I congratulate the authors again on a most thought-provoking study. I look forward to their feedback about these considerations.

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Reply to the Editor:

We appreciate the comments provided by Dr Augoustides in regard to our recent article "Ventilatory Dependency After Cardiovascular Surgery." Our article attempts to define the risks for ventilator dependency and determine the outcomes of patients who develop this problem after their index cardiovascular surgery. Two inquiries were made (as points [a] and [c] are closely related).

We were surprised ourselves to find that there has been a continuing trend toward a lower prevalence of ventilator dependency at our institute. A declining trend was noted in previous studies from our institute published several years before. That this trend has continued through the current study has both thrilled and perplexed us. It was impossible for us to determine which quantifiable variables might be responsible for this trend because of a variety of confounding factors, not the least of which is time itself. What is even more interesting is that this phenomenon has occurred in the setting of increasing complexity and acuity of illness. It is our suspicion that several coincident changes might be responsible. There has been a steady improvement in myocardial

protection over the years and a progressive increase of intraoperative echocardiography use; these factors likely promote more rapid myocardial recovery. Less narcotics are being given intraoperatively, and this may facilitate earlier separation from mechanical ventilatory support. In addition, there has been a noticeable increase in intensivist staffing, such that there is seemingly more planning and continuity of care in the intensive care unit, which again, might favorably affect weaning.

Second, aprotinin is used sparingly at our institution (in <1% of patients). This is, in part, secondary to cost, but also for concerns regarding thrombotic complication and renal dysfunction, all without clearly demonstrable benefit. There are 2 populations for whom we do use aprotinin on more occasions: patients with ventricular-assist devices and patients undergoing a second cardiac transplant. These groups were excluded from our study.

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Prosthesis-patient mismatch after mitral valve replacement: Back to reality

To the Editor:

We read with interest the article of Totaro and Argano.¹ We have, however, several concerns with regard to the validity of the results and conclusions presented in this article. First, the title of the article, "Patient-Prosthesis Mismatch after Mitral Valve Replacement: Myth or Reality?" is inappropriate. Mitral prosthesis-patient mismatch (PPM) is equivalent to a residual mitral stenosis, related to the fact that most prosthetic valves have a hemodynamic performance, and thus a valve effective orifice area (EOA), that is inferior to that of the normal native valve. Thus concluding that PPM is a myth would be equivalent to saying that mitral stenosis (or aortic stenosis for aortic PPM) does not exist, and that this is a benign phenomenon, which is of course not the case. In this regard, several studies have demonstrated that PPM is a frequent hemodynamic phenomenon after mitral or aortic valve replacement.²⁻⁵ The important question is rather to determine the impact of PPM on the hemodynamic, functional, and

clinical outcomes, and at which degree of severity and in which categories of patients this impact of PPM becomes statistically significant and clinically relevant. Unfortunately, the data provided by Totaro and Argano¹ do not permit an answer to these important questions.

There are serious concerns about the validity of the Doppler echocardiographic data, and especially of those of the valve EOA. This is a crucial aspect, because the identification and quantification of PPM are based on these data. First, it is intriguing to see that the EOAs measured in vivo by Doppler echocardiography, especially for the 25- and 27-mm valves, were larger than the EOAs measured in vitro by the manufacturer. A recent study has indeed demonstrated that, as opposed to the observation in this study, the in vitro EOAs provided by the manufacturer grossly overestimate the in vivo EOAs and are thus not valid for prediction of PPM.⁶ Totaro and Argano also used the label prosthesis size as a surrogate for PPM, whereas previous studies have shown that this parameter is not valid for identification of PPM and prediction of its hemodynamic and clinical consequences.^{3,6,7}

Moreover, the huge variability in the EOA measurements for a given prosthesis size (from 1.0 to 4.9 cm² for the 29-mm prostheses!), the complete absence of correlation between the EOA and the transprosthetic gradient, and the recording of high transprosthetic gradients (>15 mm Hg) in several patients despite the calculation of large EOAs and indexed EOAs (see Figure 3 of the article¹) further support the concerns regarding the validity of the EOA measurements and thus the identification of PPM. The presence of gradients greater than 15 mm Hg in the mitral position definitely cannot be considered as "favorable hemodynamics," as concluded by Totaro and Argano.¹ In fine, these observations suggest that a large proportion of the patients included in this series were misclassified with respect to the presence or absence of PPM.

Totaro and Argano only measured the valve hemodynamics and systolic pulmonary arterial pressure at predischARGE examination or at 30 days. It is well known that the measurements of valve EOA, transvalvular gradients, and pulmonary pressure in the early postoperative period are often unreliable because of the poor acoustic window, hyperdynamic state, or flow acceleration in the left ventricular outflow tract.

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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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