

LETTERS TO THE EDITOR

Batista’s Operation: What Have We Learned?

The recent editorial comment by Dr. Ratcliffe (1) on two articles (2,3) concerning partial left ventriculectomy (PLV) raises some issues that we would like to clarify. In his comment Dr. Ratcliffe questions the accuracy of the conductance catheter method based upon a comparison between our measurements obtained by thermodilution and conductance catheter. The illustration presented (Fig. 1 of his comment) appears to demonstrate poor agreement between the two methods. Unfortunately, this is due to a misinterpretation of the data presented in Table 2 in our article (3). Figure 1 in Dr. Ratcliffe’s comment shows a comparison between stroke volume (SV) measured by thermodilution and by conductance catheter, the latter using the difference between left ventricular (LV) end-diastolic volume (EDV) and end-systolic volume (ESV). However, whereas thermodilution measures effective forward flow, the difference between EDV and ESV also includes both mitral and aortic regurgitant flows. Therefore, the difference between the measurements by the two methods, as plotted on the y-axis of Figure 1, in fact represents the sum of aortic and mitral regurgitant stroke volumes. Thus, the finding that this difference is substantial (mean difference 18 ml) merely reflects the presence of substantial mitral and aortic regurgitation in this patient group.

In retrospect we performed the same analysis as Dr. Ratcliffe, and we subsequently separately analyzed data before and after PLV. Before PLV, mean regurgitant SV was 27 ± 9 ml, whereas post-PLV it was reduced to 12 ± 10 ml, consistent with the aortic valve surgery performed in one patient and mitral valve surgery performed in seven patients. Furthermore, the conductance volumes as presented in Table 2 in our article (3) are, in fact, calibrated by matching thermodilution-derived SV with effective conductance SV (p. 2106 of our article). The latter is obtained as the difference between conductance volumes at the times of dP/dtMIN and dP/dtMAX which largely eliminates the contribution of regurgitant flows. As mentioned in our study, this calibration on the basis of thermodilution was performed in each patient at each condition. Therefore, a comparison between thermodilution and conductance-derived SV, as attempted in the editorial comment, is not meaningful.

A striking observation of the present and other PLV studies is the unchanged SV after the procedure in these patients. One of our main findings, unfortunately not discussed in the editorial comment, was the improvement in mechanical LV synchrony. Mechanical nonuniformity as demonstrated in these patients is an important factor in cardiac dysfunction in heart failure patients. Our results indicate, as demonstrated by the highly significant positive correlation between cardiac index and LV synchrony index, that PLV produces its beneficial effects partly by improving mechanical synchrony. The increase in LV synchrony immediately after PLV may lead to an improved mechanical efficiency of ventricular ejection, compensating an expected decrease in SV.

In our view the occurrence of the marked change in LV segmental asynchrony precludes realistic estimation of the effects of PLV on the Starling relationship (SV vs. sarcomere length), because mean sarcomere lengths before and after PLV are unknown and cannot simply be derived from LV end-diastolic pressure (EDP) or LV EDV due to the change in LV segmental asynchrony. Moreover, the excision of LV mass and concomitant change of LV intracavitary volume also preclude estimation of sarcomere length change from both EDV and EDP.

Finally, it would be interesting to implement LV segmental volume asynchrony in models such as used by Dickstein et al. (4) and by Ratcliffe et al. (5). In general, we agree that numerical models may provide data that are difficult or impossible to obtain in vivo studies. However, studies such as described by Starling et al. (2) and by us (3) will always be required initially to feed the numerical models with input data and ultimately to check whether the results produced by these models are meaningful and consistent with data obtained in the intact heart.

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A Randomized Trial of Multivessel Stent Versus Coronary Bypass

In a recent Journal article, Rodriguez et al. (1) reported that the rates of death and myocardial infarction during long-term follow-up were lower in patients undergoing percutaneous transluminal coronary revascularization (PTCR) than in patients undergoing coronary artery bypass graft (CABG) surgery. This conclusion requires careful scrutiny owing to possible under-reporting of major events.

The study was designed with a composite primary end point that included death, myocardial infarction, cerebrovascular accident and myocardial re-vascularization. Presumably, once a patient experienced a primary end point event, that patient would be withdrawn from further analysis. A patient who underwent a second revascularization procedure and subsequently died would not be reported as a death, for example. In the Rodriguez et al. study, patients randomized to PTCR were more likely to have a
second revascularization procedure than were those assigned to CABG. Subsequent major events occurring in such patients would not be reported. This would bias the mortality and myocardial infarction rates in favor of PTCR.

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REPLY
We disagree with the comments made by Dr. F. James Brennan that major events were underreported in our recent Journal article (1). Although we used a composite end point that included death, myocardial infarction, cerebrovascular accident and myocardial revascularization, we also separately reported mortality, myocardial infarction, repeat revascularization procedures and stroke (1).

For example, as outlined in the Results section, there were thirteen 30-day deaths in the coronary artery bypass graft (CABG) group and two deaths in the percutaneous transluminal coronary revascularization (PTCR) group. After one month, five patients died in the PTCR group and four in the CABG group. These deaths are reflected in the Kaplan-Meier survival curves shown in our Figure 2, demonstrating a better survival in the PTCR group compared with CABG group (96.9% vs. 92.5%, respectively, p < 0.017).

We employed the Kaplan-Meier method to estimate length of survival, freedom from myocardial infarction, freedom from repeat revascularization procedures and freedom from combined events for patients treated with CABG and with PTCR (see our Figs. 2–4). Comparison between groups was performed using the log-rank test. The Kaplan-Meier method is an appropriate means of estimating survival and other major events in our study. Because each of the events (mortality, myocardial infarction and repeat revascularization procedures) was analyzed separately using the Kaplan-Meier method, there was no under-reporting of major events. Although a given patient may have experienced a nonfatal primary end point, he or she was not excluded from the analysis of the other events. The Kaplan-Meier method allows the estimation of survival time of each patient who dies and provides exact survival proportions; this is because it uses exact survival times. The same principle was applied in the calculation of freedom from myocardial infarction and freedom from repeat revascularization procedures.

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Lean Tissue Adjusted Peak Oxygen Consumption in Congestive Heart Failure
Osman et al. (1) recently reported data on the prognosis of 225 patients with heart failure. In their analysis they sought to test the hypothesis that peak oxygen uptake gains prognostic power when expressed per lean body mass. Taking the present and other available data (2,3), we entirely agree that this may in fact be possible. However, we also see a number of problems in the present article with regard to exercise testing, the patient group itself and its very low event rate, as well as the statistical analysis. Therefore, we cannot consider their article to offer proof of its conclusion.

The mean peak oxygen uptake (VO2) of their population of heart failure patients was 16 ml/kg/min. The mean anaerobic threshold was 12.7 ml/kg/min, and the peak heart rate was only 126 beats/min. Although mentioned in their Methods section, the investigators’ data for respiratory exchange ratio at peak exercise, a marker of metabolic stress, are not given anywhere in their report. The investigators chose to present event rates based on 14 deaths and 15 urgent transplants. In other words, the total mortality rate in this population was about 7% during 19 months of follow-up. This study group appears on average to have been in mild heart failure. This low mortality rate is surprising given the mean peak VO2 of 16 ml/kg/min. Even if urgent transplant is included in the end point, the 12-month event rate in patients with peak VO2 ≤14 ml/kg/min is still only about 15%, which seems very low. Using the same cutoff, Mancini et al. (4) reported a mortality rate of 39% to 53% in 12 months. Taken together, this raises concern about the validity of their exercise tests, suggesting that an adequate VO2 may not have been reached.

Whenever one examines the data, the total event number of 29 appears too small to perform extensive statistical analyses. The main statistical analysis in the Osman et al. (1) article is concerned with comparing peak VO2/weight versus peak VO2/lean weight as a continuous or dichotomous variable, respectively. Unfortunately, the receiver-operator curves (ROCs) for the two continuous variables are not presented, and the arguments are based on somewhat different chi-square (20.53 vs. 17.17) and p values (0.0001 vs. 0.0007). No statistical comparison for the two continuous variables is provided. Also, the comparison of the two cutoffs appears difficult to interpret.

First, the data in their Table 2 (1) do not specify the follow-up period to which they relate. Second, calculation of the ROC is reserved for variables with many different levels. In their Table 2,