HEART REVIEW

Prosthesis-patient mismatch: definition, clinical impact, and prevention

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Prosthesis-patient mismatch (PPM) is present when the effective orifice area of the inserted prosthetic valve is too small in relation to body size. Its main haemodynamic consequence is to generate higher than expected gradients through normally functioning prosthetic valves. This review updates the present knowledge about the impact of PPM on clinical outcomes. PPM is common (20–70% of aortic valve replacements) and has been shown to be associated with worse haemodynamic function, less regression of left ventricular hypertrophy, more cardiac events, and lower survival. Moreover, as opposed to most other risk factors, PPM can largely be prevented by using a prospective strategy at the time of operation.

Prosthesis-patient mismatch (PPM) was first described in 1978 by Rahimtoola as follows: ''Mismatch can be considered to be present when the effective prosthetic valve area, after insertion into the patient, is less than that of a normal human valve''.[1] Inherent in this concept is that a smaller than expected effective orifice area (EOA) in relation to the patient's body surface area (BSA) will result in higher transvalvar gradients. This is best exemplified by the hydraulic equation $TPG = Q^2/[k \times EOA^2]$, which shows that the transvalvar pressure gradient (TPG) is directly related to the square of transvalvar flow (Q) and inversely related to the square of the valve EOA; k is a constant. Hence, the EOA must be proportionate to flow requirement for gradients to remain low. At rest, transvalvar flow is largely related to cardiac output, which in turn is determined by BSA. PPM thus occurs when the EOA of the prosthetic valve is too small in relation to a patient's body size.[1–4] The immediate consequence is the persistence of abnormally high TPGs.

For instance, assuming a normal cardiac index of 3 l/min/m^2, the implantation of a prosthesis with an EOA of 1.3 cm^2 in a patient with a BSA of 1.5 m^2 will theoretically result in a mean TPG of about 13 mm Hg. The mean TPG will theoretically be 35 mm Hg if the same prosthesis is implanted in a patient with a BSA of 2.5 m^2 (table 1[5]). Moreover, the difference in TPGs between these two patients would be even more important during exercise, given that gradients are a square function of flow.

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PARAMETER USED TO DEFINE MISMATCH

Consistent with these physiological considerations, the parameter used to characterise PPM is the indexed EOA—that is, the EOA of the prosthesis divided by the patient's BSA. The indexed EOA is in fact the only parameter that has been found to consistently correlate with postoperative gradients. Figure 1 shows that the relation between the TPG and the indexed EOA is curvilinear and that gradients increase exponentially when the indexed EOA is $\leqslant 0.8$ to 0.9 cm^2/m^2.[6] On the basis of this relation, an indexed EOA $\leqslant 0.85$ cm^2/m^2 is generally regarded as the threshold for PPM in the aortic position with values between 0.65–0.85 cm^2/m^2 being classified as moderate PPM and those < 0.65 cm^2/m^2 as severe PPM.[2 3 6–8] Such categorisation is important because the impact of PPM on clinical outcomes increases with severity. Depending on studies, the reported prevalence of moderate PPM varies between 20–70%, whereas that of severe PPM is between 2% and 11%.[6–20]

It should be noted that some authors have attempted to characterise PPM by using the internal geometric area (IGA) of the prosthesis rather than the EOA because IGA is more reproducible. The IGA is a static manufacturing specification based on the ex vivo measurement of the diameter of the prosthesis. The criteria used for its measurement unfortunately differ from one type of prosthesis to another so that, for instance, the IGA grossly overestimates the EOA but to a much larger extent in the case of a bioprosthesis than in the case of a mechanical prosthesis (fig 2).[18 19] Hence, the relation between IGA and EOA varies extensively depending on the type and size of prosthesis and it has been shown that the indexed IGA cannot be used to predict postoperative gradients (fig 3A).[14 19–21] This observation is further corroborated by the recent data of Koch *et al*[21] that, as compared with homografts, pericardial valves have similar values for indexed IGA but more or less two-fold values for peak and mean gradients. The same authors also found no relation between the indexed IGA and functional improvement after an aortic valve replacement (AVR). As well, most studies that have used the indexed IGA have failed to find any significant relation between this parameter and adverse

Abbreviations: AVR, aortic valve replacement; BSA, body surface area; CFR, coronary flow reserve; EOA, effective orifice area; IGA, internal geometric area; LV, left ventricular; PPM, prosthesis-patient mismatch; TPG, transvalvar pressure gradient

Table 1 Theoretical comparison of mean transvalvar pressure gradient in five hypothetical patients receiving the same prosthetic valve but having different body surface areas

	Patient number				
	1	2	3	4	5
Body surface area (m^2)	1.5	1.75	2.0	2.25	2.5
Cardiac output (l/min)	4.5	5.25	6.0	6.75	7.5
Valve EOA (cm^2)	1.3	1.3	1.3	1.3	1.3
Mean pressure gradient (mm Hg)	13	17	22	28	35

For this simulation, mean pressure gradient was calculated assuming a cardiac index of 3 l/min/m^2, a heart rate of 65 beats/min, and a systolic ejection time of 300 ms.
EOA, effective orifice area.
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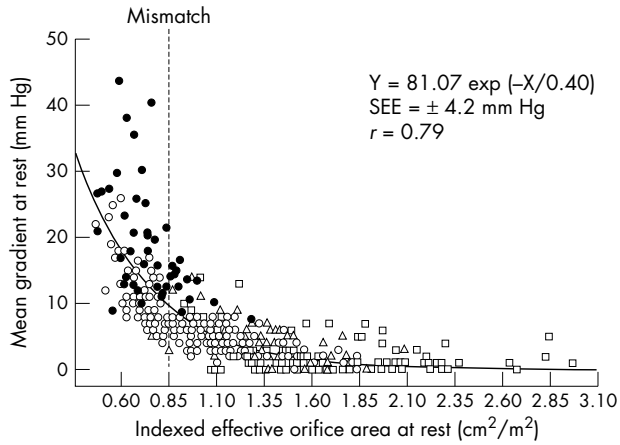

Figure 1 Correlation between mean transvalvar gradient and indexed effective orifice area in patients with a stented bioprosthesis (n = 51; solid circles), a stentless bioprosthesis (n = 194; open circles), an aortic homograft (n = 55; triangles), and a pulmonary autograft (n = 96; squares). Several points overlap. Reproduced from Pibarot and Dumesnil[6] with permission of the American College of Cardiology.

clinical outcomes (table 2).[21–25] This should, however, come as no surprise since, as mentioned, the indexed IGA does not bear any relation whatsoever to postoperative haemodynamic function.[14 19–21] In contrast, the indexed EOA has consistently been shown to correlate with postoperative gradients (fig 1, fig 3B), as well as being highly predictive of adverse outcomes (table 2).[2 5–11 13–20 26–30]

In view of the above, the following points need to be emphasised. Firstly, it is not the size (labelled size or IGA) of the prosthesis that matters but rather its EOA and in whom you implant it. Secondly, the only parameter yet shown to be valid to define PPM is the indexed EOA. Thirdly, the indexed IGA and labelled valve size cannot be used to identify PPM or to characterise its severity. To avoid any confusion about the interpretation of results of the different studies, the terminology used to describe these phenomena also apparently must be consistent and without ambiguity. Hence, ''indexed orifice area'' should not be used without specifying whether it is the indexed IGA or the indexed EOA. Also, given that the term PPM stems from a haemodynamic concept, its use should be reserved for data relating to haemodynamic function (that is, indexed EOAs and gradients), whereas the results of studies based on the analysis of the IGA or labelled valve size would be more appropriately described in terms of patient-prosthesis size but without using the term mismatch, thus avoiding much confusion. The use of adequate terminology has important clinical implications, since, as mentioned, patient-prosthesis size has little relevance to adverse clinical outcomes, whereas PPM can be viewed as a major risk factor in this regard.[6–8 10 11 13–15 19 26–29]

IMPACT OF PPM ON CLINICAL OUTCOMES

Table 2 summarises the impact of PPM on clinical outcomes. As indicated above, the studies based on the indexed IGA are not really descriptive of PPM. They are given in the interest of completeness and to dispel any confusion but will not be described in any further detail.

Left ventricular mass and function and adverse cardiac events

In a study of 1103 patients with a porcine bioprosthetic valve, Del Rizzo *et al*[29] found a strong and independent relation between the indexed EOA and the extent of left ventricular (LV) mass regression after AVR. In a smaller series, Tasca *et al*[15] also found that the normalisation of LV mass is negatively and independently influenced by PPM. Whereas some authors have found that the persistence of PPM results in less regression of LV hypertrophy, others have reported that patients with PPM or small prostheses exhibited significant reductions in LV mass and, on this basis, concluded that PPM was not an important issue.[12 22 31 32] Interestingly, Tasca *et al*[27] found that patients with PPM nonetheless exhibit LV mass regression after operation but that the extent of such regression varies considerably from one patient to another and can be largely related to the extent of valve EOA increase after operation. These findings remind us of the following important pathophysiological concepts.[27 33] Firstly, even in the presence of PPM, surgery normally results in improved haemodynamic function, the extent of which can be quite important. Secondly, a more optimal result can be expected if PPM is completely avoided. Thirdly, in analysing the results of AVR, it is important to remember that the relation between gradients and the indexed EOA is curvilinear and that the implications for a given patient will be directly related to his or her original and final positions on the indexed EOA-gradient curve (fig 1).

Besides LV mass, other factors including the coronary flow reserve (CFR) may influence the normalisation of LV function and the regression of symptoms after AVR. It is well known that patients with aortic stenosis may experience angina pectoris and present ECG signs suggestive of myocardial ischaemia, even if they have angiographically normal epicardial coronary arteries.[34 35] Angina is associated with a major increase in the risk of sudden death in these patients and is relieved immediately after AVR, whereas LV hypertrophy may regress over the next several months to years.[34 36 37] The reduction in CFR is the key factor responsible for myocardial ischaemia in patients with aortic stenosis and this may contribute to the development of LV dysfunction, symptoms, and adverse outcomes.[38] In a study of 20 patients with aortic stenosis with angiographically normal coronary arteries, Rajappan *et al*[38] found that the severity of impairment of CFR measured by positron emission tomography was related to the severity of valve stenosis (valve EOA, gradient, LV systolic pressure) and diastolic perfusion time rather than to LV mass. In a subsequent study, the same team reported that changes in CFR after AVR were not directly related to regression of LV mass but rather were dependent on the change in valve EOA achieved with AVR.[39] These findings suggest that PPM may compromise the postoperative normalisation of CFR and thereby have a detrimental impact on LV function after AVR.

Consistently, we have found that PPM is associated with a significant reduction in cardiac index during the postoperative course, the greatest deterioration being seen in

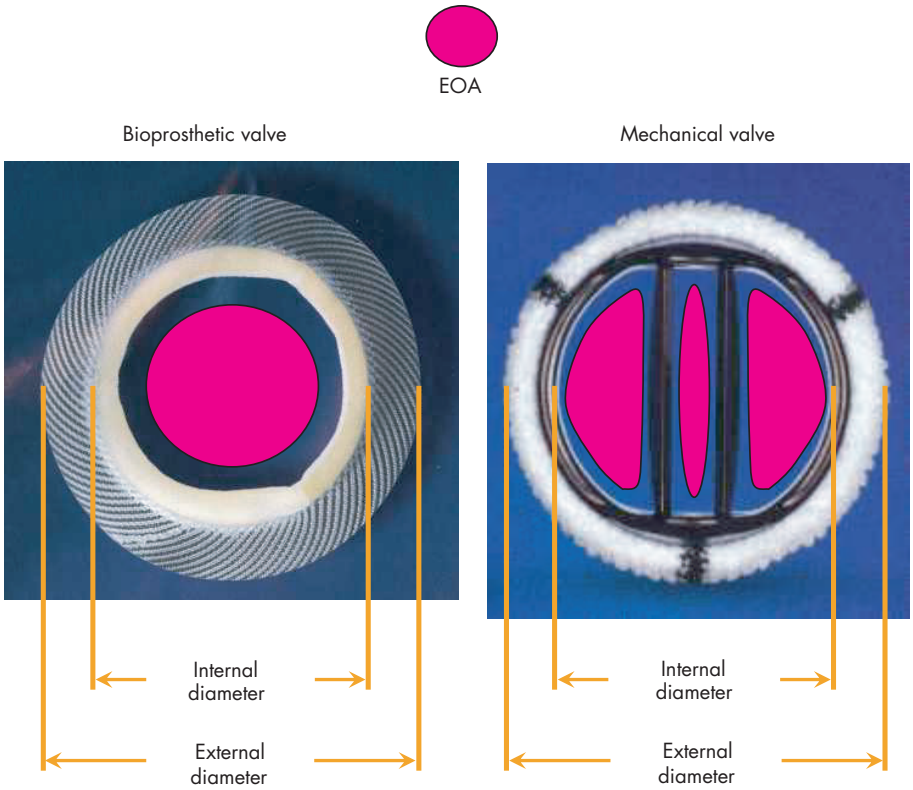

Figure 2 View of a bioprosthesis and a bileaflet mechanical valve with the leaflets in a fully open position. The area highlighted in pink is the effective orifice area (EOA). Reproduced and modified from Pibarot and Dumesnil[5] with permission of Remedica Publishing.

patients with the most severe PPM.[8] Moreover, the incidence of congestive heart failure was significantly higher in patients with PPM. These findings are consistent with those of Milano *et al*,[11] who reported that patients with severe PPM had many more late cardiac events (most of them being congestive heart failure) after AVR (fig 4) and that PPM was an independent predictor of these late cardiac events. More recently, Ruel *et al*[26] analysed the factors associated with persistent or recurrent heart failure in 1563 patients having undergone AVR and found that PPM defined as an indexed EOA $\leqslant$ 0.80 cm^2/m^2 was an independent risk factor associated with a 60% increase in the risk of heart failure. Other recent studies also further confirmed that PPM is a strong risk factor for the occurrence of cardiac events after AVR (table 2).[14 19]

Early mortality

The impact of PPM on early mortality may be particularly important given that the left ventricle is more vulnerable during the early postoperative period and that it may thus be more sensitive to the increased haemodynamic burden imposed by PPM. In this regard, several studies reported that early mortality is significantly increased in patients with PPM (table 2).[10 12 13 30] In a series of 2154 patients, Rao *et al*[10] found that 30 day mortality was significantly higher (7.9% *v* 4.6%, p = 0.03) in patients with PPM. However, PPM was found to be an independent predictor only of late mortality, not of early mortality. We recently showed that PPM had a profound impact on early mortality in a series of 1265 consecutive patients undergoing AVR.[13] In-hospital mortality was 4.6% in this series and moderate PPM had a risk ratio of 2.1 (95% confidence interval (CI) 1.2 to 3.7), whereas severe PPM had a risk ratio of 11.4 (95% CI 4.4 to 29.5). Moreover, the adverse impact of PPM was much more pronounced in patients having an impaired LV ejection fraction ($\leqslant$ 40%). As fig 5 shows, the mortality risk was relatively low (mortality 2–5%) in the case of patients with preserved LV function who had non-significant or moderate PPM. On the other hand, the mortality risk was dramatically increased (mortality 67%) in the patients having poor LV function and, concomitantly, severe PPM. In addition, even moderate PPM had a highly detrimental impact in the context of depressed LV function (mortality 16%). These findings are consistent with the concept that a failing ventricle is much more sensitive than a normal ventricle to an increase in afterload. In light of these results, avoidance of potential PPM should become a particularly mandatory consideration for patients with LV dysfunction. From the standpoint of pathophysiology, it would also make sense to consider that these high risk patients have a decreased ventricular reserve and are thus more vulnerable to the different degrees of PPM, particularly in the critical perioperative period.

Late mortality

Several previous studies of relatively small numbers of patients found no negative impact of PPM on mid-term (up to eight years) mortality.[8 11 12] However, in a study of 2516 patients who underwent AVR with a stented bioprosthetic valve, Rao *et al*[10] reported that freedom from valve related death at 12 years was significantly (p = 0.004) lower in patients with an indexed EOA $\leqslant$ 0.75 cm^2/m^2 (75.5%) compared with those with a larger indexed EOA (84.2%). Moreover, the patient's age and indexed valve EOA were found to be independent predictors of valve related death. In a recent study by Tasca *et al*[19] of 315 patients with pure aortic stenosis, five year overall survival and cardiac event-free survival were significantly (p $\leqslant$ 0.01) lower in patients with PPM (mean (SD) 82 (3)%, and 75 (4)%, respectively) than in those without PPM (93 (3)% and 87 (4%)). In another recent study by Mohty-Echahidi *et al*[14] of 388 patients with a 19 or 21 mm Standard St Jude valve, eight year survival (41 (8)%) of patients with severe PPM was significantly less than that of patients with moderate PPM (65 (5)%, p = 0.026) or no PPM (74 (5%), p = 0.002). On multivariate

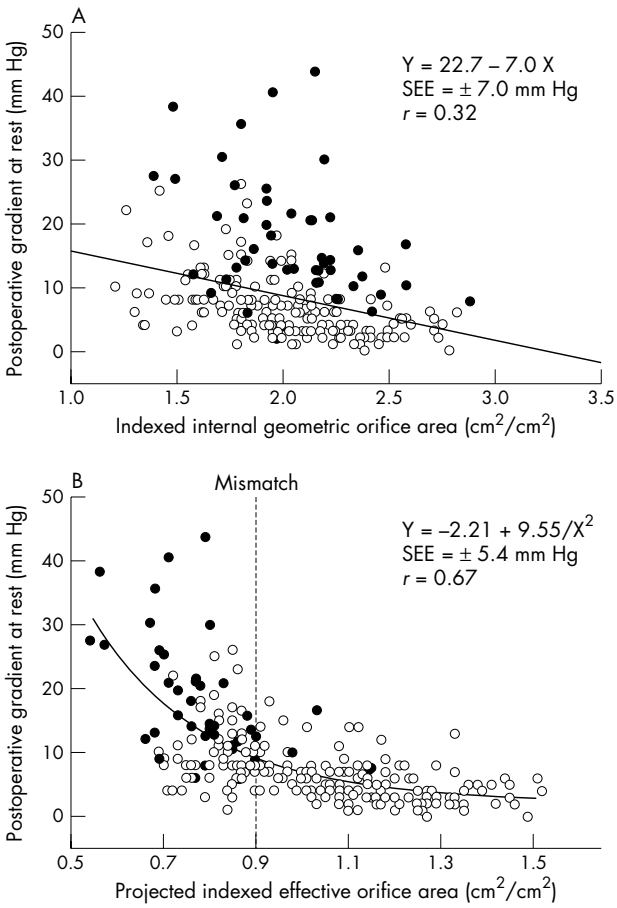

Figure 3 Correlation between postoperative mean gradient and parameters calculated at the time of operation: (A) indexed internal geometric area and (B) projected indexed EOA. Open circles represent stentless bioprostheses; closed circles represent stented bioprostheses. Reproduced from Pibarot *et al*[20] with permission of the Society of Thoracic Surgeons.

analysis after adjustment for other predictors of outcome, severe PPM was associated with higher mortality than was no PPM.

When analysed collectively, these previous studies suggest that the greatest impact of PPM on survival is in the early postoperative period when the left ventricle is most vulnerable. They also suggest that PPM has a significant impact on late mortality. A possible explanation for this late effect may be that patients with PPM undergoing long term valve degeneration or development of pannus have less EOA ''reserve'' and will therefore develop severe valve stenosis more rapidly than will the patients without PPM undergoing the same processes. As well, given that their LV is subjected to an increased haemodynamic burden at the outset, patients with PPM may not be able to tolerate as well or as long the additional workload associated with other concomitant diseases such as hypertension, coronary artery disease, and mitral insufficiency.

Bleeding complications

Vincentelli *et al*[28] reported that abnormalities of von Willebrand factor and associated bleeding complications are common in patients with severe aortic stenosis. They also showed that von Willebrand abnormalities are directly related to the TPG and the stenosis induced shear stress. Interestingly, these abnormalities were generally improved by AVR in patients with no PPM but, on the other hand, they persisted in those with PPM. Further studies are now needed to determine whether the persistence of high shear stress caused by PPM is associated with an increased occurrence of bleeding complications after AVR.

PREVENTION OF MISMATCH

As opposed to most other risk factors associated with adverse clinical outcomes, PPM is modifiable and can be largely avoided by using a simple strategy at the time of operation.[6 20] Our original description of this strategy was as follows. Step 1: calculate the patient's BSA from his or her weight and height. Step 2: multiply BSA by 0.85 cm^2/m^2, the result being the minimum EOA that the prosthesis to be implanted should have to avoid PPM. For example, if the patient's BSA is 1.80 m^2, then $1.80 \times 0.85 = 1.53$ cm^2 is the minimum EOA to avoid PPM. Step 3: choose the prosthesis in light of the result obtained in step 2 and the reference values for the different types and sizes of prosthesis (table 3[6 40–42]). Hence, the EOA of the prosthesis to be implanted in the example chosen would have to be > 1.53 cm^2 to completely avoid PPM and if, for example, the surgeon had intended to implant a Carpentier-Edwards Perimount prosthesis, it would have had to be a size $\geqslant 23$. Fortunately, most prosthesis manufacturers have now made this exercise easier by providing charts that give the projected indexed EOAs for the patient's BSA and prosthesis sizes (fig 6). With regard to reference values for EOA and indexed EOA, three caveats are worth reiterating. Firstly, the values should be derived from in vivo rather than in vitro data, since in vitro data are usually too optimistic, particularly in the case of stentless valves.[43] Secondly, values derived from geometric measurements (for example, internal diameters or geometric areas) are inadequate, since they do not predict postoperative TPGs (figs 2 and 3).[20] Thirdly, when using these charts (or table 3), it is important to remember that there are often important discrepancies between the sizes of the different types of prostheses and the size for a given patient's annulus; the labelled size that fits may vary from one type of prosthesis to another.

If PPM is projected with the type of prosthesis that was originally intended to be implanted, the following options can be considered:

- Option 1: implant another type of prosthesis with a larger EOA such as a stentless bioprosthesis, a new generation mechanical prosthesis, or an aortic homograft (see table 3).
- Option 2: enlarge the aortic root to accommodate a larger prosthesis of the same type.
- Option 3: accept PPM in light of other clinical conditions.

The alternative options to avoid PPM (options 1 and 2) should of course be considered in light of the patient's clinical condition and overall risk to benefit ratio. If, for example, the projected indexed EOA is 0.75–0.80 cm^2/m^2 in a patient who is sedentary and has good LV function and it is evaluated that option 1 or 2 would significantly increase the operative risk, accepting this level of PPM may be the best option for this particular patient. If, on the other hand, severe PPM is projected or if the patient has poor LV function, the risks associated with PPM would be much higher and would probably outweigh any additional risks considered to be associated with options 1 and 2. Whatever the option chosen, we advocate performing this simple exercise in all cases, since it allows an enlightened clinical decision about the overall risks confronting the patient.

Castro *et al*[44] used this prospective strategy for 657 consecutive patients whereby the aortic root was enlarged (option 2) whenever the indexed EOA was projected to be < 0.85 cm^2/m^2. As a result, the overall incidence of PPM in this series was only 2.5% instead of the 17% that would have

Table 2 Summary of the clinical impact of prosthesis-patient mismatch (PPM)

Outcome variable	Parameter*	Clinical impact of PPM	Reference
LV mass and function			
LV mass regression	EOA-I	Decreased 4.5% with PPM *v* 23% without PPM (p=0.0001)	29
	EOA-I	Decreased at 2 years from baseline Δ−47 g with PPM *v* Δ−77 g without PPM (p=0.002)	15
	IGA-I	No influence on LV mass regression	22
LV systolic function (cardiac index)	EOA-I	Decreased at 5 years from baseline Δ−0.54 (l/min/m^2) with PPM *v* Δ−0.17 (l/min/m^2) without PPM (p=0.04)	8
Improvement of NYHA classification	EOA-I	Functional class improved +1.5 with PPM *v* +1.9 without PPM (p=0.009)	8
	IGA-I	No influence on functional recovery	21
Freedom from CHF	EOA-I	60% increase in the risk of CHF	26
	EOA-I	71% with severe PPM *v* 86% with moderate PPM and 87% without PPM 8 years postoperatively (p=0.02)	14
Freedom from late cardiac events	EOA-I	56% with severe PPM *v* 80% with moderate PPM and 94% without PPM 15 years postoperatively (p=0.03)	11
	EOA-I	75% with PPM *v* 87% without PPM 5 years postoperatively (p=0.005)	19
Early mortality	EOA-I	7.9% with PPM *v* 4.6% without PPM (p=0.03)	10
	EOA-I	26% with severe PPM *v* 6% with moderate PPM and 3% without PPM (p<0.001)	13
	IGA-I	No influence on early mortality	23
	IGA-I	No influence on early mortality	24
	IGA-I	1–2% increase in early mortality (p=0.002)	25
Freedom from late mortality	EOA-I	75% with PPM *v* 84% without PPM 12 years postoperatively (p=0.0004)	10
	EOA-I	41% with severe PPM *v* 65% with moderate PPM and 74% without PPM 8 years postoperatively (p=0.002)	14
	EOA-I	82% with PPM *v* 93% without PPM 5 years postoperatively (p=0.01)	19
	IGA-I	No influence on late mortality	24
	IGA-I	No influence on late mortality	25
High risk patients			
Early mortality (LVEF <35%)	NA	47% with small prosthesis *v* 15% with larger prosthesis (p=0.03)	30
Early mortality (LVEF <40%)	EOA-I	67% with severe PPM *v* 16% with moderate PPM and 7% without PPM (p<0.001)	13

*Parameter used to define PPM.
CHF, congestive heart failure; EOA-I, indexed effective orifice area; IGA-I, indexed internal geometric orifice area; LV, left ventricular; LVEF, left ventricular ejection fraction; NA, not available; NYHA, New York Heart Association.

been expected had the prospective strategy not been used. Moreover, operative mortality was not increased as a result of the root enlargement that was performed by a novel technique consisting of the insertion of a patch made of Hemasheild (Boston Scientific, Natick, Massachusetts, USA). These results show that the prospective strategy to avoid PPM is feasible and can be applied with success. More recent studies that have used an independent sizer to measure the actual diameter of the annulus also show that most stented bioprosthesis will result in an unacceptably high incidence of PPM in patients with an annulus < 22 mm (45% of the series) and are further evidence that the prospective strategy should be mandatory in all cases.[16 17]

IMPLICATIONS FOR THE INTERPRETATION OF HIGH POSTOPERATIVE GRADIENTS

A simple algorithm may be used to assess abnormally high gradients in the postoperative period (fig 7[45]). The first step is to compare the EOA measured by Doppler echocardiography with the normal reference value of EOA (see table 3) for the type and size of prosthesis that has been implanted in the patient. If the measured EOA is much lower than the normal

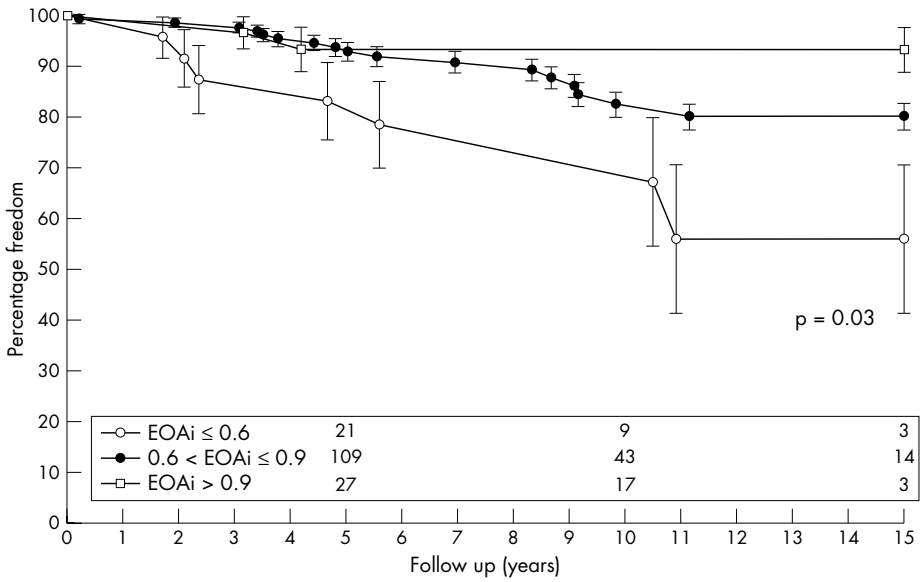

Figure 4 Freedom from late cardiac events in patients with non-significant (indexed EOA (EOAi) >0.9 cm^2/m^2; squares), moderate (EOAi >0.6 cm^2/m^2 and ⩽0.9 cm^2/m^2; solid circles), or severe (EOAi ⩽0.6 cm^2/m^2; open circles) mismatch. Reproduced from Milano *et al*[11] with permission of the Society of Thoracic Surgeons.

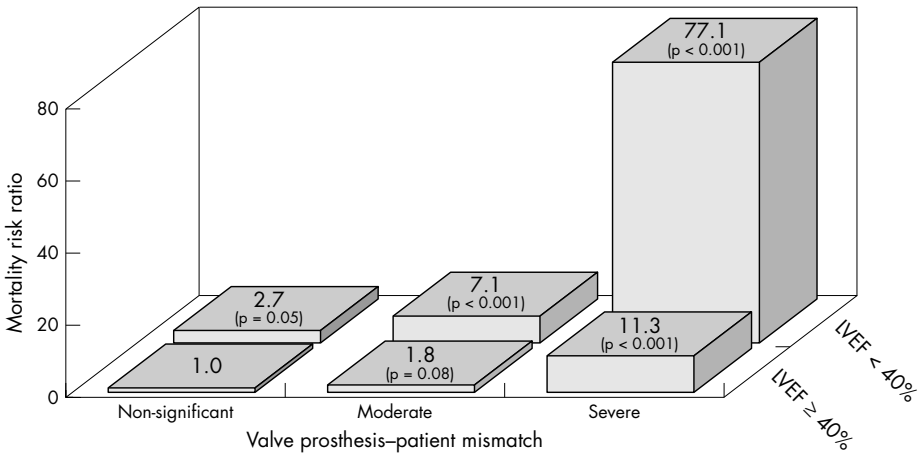

Figure 5 Relative risk ratio for short term mortality according to valve prosthesis-patient mismatch and preoperative left ventricular ejection fraction (LVEF). Numbers above the bars indicate the relative risk ratio for mortality compared with the group with non-significant mismatch and normal LVEF. Reproduced from Blais *et al*[13] with permission from the American Heart Association.

reference value and, moreover, has decreased over time during follow up, the diagnosis of intrinsic dysfunction should be raised. Causes of such dysfunction include the presence of leaflet calcific degeneration, thrombosis, pannus, or endocarditis. Occasionally, an abnormally high velocity jet corresponding to a localised gradient may be recorded by continuous wave Doppler interrogation through the smaller central slit-like orifice of a bileaflet mechanical prosthesis. If, on the other hand, the EOA is comparable with its normal reference value, the next step is to calculate the indexed EOA. An indexed EOA < 0.85 cm^2/m^2 indicates the presence of PPM. And, lastly, it is also important to keep in mind that both phenomena—that is, PPM and intrinsic dysfunction—may coexist.

PPM IN THE MITRAL POSITION

Rahimtoola and Murphy[46] were the first to describe the case of a patient with PPM in the mitral position. In subsequent studies, Dumesnil *et al*[3 4] then showed that the indexed EOA of mitral prostheses should ideally not be less than 1.2–1.3 cm^2/m^2 to avoid abnormally high residual TPGs. In a recent series, we found that PPM defined as an indexed EOA $\leqslant 1.2$ cm^2/m^2 is common (71%) after mitral valve replacement and that it is associated with persisting pulmonary hypertension.[47] Indeed, the prevalence of pulmonary hypertension decreased from 69% before operation to 19% after operation in patients with no PPM, whereas it remained unchanged in those with PPM (69% before *v* 68% after replacement). Consistently, Masuda *et al*[48] found that the maximum transprosthetic flow velocity is a strong determinant of the pulmonary capillary wedge pressure in children with mitral prostheses. In this series, the indexed mitral valve area did not correlate with pulmonary pressure; however, it should be pointed out that the valve area was derived from the IGA and not from the EOA. These results show that mitral PPM is not a rare occurrence and definitely warrants further documentation.

The prevention of PPM in the mitral position is a particularly demanding challenge because, as opposed to the aortic position, for the mitral position there is no alternative technique allowing implantation of a larger prosthesis. The preventive strategy should therefore be focused on the implantation of the prosthesis having the largest EOA for a given size. This observation also underlines the need for the development of better performing mitral prostheses and provides further motivation for repairing rather than replacing the valve whenever possible.

CONCLUSION

PPM is a common and modifiable risk factor leading to worse haemodynamic function, less regression of LV hypertrophy, more cardiac events, and lower survival. The projected indexed EOA should be systematically calculated at the time of the operation to estimate the risk of PPM and, if PPM is anticipated, alternative options should be considered in light of the patient's overall clinical condition and risk to benefit ratio.

Table 3 Normal reference values of EOAs* for prosthetic valves

Valve type	Prosthetic valve size (mm)						Reference
	19	21	23	25	27	29	
Stented bioprosthetic valves							
Medtronic Mosaic	1.20	1.22	1.38	1.65	1.80	2.00	6
Hancock II	NA	1.18	1.33	1.46	1.55	1.60	6
Carpentier-Edwards Perimount	1.10	1.30	1.50	1.80	1.80	NA	6
Stentless bioprosthetic valves							
Medtronic Freestyle	1.15	1.35	1.48	2.00	2.32	NA	6
St Jude Medical Toronto SPV	–	1.30	1.50	1.70	2.00	2.50	6
Prima Edwards	0.80	1.10	1.50	1.80	2.30	2.80	6
Mechanical valves							
Medtronic-Hall	1.19	1.34	NA	NA	NA	NA	6
St Jude Medical Standard	1.04	1.38	1.52	2.08	2.65	3.23	6
St Jude Medical Regent	1.60	2.00	2.20	2.50	3.60	4.40	40
MCRI On-X	1.50	1.70	2.00	2.40	3.20	3.20	41
Carbomedics	1.00	1.54	1.63	1.98	2.41	2.63	6
Sorin Bicarbon	NA	1.66	1.96	NA	NA	NA	42

*Expressed as mean values available in the literature.

	EOAi by Prosthesis size (mm)					
Prosthesis size (mm)	**19**	**21**	**23**	**25**	**27**	**29**
Average EOA (cm^2)	1.1	1.3	1.5	1.8	2.3	2.7
BSA (m^2)						
0.6	1.83	2.17	2.50	3.00	3.83	4.50
0.7	1.57	1.86	2.14	2.57	3.29	3.86
0.8	1.38	1.63	1.88	2.25	2.88	3.38
0.9	1.22	1.44	1.67	2.00	2.56	3.00
1	1.10	1.30	1.50	1.80	2.30	2.70
1.1	1.00	1.18	1.36	1.64	2.09	2.45
1.2	0.92	1.08	1.25	1.50	1.92	2.25
1.3	0.85	1.00	1.15	1.38	1.77	2.08
1.4	0.79	0.93	1.07	1.29	1.64	1.93
1.5	0.73	0.87	1.00	1.20	1.53	1.80
1.6	0.49	0.88	0.88	0.88	0.88	1.69
1.7	0.65	0.76	0.88	1.06	1.35	1.59
1.8	0.61	0.72	0.83	1.00	1.28	1.50
1.9	0.58	0.68	0.79	0.95	1.21	1.42
2	0.55	0.65	0.75	0.90	1.15	1.35
2.1	0.52	0.62	0.71	0.86	1.10	1.29
2.2	0.50	0.59	0.68	0.82	1.05	1.23
2.3	0.48	0.57	0.65	0.78	1.00	1.17
2.4	0.46	0.54	0.63	0.75	0.96	1.13
2.5	0.44	0.52	0.60	0.72	0.92	1.08

Figure 6 Example of a chart provided by the manufacturers for the risk assessment of prosthesis-patient mismatch. The chart gives the projected indexed effective orifice area (EOAi) for each level of patient's body surface area (BSA; left side) and size (top of chart) of a given model of prosthesis (hypothetical model in this example). Green cells indicate that the projected EOAi is > 0.85 cm^2/m^2, yellow cells indicate borderline values, and red cells indicate a risk of mismatch.

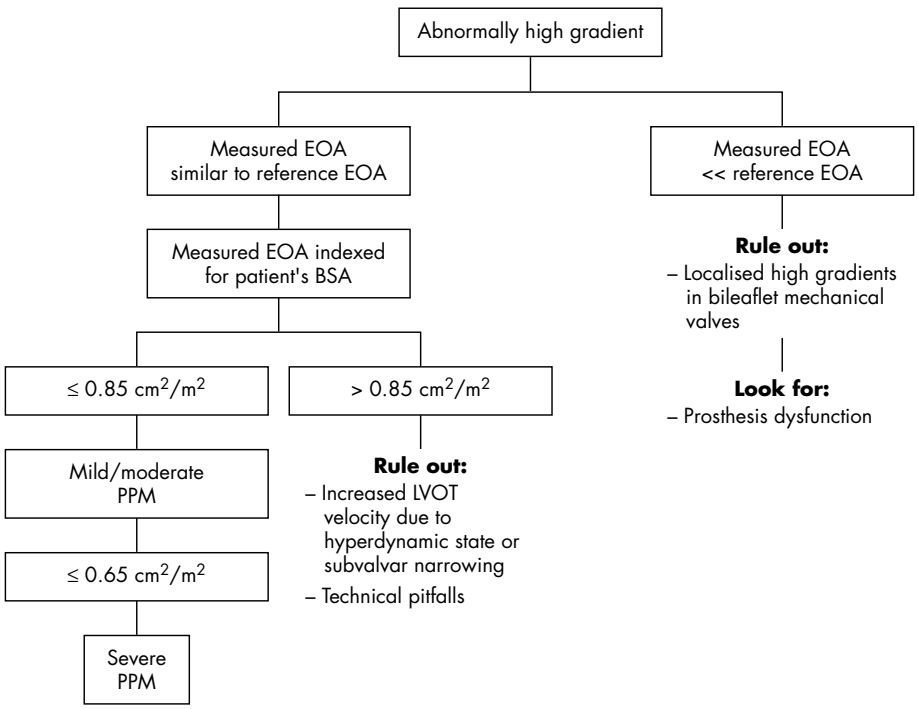

Figure 7 Algorithm used for evaluating abnormally high transvalvar pressure gradients after aortic valve replacement. LVOT, left ventricular outflow tract; PPM, prosthesis-patient mismatch. Reproduced and modified from Dumesnil and Pibarot[45] with permission from Marcel Dekker Inc.

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