increase at 2–3 years after surgery. They concluded that the risk of early SVD is high for the Mitroflow prosthesis, especially if the prosthesis is small. Indeed, severe SVD was observed in 44% of patients with prosthesis Size 19 and in 23% of patients with Size 21. In a recently published article, we reviewed 459 patients undergoing aortic valve replacement using the Mitroflow prosthesis [2]. During a 6-year follow-up period, the incidence of moderate and severe SVD was 12.7% (43 patients) and 4.1% (14 patients), respectively. Although the freedom from SVD was approximately the same in both experiences, we observed an increased rate of early degeneration in the presence of patient–prosthesis mismatch, which was unfortunately common in our series. Overall, the incidence of SVD is higher than that reported for other biological valve prostheses, which are expected to be associated with an SVD rate of less than 10% at 10 years [3]. Surprisingly, we faced a new and unexpected problem: the more recent DL model, pretreated with octanediol to prevent heterotopic calcification of leaflets [4], appeared to deteriorate even earlier in comparison with the untreated XL model. We have found that the DL model is a predictor of SVD, with a 4-year freedom from moderate SVD and severe SVD of 71 ± 5% and 78 ± 6% vs 92 ± 3% and 96 ± 2% in the XL model, respectively. This could be possibly due to a biophysical or biochemical modification induced by the so-called PRT process itself. We wonder whether Issa *et al.* noticed a similar worse behaviour in the DL or the Crown models.

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Reply to Nardi *et al.*

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We thank Nardi *et al.* [1] for their interest in our recent article [2] and for raising the important and interesting question of the impact of the Mitroflow bioprosthesis model on occurrence of structural valve degeneration (SVD). In our study, 748 (48%) patients received a Model 12A prosthesis, 708 (46%) a Model LXA prosthesis and 98 (6%) patients a Model DL. Based on the cross-sectional design of our study and the fact that different models were used in different time periods with Model 12A used from the start of the study from January 2000 to March 2006, model LAX thereafter, whereas model DL only was used for the last few months, the study design is not suited to assess differences in SVD between models. However, a reoperation was performed in 40 patients due to SVD prior to the cross-sectional investigation. These patients were treated based on significant SVD and symptoms and, thus, unbiased of the cross-sectional investigation. Median follow-up after aortic valve surgery for Model 12A was 11.0 (interquartile range 9.8–12.1) years when compared with median follow-up of 5.0 (3.0–7.3) years for Model LXA. Given the considerably higher prosthesis age, it was expected that numerically more patients with a prosthesis Model 12A underwent a reoperation ($n = 32$) when compared with 8 patients with a prosthesis Model LXA. Despite this, we found a clear trend that a reoperation for SVD occurred earlier with Model LXA than Model 12A.

Thus, in agreement with the observation by Nardi *et al.*, our data, with the limitation of an overall low number of reoperations, suggest that the risk of significant SVD may occur earlier with the more recent model. Unfortunately, our data do not allow any insight into the risk of developing SVD with the DL model.

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