Moreover, the adverse effect of PPM on pulmonary arterial pressure may in large part occur in the long term. Thus the early postoperative pulmonary arterial pressure is obviously not an appropriate and reliable end point for adequate assessment of the effects of PPM. As opposed to the results of this study, three recent previous studies have reported that PPM is associated with a higher incidence of persistent pulmonary hypertension after mitral valve replacement.²⁻⁴

The number of patients is far too small and the follow-up too short to draw any meaningful conclusion with regard to the impact of PPM on operative and late mortalities, on the persistence of pulmonary hypertension, or on any other clinical outcomes. Importantly, there is a major discrepancy between the data reported in this small series of patients with very short follow-up and the data reported by two independent groups of investigators in much larger series of patients (>800) with more than 10 years of follow-up.^{3,4} These larger studies concluded that mitral PPM, and especially severe PPM, is an independent predictor of the persistence of pulmonary hypertension, the occurrence of congestive heart failure, and late mortality.

In light of the important limitations of this study and of the results of previous studies,²⁻⁴ the results and conclusions of the article of Totaro and Argano¹ cannot be accepted at face value. We believe that among several prosthesis models that are equivalent in terms of durability and thromboresistance the surgeon should logically select the one with the largest possible EOA in a given annulus size to minimize the risk of PPM and associated complications after mitral valve replacement.

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References

1. Totaro P, Argano V. Patient-prosthesis mismatch after mitral valve replacement: myth or reality? *J Thorac Cardiovasc Surg.* 2007; 134:697-701.
2. Li M, Dumesnil JG, Mathieu P, Pibarot P. Impact of valve prosthesis-patient mismatch on pulmonary arterial pressure after mitral valve replacement. *J Am Coll Cardiol.* 2005;45: 1034-40.
3. Magne J, Mathieu P, Dumesnil JG, Tanné D, Dagenais F, Doyle D, et al. Impact of prosthesis mismatch on survival after mitral valve replacement. *Circulation.* 2007;115:1417-25.
4. Lam BK, Chan V, Hendry P, Ruel M, Masters R, Bedard P, et al. The impact of patient-prosthesis mismatch on late outcomes after mitral valve replacement. *J Thorac Cardiovasc Surg.* 2007;133:1464-73.
5. Pibarot P, Dumesnil JG. Prosthesis-patient mismatch: definition, clinical impact, and prevention. *Heart.* 2006;92:1022-9.
6. Bleiziffer S, Eichinger WB, Hettich I, Guenzinger R, Ruzicka D, Bauernschmitt R, et al. Prediction of valve prosthesis-patient mismatch prior to aortic valve replacement: which is the best method? *Heart.* 2007;93: 615-20.
7. Pibarot P, Dumesnil JG, Cartier PC, Métras J, Lemieux M. Patient-prosthesis mismatch can be predicted at the time of operation. *Ann Thorac Surg.* 2001;71(5 Suppl):S265-8.
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Reply to the Editor:

We thank Pibarot and coauthors for their comments regarding our article.¹ It is surely a privilege that such distinguished colleagues take an interest in our study. Nevertheless, we found some of the comments inappropriate.

First, as far as the title was concerned, it was obviously provocative and was chosen just to underline an important matter related to this issue. As Pibarot and coauthors stressed in their comment, patient-prosthesis mismatch (PPM) is a hemodynamic phenomenon and therefore should be diagnosed on the basis of hemodynamic parameters related to a specific patient. This is the key point related to PPM after mitral valve replacement. In other words, to evaluate clearly the impact of PPM on functional and clinical outcomes, we have to be sure that we are comparing patients with PPM to patients without PPM.

As we clearly stated, this was the main objective of our study, which was indeed focused on the identification of patients with real PPM and on the evaluation of accuracy of prediction and diagnosis of PPM with data not obtained in vivo postoperatively and therefore not related to a specific patient.

In their comments, Pibarot and coauthors express serious concerns about the validity of our Doppler echocardiographic data, especially those related to effective orifice area (EOA). We would stress that the methodology we used (continuity equation method) was validated for assessing mitral

valve bioprosthesis by their own group²; they also confirmed the good correlation between the mean in vitro and in vivo areas for mitral prostheses. In our hands in vivo postoperative measured EOA was indeed larger than the EOA measured in vitro by the manufacturer. We stressed such interesting results in our discussion, and we also stressed that these findings were surely unexpected on the basis of what has clearly been demonstrated for aortic prostheses. We do believe, however, that our results deserve a further analysis rather than mere criticism. It is notable that, despite few data on postoperative hemodynamic performance after mitral valve replacement being available, Firstenberg and colleagues³ in 2001 reported echocardiographic data consistent with our findings. They specifically evaluated postoperative in vivo hemodynamic performances of the mitral Carpentier Edwards Perimount valve, and the postoperative in vivo EOAs they reported were consistent with our data. We do stress that they also reported a discrete variability (expressed in terms of SD), once more similar to our own findings. We are still collecting and analyzing our data for better evaluation of the reason for such variability, but we do believe that it is related to the fact that the postoperative prosthetic valve in vivo EOA depends on other physiopathologic parameters than simple prosthesis size. Different sensitivities of mitral valve hemodynamics to chronotropic conditions, which may vary extensively from one patient to another was furthermore previously noted by Pibarot and Dumesnil⁴ themselves in their *Journal* editorial of June 2007. Further studies addressing postoperative in vivo hemodynamic parameters could be more helpful in better understanding such variability, rather than criticizing our results and doubting their validity. In contrast to what they stated in their comments, we did not mention at all the “the complete absence of correlation between the EOA and the trans-prosthetic gradient” (which was, however, previously shown by Firstenberg and co-workers³), but rather the absence of correlation between increased prosthesis size and improved postoperative hemodynamic performance. A similar concept was also previously presented by Badano and associates⁵ and therefore is not a unique finding of our study.

We do not agree at all with Pibarot and coauthors' concerns about the “large proportion of the patients included in this series

[who] were misclassified with respect to the presence or absence of PPM." Misclassification is definitively more frequent when the classification is based on fixed data, either from in vitro study or previously published studies, and not when the classification is based on real in vivo data that reflect the hemodynamic condition of a specific patient. The question of why some patients with the same prosthesis size had such different postoperative EOAs is surely matter for further discussion and evaluation, but postoperative in vivo EOA must anyway be considered the criterion standard for PPM identification and cannot be considered simply misleading just because different patients have different EOAs.

The final two comments of Pibarot and coauthors are all related to the clinical effect of PPM. With respect to the effect of PPM on pulmonary arterial pressure, Pibarot and coauthors simply underlined what we had already stated in our Discussion section. Furthermore, among three recent studies quoted by Pibarot and coauthors, the one from Li and colleagues⁶ was also a key reference in our study, and the two from Magne and coauthors⁷ and Lam and coworkers⁸ were published well after we sent our article for consideration for publication in the *Journal* (February 2007, as clearly reported on the first page¹).

With respect to clinical impact of PPM in terms of operative and late mortalities, we also clearly stated in the Discussion section that our study could not be elucidative because only 8 patients had PPM. Clinical relevance of PPM has to be better elucidated and surely represents an important matter. We would like to stress, however, that once more the evidence must come from a complete evaluation and analysis of different experiences published in the literature. Recent experiences quoted by Pibarot and coauthors^{7,8} clearly identify PPM as risk factor for poor outcome. Previous experience from Lorusso and associates⁹ and Ruel and coworkers,¹⁰ however, did not confirm different outcome in patients receiving small prostheses. Because Lam and coworkers' definition of patients with PPM was not based on postoperative in vivo data,⁸ patients classified as having PPM should mainly be patients receiving small prostheses. These data are therefore in contrast and deserve further evaluation and further studies. In conclusion, in our opinion only a multicenter, prospective

study that is based on in vivo postoperative evaluation of PPM could reach a better level of evidence regarding the exact definition of PPM at the mitral level, the real incidence of postoperative mitral PPM, and its clinical impact. Our study was not meant to contradict results from other researchers but simply to warn about the real potential for misclassification of patients has having PPM solely on the basis of fixed data rather than dynamic data specific to that patient. For the patient receiving a mitral prosthesis, specific postoperative in vivo evaluation should be mandatory to evaluate the real incidence of mitral PPM.

Finally, we would respectfully underline that the appropriate scientific controversy should call for a different attitude and approach to other investigators simply driven by the same enthusiasm for proven data about a scientific aspect whose explanation, unless proven, is usually not in the hands of a unique group of investigators.

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References

1. Totaro P, Argano V. Patient-prosthesis mismatch after mitral valve replacement: myth or reality? *J Thorac Cardiovasc Surg.* 2007; 134:697-701.
2. Dumesnil JG, Honos GM, Lemieux M, Beauchemin J. Validation and applications of mitral prosthetic valvular area calculated by Doppler echocardiography. *Am J Cardiol.* 1990;65:1443-8.
3. Firstenberg MS, Morehead AJ, Thomas JD, Smedira NG, Cosgrove DM 3rd, Marchand MA. Short-term hemodynamic performance of the mitral Carpentier-Edwards PERIMOUNT pericardial valve. Carpentier-Edwards PERIMOUNT Investigators. *Ann Thorac Surg.* 2001;71(5 Suppl):S285-8.
4. Pibarot P, Dumesnil JG. Prosthesis-patient mismatch in the mitral position: old concept, new evidences. *J Thorac Cardiovasc Surg.* 2007;133:1405-8.
5. Badano L, Mocchegiani R, Bertoli D, DeGaetano G, Carratino L, Pasetti L, et al. Normal echocardiographic characteristics of the Sorin Bicarbon bileaflet prosthetic heart valve in the mitral and aortic positions. *J Am Soc Echocardiogr.* 1997;10:632-43.
6. Li M, Dumesnil JG, Mathieu P, Pibarot P. Impact of valve prosthesis-patient mismatch on pulmonary arterial pressure after mitral valve replacement. *J Am Coll Cardiol.* 2005;45:1034-40.
7. Lam BK, Chan V, Hendry P, Ruel M, Masters R, Bedard P, et al. The impact of patient-prosthesis mismatch on late outcomes after mitral valve replacement. *J Thorac Cardiovasc Surg.* 2007;133:1464-73.
8. Magne J, Mathieu P, Dumesnil JG, Tanné D, Dagenais F, Doyle D, et al. Impact of prosthesis mismatch on survival after mitral valve replacement. *Circulation.* 2007;115:1417-25.
9. Lorusso R, Beghi C, Gerometta PS, Russo C, Gherli T, Casari S, et al. Mitral valve replacement with small prosthesis (size 23-25) in adult subjects: long-term postoperative results from a multicentre retrospective study. *Circulation.* 2002;106(abstr):674.
10. Ruel M, Rubens F, Masters R, Pipe AL, Bedard P, Mesana TG. Late incidence and predictors of persistent or recurrent heart failure in patients with mitral prosthetic valves. *J Thorac Cardiovasc Surg.* 2004; 128:278-83.

doi:10.1016/j.jtcvs.2007.10.028

Thoracic epidural anesthesia and atrial fibrillation after coronary bypass grafting To the Editor:

I read with great interest the recent article by Dr Bakhtiary and colleagues¹ detailing the protective effect of high thoracic epidural anesthesia against atrial fibrillation after off-pump coronary bypass grafting. The following clinical questions arise as a result of this fascinating study:

1. Why was the placement of the thoracic epidural catheters the day before the operation?
2. Were there any thoracic epidural placements complicated by significant puncture-site bleeding? What was the protocol for managing this uncommon but important event? Was another epidural space selected?
3. How was the epidural catheter tested to ensure effective bilateral thoracic anesthesia?
4. What was the management of postoperative β -blockade? Was β -blocker withdrawal a possible confounder in this study?
5. Is there any explanation for the observation that women were more likely to experience nausea and vomiting in the intervention group?
6. What was the power calculation for this study based on the existing literature? What is the probability of a type I error, a type II error, or both?