EDITORIAL

# Prevention of valve prosthesis—patient mismatch before aortic valve replacement: does it matter and is it feasible?

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In this issue of *Heart*, Bleiziffer *et al*[1] present a study evaluating the best method to predict valve prosthesis–patient mismatch (VP–PM) before aortic valve replacement. As they point out, VP–PM remains a controversial issue, and the use of different methods to identify VP–PM might have contributed to the contradictory results reported in previous studies. Their findings are important from two standpoints: (1) they demonstrate that not all methods are equally efficient in this regard and that in fact some approaches that purport to attain this objective are for all practical purposes useless; (2) they further confirm that when the right method is used, VP–PM can be predicted and also largely prevented by using a simple strategy at the time of operation.

## DEFINITION OF VP–PM

The term VP–PM was first proposed in 1978 by Rahimtoola.[2] VP–PM occurs when the effective orifice area (EOA) of the prosthesis is too small in relation to the patient's body size, resulting in an abnormally high postoperative gradient.[3 4] Hence, the parameter generally used to identify VP–PM is the EOA of the prosthesis indexed for the patient's body surface area. The rationale behind the normalisation of the EOA for body surface area is to account for cardiac output requirements, since transvalvular pressure gradients are essentially determined by the EOA and transvalvular flow, which in turn is largely determined by body size. In the aortic position, the threshold for VP–PM is generally defined as an indexed EOA $\leqslant 0.85$ cm$^2$/m$^2$ and it is defined as moderate when between 0.65 and 0.85 cm$^2$/m$^2$ and as severe when $\leqslant 0.65$ cm$^2$/m$^2$. Moderate VP–PM may be quite prevalent (20–70%) in patients undergoing aortic valve replacement (AVR), whereas the prevalence of severe VP–PM ranges from 2% to 11% depending on the series.[4] It should be emphasised that the original descriptions of VP–PM were based on the measurement of the in vivo EOA, and attempts to use the internal geometric area or the in vitro EOA as a substitute for this parameter have come subsequently and have never really been validated with regard to predicting postoperative haemodynamics and/or clinical outcomes.

## DOES VP–PM MATTER?

There is now a strong body of evidence showing that, when it is defined on the basis of the right parameter, VP–PM is an important risk factor with regard to clinical outcomes.[4 5] Indeed, VP–PM in the aortic position is associated with less improvement in symptoms and functional class,[6] lesser regression of left ventricular hypertrophy[7] and more adverse cardiac events.[6 8–11] Moreover, VP–PM has a significant impact on both short-term[12] and long-term mortality,[8 10 11 13] particularly if left ventricular (LV) dysfunction is present.

## WHEN DOES VP–PM MATTER THE MOST?

### Patients with impaired left ventricular function

Recent studies have reported a strong interaction between VP–PM and depressed LV function with regard to occurrence of heart failure as well as early and late mortality after AVR.[8 11 12] These findings are consistent with the fact that an increased haemodynamic burden is less well tolerated by a poorly functioning ventricle than by a normal ventricle. Hence, every effort should be made to avoid VP–PM in high-risk patients with depressed LV function.

### Young patients

Moon *et al*[14] recently reported that the impact of VP–PM is more pronounced in young patients than in older ones.[14] This finding might be related to the fact that younger patients have higher cardiac output requirements and that they are exposed to the risk of VP–PM for a longer period of time.

### Athlete patients

In a study recently presented by Bleiziffer *et al*[15] at the 2006 meeting of the European Association of Cardio-Thoracic Surgeons, VP–PM was identified as one of the most powerful independent predictors of maximal exercise capacity after AVR. In athlete patients, the objective should thus be to optimise the postoperative indexed EOA in order to accommodate the larger cardiac output requirements of these patients under exercise conditions, and in this context an indexed EOA $\geqslant 1.0$ cm$^2$/m$^2$ would probably appear to be a realistic value.

## DEFINITION OF VP–PM: THE PARAMETER DOES MATTER

The article of Bleiziffer *et al* reveals that the best parameter to predict VP–PM at the time of operation is the projected indexed EOA derived from reference values published in the literature.[1] In contrast, the indexed EOA projected from the manufacturer's in vitro data and the indexed geometric orifice area (GOA) had very low sensitivities (0–26%), indicating that these parameters are not valid to detect VP–PM. The only

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manufacturer's chart that had a good performance to predict VP–PM was the one derived from in vivo echocardiographic EOA data. Hence, it is not surprising that previous studies in which VP–PM was defined with the use of the indexed GOA or the indexed EOA derived from manufacturer's in vitro data found no association between VP–PM and clinical outcomes.[16–18] However, the vast majority of previous studies that have used the indexed EOA directly measured after operation[13] or estimated at the time of operation from reference in vivo EOA values[6–12] reported that VP–PM is associated with worse postoperative haemodynamics and outcomes. Hence, these data are compelling in that they largely contribute to reconcile previous discrepancies, and they confirm that the only valid reference values diagnose and predict VP–PM are those derived from in vivo measurements of the indexed EOA.

A very important point made by the authors within this context is that the reference values should be as reliable as possible, which is probably best achieved by using values derived from large, preferably multi-centre, series with a sufficient number of patients for each type and size of prosthesis (method 4 in the paper). However, and as they also point out, caution should be exercised when measurements originate from a single laboratory, as there may be a bias in measurement, and the ideal situation would appear to be concordant measurements originating from various laboratories.

One of the most relevant results of Bleiziffer *et al*'s study is the observation that the charts provided by manufacturers were inaccurate in two out of three cases. Clearly, VP–PM is now becoming widely accepted as an important risk factor, which can largely be prevented by the use of a prospective strategy at the time of operation, as shown again by the present results. In this context, it would appear that manufacturers have a clear responsibility in providing and disseminating accurate reference values so that the prediction and prevention of VP–PM can be easily incorporated into the clinical decision-making process. Hence, the charts should be as reliable and user friendly as possible, and should be conceived as a tool to improve patient outcome rather than as a sales argument.

### Obesity: a confounding variable?

The prevalence of obesity has increased dramatically in the past decades in the western world, especially in the United States, and it is often questioned whether the utilisation of the body surface area for the normalisation of EOA may not overestimate the degree of VP–PM in obese patients. The differences in the prevalence of obesity in the patient populations may also contribute in explaining the discrepancies observed among previous studies. The indexation of EOA to height or a power of height as a reflection of lean body mass does not, however, appear to be a solution, because overweight/obesity is generally associated with a 10–30% increase in cardiac output requirements. Future studies will be necessary to determine if the indexation of EOA cannot be improved or refined in the case of obese patients

## PREVENTION OF VP–PM: IS IT FEASIBLE?

The study of Bleiziffer *et al* also provides further compelling evidence that the prevention of VP–PM is feasible.[1] They compared the prevalence of VP–PM in their series of patients before and after the introduction of systematic calculation of the projected indexed EOA at the time of operation. The prevalence of moderate VP–PM was reduced from 44% to 30%, and that of severe VP–PM from 9% to 1%. This important finding supports the notion that being aware of the risk of VP–PM at the time of operation can influence the selection of the prosthesis and result in a substantial reduction in the prevalence of VP–PM. Hence, it would appear that there is a significant advantage in systematically calculating the projected indexed EOA of the prosthesis to be inserted, and, in the case of anticipated VP–PM, to consider alternate procedures such as aortic root enlargement or insertion of a better performing valve substitute (eg, supra-annular bioprostheses, stentless valves, newer generation mechanical valves, homografts or Ross operation). Validation of such a strategy had also been previously shown by Castro *et al*.[19]

The information of the projected indexed EOA can easily be incorporated within the clinical decision making process, and utilised in view of the other pertinent clinical factors such as age, level of physical activity, status of LV function and concomitant procedures. For instance, if one projects moderate VP–PM in an elderly patient with reduced physical activity and normal LV function, it might be estimated that the benefits of doing an alternate procedure to avoid VP–PM are outweighed by the inherent risks or disadvantages of doing such a procedure. However, the prevention of VP–PM becomes a mandatory consideration if there is evidence of impaired LV function.

## CONCLUSION

VP–PM is associated with worse haemodynamics, reduced regression of LV hypertrophy, more cardiac events and increased short-term and long-term mortality after valve replacement. The study of Bleiziffer *et al* provides further evidence that the prevention of aortic VP–PM is feasible, and that the projected indexed EOA should routinely be calculated at the time of operation. This study also shows that the indexed GOA or the indexed EOA derived from manufacturer's in vitro data are not valid to project the postoperative indexed EOA. In this context, manufacturers should be strongly encouraged to provide user-friendly charts based on accurate reference values so that the prediction and prevention of VP–PM can be advantageously incorporated into the clinical decision making process and hence be useful in improving patient clinical outcomes.


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# IMAGES IN CARDIOLOGY

## Real-time three-dimensional echocardiographic diagnosis of postmyocardial infarction ventricular septal defect and guidance of transcatheter closure

A 51-year-old woman with no history of heart disease presented to the emergency room complaining of chest pain of 48 h duration and sudden onset of severe dyspnoea. The blood pressure was 92/60 mm Hg and heart rate was 105 beats per minute. Auscultation showed bilateral inspiratory rales and a grade 3/6 harsh, holosystolic murmur along the left sternal border. An ECG showed ST-segment elevation in leads $V_2$–$V_5$. Real-time three-dimensional (RT3-D) transthoracic echocardiography disclosed a 16 mm diameter through-and-through infarct ventricular septal defect (VSD; panels A, B). The patient was taken immediately to the cardiac catheterisation laboratory. Coronary angiography revealed an occluded proximal left anterior descending artery without collateral circulation. Intra-aortic balloon pump counter-pulsation failed to improve the haemodynamic status. With the patient in cardiogenic shock, a decision was made for percutaneous transcatheter closure as an alternative to urgent surgical repair. A 22 mm Amplatzer occluder device (AGA Medical Corporation, Minneapolis, Minnesota, USA) was placed via the femoral vein under combined fluoroscopic and RT3-D echocardiographic guidance (panel C). Careful imaging was needed to identify ventricular septal tissue positioned between both discs. Placement of the occluder resulted in immediate clinical improvement and a decrease in the pulmonary artery oxygen saturation from 80% to 58%. After 6 weeks, the patient underwent successful surgical repair of a persistent small residual shunt for prognostic reasons.

Our case illustrates the clinical usefulness of RT3-D echocardiography in defining the exact location of a post-myocardial infarction VSD, guiding the interventional closure, and immediately evaluating the result.

To view video footage visit the *Heart* website–http://heart.bmj.com/supplemental

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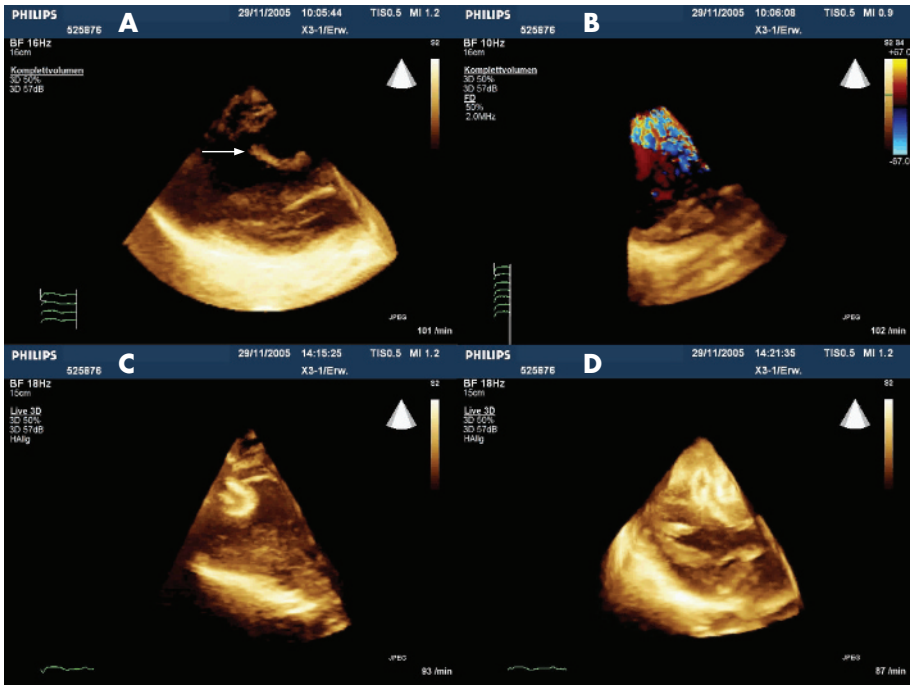


Real-time three-dimensional echocardiography. (A) Parasternal long-axis view locating the through-and-through defect to the mid anteroseptal segment (arrow). (B) Colour flow imaging showing a large left-to-right shunt across the defect. (C) Deployment of both discs with the device still attached to the delivery cable. (D) Appropriate device position after release.