of hemodynamics and, parenthetically, cardiovascular equilibrium. In our article, and more precisely in our previous response to

Parachuri, we emphasized that post-SVR SV reduction was not present in all patients but only in those with resting SV higher than normal, whereas in patients with reduced resting SV, SVR induces an increase in SV. We hypothesized that the resting SV higher than normal and proportional to end-diastolic volume increase was due to “congestion” in a vision of backward heart failure mechanism, whereas the increase in SV in patients with resting decreased SV was likely due to ventricular afterload reduction after SVR. The message of our article was that SVR, on the basis of our data, tends to normalize resting SV, and this is not at all in disagreement with postoperative clinical improvement.

We think that one should not interpret hemodynamic changes in a biased way, taking into account mainly or only the direction of changes, but should frame them in a correct pathophysiologic picture considering the setting in which changes begin, the meaning, and the mechanism underlying such changes.

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References

1. Di Donato M, Fantini F, Toso A, Castelvich S, Menicanti L, Annet L, et al. Impact of surgical ventricular reconstruction on stroke volume in patients with ischemic cardiomyopathy. *J Thorac Cardiovasc Surg.* 2010;140:1325-31.
2. ten Brinke EA, Klautz RJ, Tulner SA, Verwey HF, Bax JJ, Schalij MJ, et al. Long-term effects of surgical ventricular restoration with additional restrictive mitral annuloplasty and/or coronary artery bypass grafting on left ventricular function: six-month follow-up by pressure-volume loops. *J Thorac Cardiovasc Surg.* 2010;140:1338-44.
3. Di Donato M, Toso A, Dor V, Sabatier M, Barletta G, Menicanti L, et al. Surgical ventricular restoration improves mechanical intraventricular dyssynchrony in ischemic cardiomyopathy. *Circulation.* 2004;109:2536-43.

4. Di Donato M, Menicanti L, Ranucci M, Castelvich S, de Vincentiis C, Salvia J, et al. Effects of surgical ventricular reconstruction on diastolic function at midterm follow-up. *J Thorac Cardiovasc Surg.* 2010;140:285-91.

5. Victor BM, Barron JT. Diastolic heart failure versus diastolic dysfunction: difference in renal function. *Clin Cardiol.* 2010;33:770-4.

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Reply to the Editor:

We thank Dr Buckberg for his thoughtful comments on our article.¹ We obviously agree that a positive correlation (or a “logical” relationship) between clinical findings (in our case New York Heart Association class, quality-of-life score, and 6-minute walking distance) and hemodynamic measurements is reassuring and supportive for the validity and consistency of the entire data set. The implicit and plausible assumption is that patients would be expected to profit from surgical ventricular restoration (SVR) if this intervention somehow improves their cardiovascular function. However, cardiovascular function has many components, and the relative importance of each component on overall cardiovascular status might depend on factors such as the level of exertion.

Clinical parameters provide a highly valuable overall outcome measure, but a positive clinical response to SVR does not necessarily imply that all components of the underlying cardiovascular function have improved. With regard to SVR, theoretical models predict that the intervention is a “balancing act” between positive effects on systolic function and negative effects on diastolic function.² Our findings support this hypothesis, but we also conclude that based on more global parameters, such as cardiac output and stroke work, and the mentioned clinical parameters, the positive effects (improved systolic function, reduced wall stress, and reduced dyssynchrony) apparently outweighed the negative effects (reduced diastolic function) in our patient group. We believe that this type of detailed analysis of cardiovascular