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TODAY

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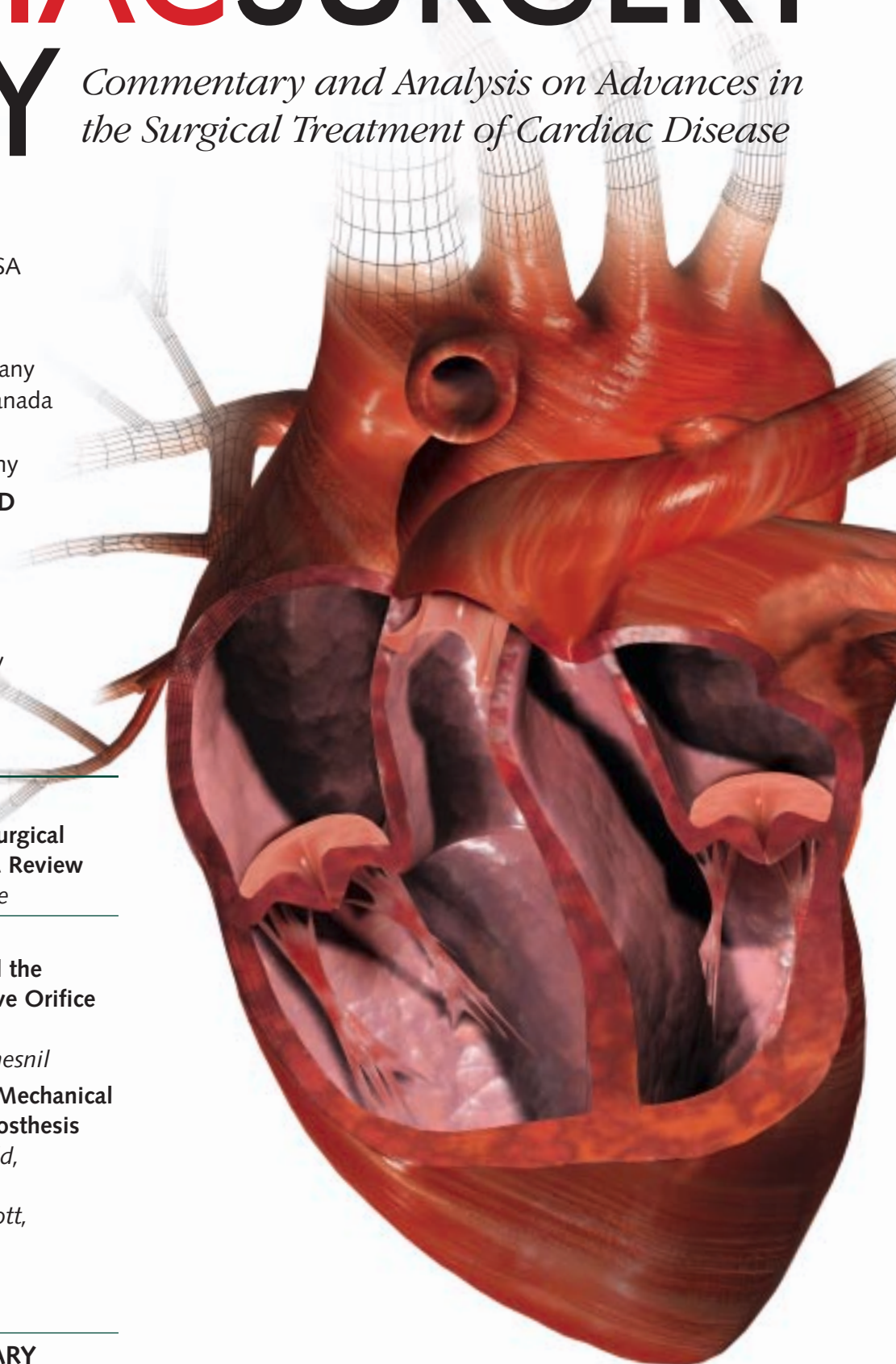
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Patient–Prosthesis Mismatch and the Predictive Use of Indexed Effective Orifice Area: Is it Relevant?

Philippe Pibarot and Jean G Dumesnil

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Patient–prosthesis mismatch (PPM) is a significant issue in valve replacement today. PPM is present when the effective orifice area (EOA) of the inserted prosthetic valve is too small relative to body surface area. PPM is defined as an EOA indexed for body surface area $\leq 0.8\text{--}0.9\text{ cm}^2/\text{m}^2$, and is a frequent problem in patients undergoing aortic valve replacement (20–70% prevalence). The main hemodynamic consequence of PPM is to generate high transvalvular gradients through normally functioning prosthetic valves. The purpose of this paper is to present an update on the present knowledge with regard to the impact of PPM on hemodynamic status, morbidity, and mortality. In addition, a simple algorithm for the prevention of this phenomenon is proposed, since it can largely be avoided if certain simple factors are taken into consideration at the time of operation. *Cardiac Surgery Today* 2003;1(2):43–51.

What is patient–prosthesis mismatch?

It is important to consider the transvalvular pressure gradient (TPG) following aortic valve replacement (AVR), since an increased gradient will result in increased left ventricular (LV) work, and thus may jeopardize the regression of LV hypertrophy and the normalization of LV function. In this context, it must be remembered that the TPG is directly related to the square of transvalvular flow (Q) and inversely related to the square of the valve effective orifice area (EOA), as illustrated by this equation (where k is a constant):

$$\text{TPG} = \frac{Q^2}{k \times \text{EOA}^2}$$

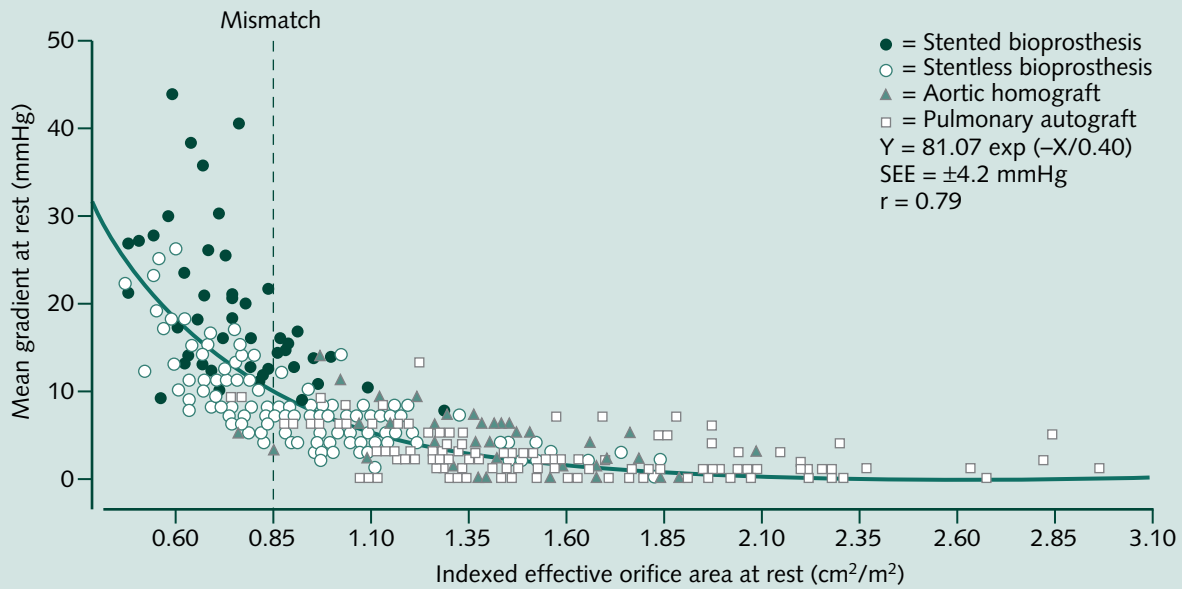
In turn, transvalvular flow is related to cardiac output, which, at rest, is largely determined by body surface area (BSA). Patient–prosthesis mismatch (PPM) occurs when the EOA of the prosthetic valve is too small relative to the patient's body size [1–3]. The immediate consequence of PPM is the persistence of abnormally high TPGs, which is often the reason for the original operation. If one wanted to caricature PPM, the epitome of this phenomenon would be

to insert a mouse's valve into the aorta of an elephant. This would evidently result in an extremely high gradient, even though the valve might be absolutely normal. Indeed, if the normal cardiac output requirement in a mouse is 50 mL/min and the normal valve EOA is 0.3 cm², the resulting gradient in accordance with the above equation would be <5 mmHg. In an elephant, the cardiac output requirement could well be 50 000 mL/min and, to obtain the same <5 mmHg gradient, the normal valve EOA would have to be 50 cm². In both cases there is a proportional relationship between valve size and cardiac output requirements, which means that the resulting gradient is the same, i.e. <5 mmHg. However, if the mouse's valve, albeit normally functioning, was implanted in the elephant, the resulting gradient would theoretically be >100 000 mmHg. Similarly, in humans there is great inter-person variability in BSA, and hence cardiac output requirement. Consequently, the implantation of a prosthesis with an EOA of 1.3 cm² in a patient with a BSA of 1.5 m² would result in a mean TPG of 13 mmHg, whereas the same prosthesis implanted in a patient with a BSA of 2.5 m² would result in a TPG of 35 mmHg (Table 1).

Hence, the main hemodynamic consequence of PPM is to generate high TPGs through normally functioning valves. Figure 1 shows the strong correlation that is consistently found between the postoperative TPG and the EOA indexed for BSA. This figure also clearly shows that when the indexed EOA becomes $\leq 0.8\text{--}0.9\text{ cm}^2/\text{m}^2$, the TPG (and thus LV work)

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Figure 1. Correlation between mean transvalvular gradient and indexed effective orifice area in patients with a stented bioprosthesis (n=51), a stentless bioprosthesis (n=194), an aortic homograft (n=55), or a pulmonary autograft (n=96). Several points are overlapped.



r: correlation coefficient; SEE: standard error of estimate.
 Reproduced with permission from [6].

Table 1. Theoretical comparison of the mean TPG in four hypothetical patients with different BSAs receiving the same prosthetic valve. For the purpose of this simulation, the mean TPG was calculated assuming a cardiac index of 3 L/min/m², a heart rate of 65 beats/min, and a systolic ejection time of 300 ms.

	Patient				
	1	2	3	4	5
BSA (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 effective orifice area (cm ²)	1.3	1.3	1.3	1.3	1.3
Mean TPG (mmHg)	13	17	22	28	35

BSA: body surface area; TPG: transvalvular pressure gradient.

increases exponentially. Based on this relationship, an indexed EOA of ≤ 0.85 cm²/m² is generally considered to be the threshold for PPM [2–6]. However, it should be noted that this definition corresponds to moderate PPM. Severe PPM is defined as an indexed EOA of ≤ 0.65 cm²/m², whereas PPM is clinically insignificant when the indexed EOA is >0.85 cm²/m² as it is associated with relatively low residual TPGs (<10 mmHg) (Fig. 1). Previous studies have reported that moderate PPM may be prevalent in patients undergoing AVR (20–70%), whereas the prevalence of severe PPM ranges from 2–11%, depending on the series [4–11].

How to predict mismatch at the time of surgery

Knowledge of the parameters that can accurately predict PPM at the time of surgery may be useful for assessing the clinical impact of PPM, as well as for developing preventative strategies. After AVR, the EOA of the prosthetic valve can be measured by Doppler echocardiography and then indexed for BSA to assess the presence and severity of PPM. The postoperative indexed EOA can also be predicted at the time of operation by calculating the “projected” indexed EOA. This parameter is easily calculated from the normal reference values of EOA for the type and size of prosthesis being implanted and the patient’s BSA (Table 2). Some investigators have also attempted to predict PPM on the basis of the labeled valve size, or the internal geometric area (provided by the manufacturer) divided by patient’s BSA [12,13]. One advantage of these parameters is that they are easily available at the time of operation; however, their predictive value is questionable. Indeed, from a physiological standpoint, the TPG (and thus the LV workload) are related to the EOA of the valve, and not to its geometric or anatomical orifice area [14]. The valve EOA is a physiological parameter that represents the minimal cross-sectional area effectively occupied by the transvalvular flow jet (Fig. 2). The internal geometric area, as provided by the manufacturer, corresponds to the area calculated from

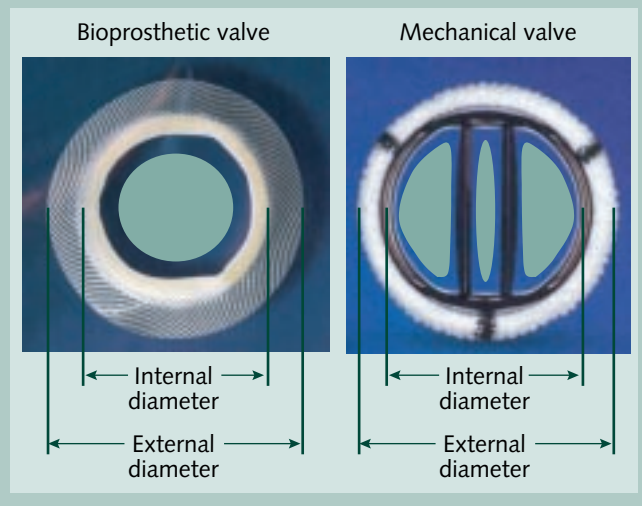
Table 2. Normal reference values of EOAs for the prosthetic valves. The EOAs are expressed as mean values available in the literature.

Prosthetic valve size (mm)	19	21	23	25	27	29
Stented bioprosthetic valves						
Medtronic Mosaic [25]	1.20	1.22	1.38	1.65	1.80	2.00
Hancock II [6]	NA	1.18	1.33	1.46	1.55	1.60
Carpentier-Edwards Perimount [6]	1.10	1.30	1.50	1.80	1.80	NA
Stentless bioprosthetic valves						
Medtronic Freestyle [6]	1.15	1.35	1.48	2.00	2.32	NA
St. Jude Medical Toronto SPV [6]	NA	1.30	1.50	1.70	2.00	2.50
Prima Edwards [26]	0.80	1.10	1.50	1.80	2.30	2.80
Mechanical valves						
Medtronic-Hall [27]	1.19	1.34	NA	NA	NA	NA
Medtronic Advantage [28]	NA	1.65	2.17	2.80	3.32	3.90
St. Jude Medical Standard [6]	1.04	1.38	1.52	2.08	2.65	3.23
St. Jude Medical Regent [29]	1.50	2.00	2.40	2.50	3.60	4.80
MCRI On-X [30]	1.50	1.70	2.00	2.40	3.20	3.20
Carbomedics [6]	1.00	1.54	1.63	1.98	2.41	2.63

EOA: effective orifice area; NA: not available.

Manufacturers: Medtronic Mosaic (Medtronic, Inc., Minneapolis, MN, USA); Hancock II (Medtronic, Inc.); Carpentier-Edwards Perimount (Edwards Lifesciences Corp., Irvine, CA, USA); Medtronic Freestyle (Medtronic, Inc.); St. Jude Medical Toronto SPV (St. Jude Medical, Inc., St Paul, MN, USA); Prima Edwards (Edwards Lifesciences Corp.); Medtronic-Hall (Medtronic, Inc.); Medtronic Advantage (Medtronic, Inc.); St. Jude Medical Standard (St. Jude Medical, Inc.); St. Jude Medical Regent (St. Jude Medical, Inc.); MCRI On-X (Medical Carbon Research Institute, Austin, TX, USA); Carbomedics (Carbomedics, Inc., Austin, TX, USA).

Figure 2. View of a bioprosthesis and a bileaflet mechanical valve with the leaflets in a fully open position. The area highlighted in green represents the effective orifice area.



the internal diameter of the stent, and this is larger than the area actually occupied by flow, since it also includes the area occupied by the valve leaflets and the support apparatus. Moreover, it should be remembered that the ratio of the EOA to the actual geometric orifice area, namely the discharge coefficient, may vary extensively (range 0.6–0.9),

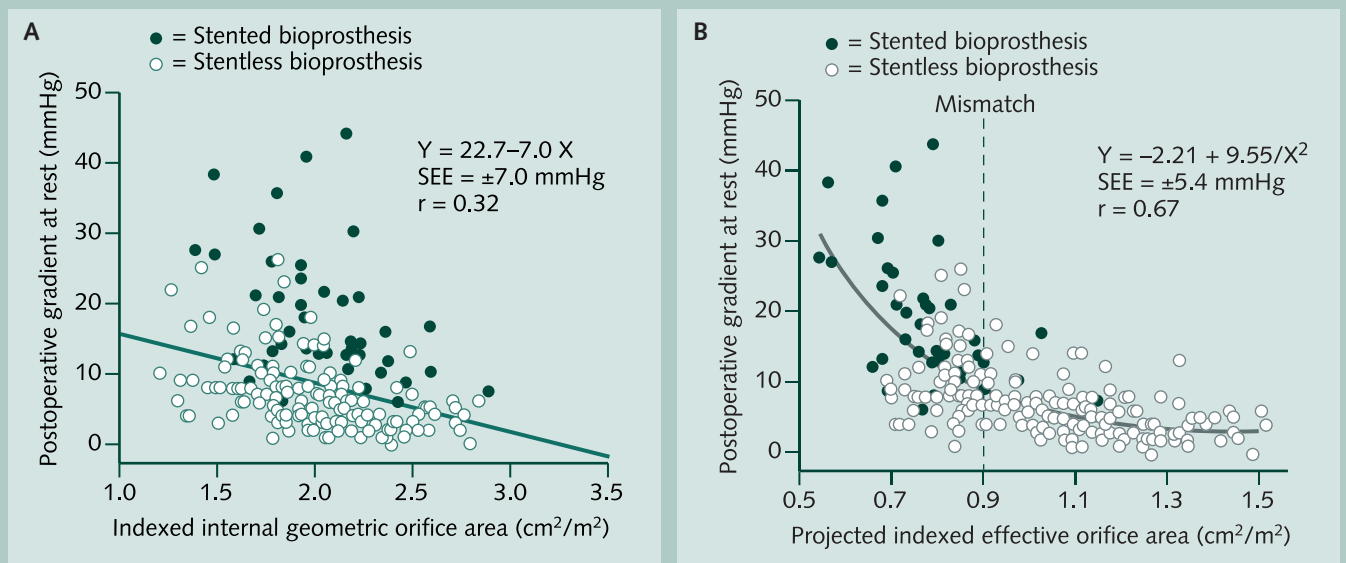
depending on the type and size of prosthesis. It is therefore not surprising that the internal geometric area overestimates the EOA in varying proportions, depending on the valves. Also, for the same reasons, the labeled valve size, which corresponds approximately to the external diameter of the stent, is not adequate to estimate the valve EOA.

Accordingly, in a previous study from our laboratory, we found a weak correlation between the postoperative TPG and the indexed internal geometric area ($r=0.32$) (Fig. 3A) [14]. Similarly, there was poor correlation between TPG and either valve size or valve size indexed for BSA ($r<0.32$). Hence, parameters based on valve size or internal geometric area cannot be used to identify patients who have a high TPG on the basis of PPM. In contrast, the projected indexed EOA calculated at the time of operation correlates well with the postoperative TPG ($r=0.67$), and the calculation of this parameter at the time of operation may be particularly useful for designing strategies for the prevention of PPM (Fig. 3B).

Two important messages can be drawn from these results:

- It is not the size of the prosthesis that matters, but rather its EOA and in whom it is implanted.
- To date, the only parameter demonstrated to be valid for defining PPM is the indexed EOA.

Figure 3. Correlation between the postoperative mean gradient and the parameters calculated at the time of operation. A: Indexed internal geometric area. B: Projected indexed effective orifice area.



r: correlation coefficient; SEE: standard error of estimate.
 Reproduced with permission from [14].

The clinical impact of mismatch

Impact on LV mass and function

A large amount of literature is available on the effect of AVR on LV hypertrophy regression. However, there are few studies that directly address this issue in relation to PPM. In a study including 1103 patients with a porcine bioprosthetic valve, Del Rizzo et al. found a strong and independent relationship between the indexed EOA and the extent of LV mass regression following AVR [15]. There was a mean decrease in LV mass of 23% in patients with an indexed EOA of >0.8 cm²/m² compared with only 4.5% in those with an indexed EOA of ≤0.8 cm²/m² (p=0.0001). In contrast to these results, Hanayama et al. found no significant relationship between PPM and regression of LV hypertrophy [10]. However, the majority of patients included in this retrospective study did not undergo a complete echocardiographic follow-up of LV mass (preoperative baseline and/or postoperative measurements not available). Furthermore, 50% of the patients with severe PPM included in this study still had significant LV hypertrophy >5 years after AVR. These findings suggest that PPM may reduce regression of LV hypertrophy following AVR.

To determine the impact of the residual gradients due to PPM on LV systolic function, we prospectively followed the cardiac index of 392 patients after AVR [5]. During the 7-year follow-up, cardiac index decreased significantly only in patients with PPM. Not surprisingly, the greatest deterioration was seen in the patients with the most severe PPM (indexed EOA ≤0.65 cm²/m²). Moreover, the incidence

of congestive heart failure (CHF) was significantly higher in patients with PPM. This observation is consistent with the observations of Milano and colleagues who reported that patients with severe PPM had a greater incidence of late cardiac events (mostly CHF) following AVR, and that PPM was an independent predictor of these late cardiac events (Fig. 4) [9]. These results suggest that PPM may have a detrimental impact on the normalization of LV mass and function following AVR.

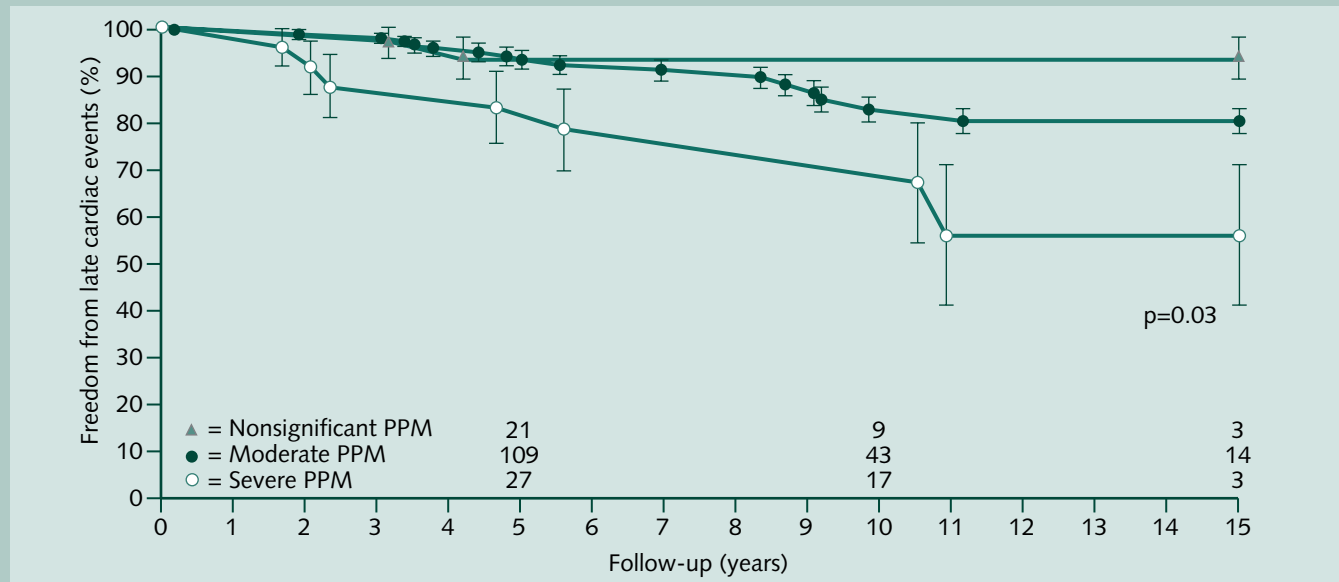
Impact on bleeding complications

Vincentelli et al. recently reported that abnormalities of von Willebrand factor (VWF) and bleeding complications are common in patients with severe aortic stenosis [16]. They also demonstrated that VWF abnormalities increased with the TPG and the stenosis-induced shear stress. Interestingly, these abnormalities were completely corrected on the first day of surgery, but tended to recur in patients with PPM (Fig. 5). Hence, further studies are needed to determine if the persistence of high shear stress due to PPM might be associated with an increased occurrence of bleeding complications after AVR.

Impact on early mortality

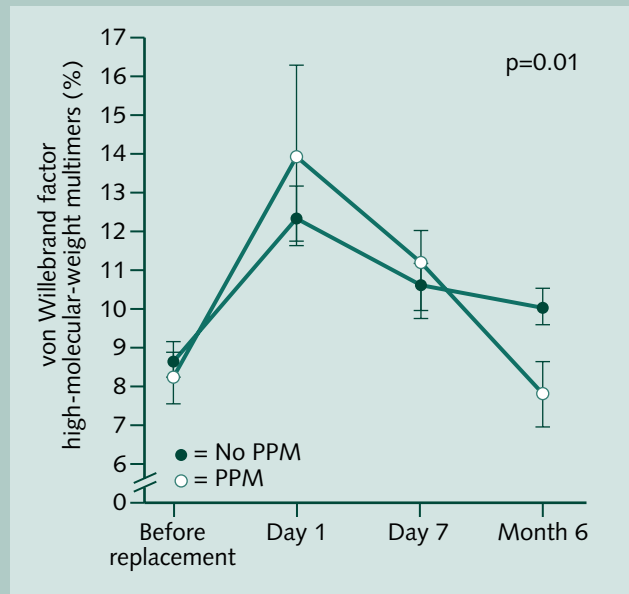
The impact of PPM on early mortality may be particularly important, given that the left ventricle is more vulnerable during the early postoperative period and may thus be more sensitive to the increased hemodynamic burden imposed by PPM. Several studies reported that early mortality is

Figure 4. Freedom from late cardiac events in patients with nonsignificant (indexed EOA >0.9 cm²/m²), moderate (indexed EOA 0.6–0.9 cm²/m²), or severe (indexed EOA ≤0.6 cm²/m²) PPM. The numbers above the X-axis represent the number of patients in each group.



EOA: effective orifice area; PPM: patient-prosthesis mismatch. Reproduced with permission from [9].

Figure 5. Evolution of highest-molecular-weight von Willebrand factor multimers after aortic valve replacement in patients with and without PPM.



PPM: patient-prosthesis mismatch. Reproduced with permission from [16].

significantly increased in patients with PPM [8,10,11]. In a cohort of 2154 patients who underwent AVR, Rao et al. found that 30-day mortality was significantly higher in patients with evidence of PPM (7.9% vs. 4.6%; p=0.03)

[8]. However, in multivariate analysis it was only found to be an independent predictor of late valve-related mortality, not of early mortality. We recently performed a study in 1266 patients to specifically examine the relationship between PPM and early mortality [11]. In this cohort, the prevalence of PPM was 38% (36% moderate PPM, 2% severe PPM). In-hospital mortality was 3% in patients with nonsignificant PPM (i.e. mild or none), 6% in patients with moderate PPM, and 26% in patients with severe PPM (p<0.001). The relative risk of mortality was increased 2.1-fold (95% confidence interval [CI] 1.2–3.7) in patients with moderate PPM, and 11.4-fold (95% CI 4.4–29.5) in those with severe PPM. Multivariate analysis demonstrated that PPM was a strong and independent predictor of mortality. The practical implications of these findings are important given that the prevalence of PPM has been reported to be high in patients undergoing AVR [4–11]. Furthermore, as opposed to other risk factors for early mortality, it can be largely avoided with the use of a proper preventive strategy at the time of operation [6,14,17].

Impact on late mortality

There persists some controversy in the literature as to the eventual consequences of PPM on late mortality. Several previous studies in a relatively small number of patients failed to demonstrate a negative impact of PPM on mid-term mortality (≤8 years) [5,9,10]. However, in a study of 2516 patients who underwent AVR with a stented

Figure 6. Freedom from valve-related mortality in patients with an indexed EOA of $\leq 0.75 \text{ cm}^2/\text{m}^2$ and in patients with an indexed EOA of $>0.75 \text{ cm}^2/\text{m}^2$. The numbers above the X-axis represent the number of patients in each group.



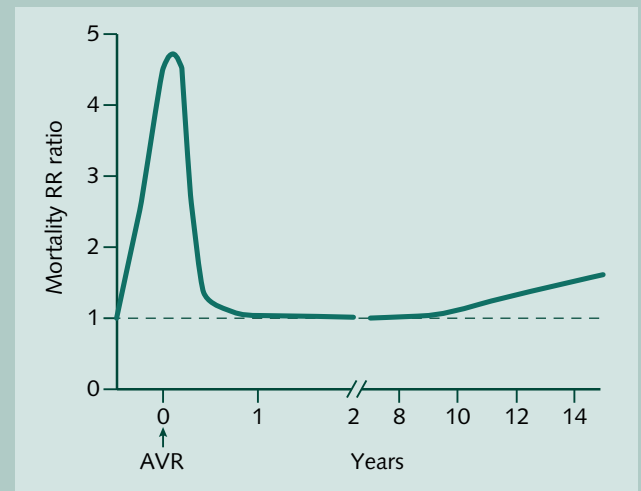
EOA: effective orifice area.
 Reproduced with permission from [8].

bioprosthetic valve, Rao and colleagues reported that freedom from valve-related mortality at 12 years was significantly lower in patients with an indexed EOA of $\leq 0.75 \text{ cm}^2/\text{m}^2$ compared with those with a larger indexed EOA (75.5% vs. 84.2%; $p=0.004$) (Fig. 6) [8]. Moreover, in this study, the patient's age and indexed valve EOA were independent predictors of valve-related mortality. In Figure 6, it should be noted that the survival curve of the PPM group starts to separate from that of the non-PPM group only after 7–8 years, which could explain why other studies with shorter follow-up periods failed to demonstrate any significant impact of PPM on late mortality [5,9,10].

Other studies that purportedly analyzed PPM in early and late mortality following AVR did not identify any major influence [12,13]. However, it should be emphasized that in these studies PPM was defined on the basis of the indexed internal geometric area calculated from the internal diameter of the prosthesis divided by the patient's BSA. As previously mentioned, this parameter cannot reliably be used to identify patients who have a high postoperative gradient on the basis of PPM, and hence these results are not valid [6,14].

When analyzed collectively, the previous studies suggest that the greatest impact of PPM on survival is in the early postoperative period when the left ventricle is most vulnerable (Fig. 7). They also suggest that there could be a natural selection process whereby many at-risk patients do

Figure 7. Hypothetical representation of the RR of mortality associated with patient–prosthesis mismatch as a function of time to surgery.



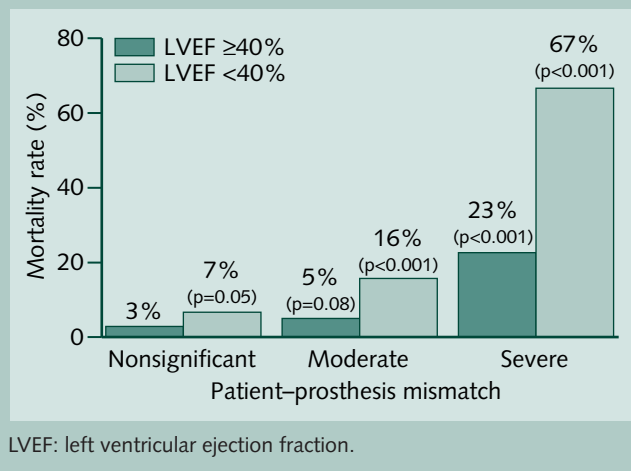
AVR: aortic valve replacement; RR: relative risk.

not survive beyond the early postoperative period, which in turn could explain the relatively better prognosis of PPM beyond that critical period. As suggested in the study of Rao et al., it is possible that PPM also has a significant impact on longer-term mortality (>10 years) [8]. It is well known that the EOA of bioprosthetic valves may progressively deteriorate during follow-up due to leaflet calcific degeneration. This deterioration generally becomes more frequent and more rapid after 8–10 years [18]. Patients with moderate or severe PPM already have significant LV outflow obstruction at the time of operation. Any further decrease in EOA during the postoperative period could lead to severe obstruction, and therefore precipitate the occurrence of negative outcomes and/or the need for re-operation. In contrast, patients without PPM have a substantial valve EOA “reserve”, which would allow them to better tolerate the progressive reduction in EOA that may occur as a consequence of leaflet calcification in the case of bioprosthetic valves, or pannus overgrowth in the case of mechanical valves. In summary, the available data suggest that PPM has an important impact on the early postoperative period, no or minimal impact in the mid-term period, and a mild-to-moderate impact in the late period (Fig. 7).

Impact of mismatch in high-risk patients

Connolly et al. reported a markedly higher mortality rate in patients with aortic stenosis and a poor LV ejection fraction (LVEF $\leq 35\%$) who received a small prosthesis (≤ 21 mm) compared with those who received a larger prosthesis (47% vs. 15%; $p=0.03$) [19]. Although this study was not a direct analysis of PPM based on the indexed EOA, it nonetheless

Figure 8. In-hospital mortality according to patient-prosthesis mismatch and preoperative LVEF. The p values above the bars correspond to the comparison with the group with nonsignificant mismatch and normal LVEF.



underlines the concept that a failing ventricle is much more sensitive to an increase in afterload than a normal ventricle [20]. Recently reported data from our institution have also shown that a combination of poor LV function (preoperative LVEF <40%) and PPM is associated with a dramatic increase in short-term mortality [11]. Figure 8 shows that in patients with a preserved LV function who have either nonsignificant or moderate PPM mortality is relatively low (2–5%). Conversely, the operative risk is definitely not acceptable in patients with poor LV function combined with a severe PPM (67% mortality). Even a moderate PPM may have a highly detrimental impact in the context of depressed LV function (16% mortality). These are very compelling data suggesting that avoidance of potential PPM should become a mandatory consideration in patients with LV dysfunction. From a pathophysiological standpoint, it would also make sense to consider that these patients have a decreased ventricular reserve, and are thus more vulnerable to the different degrees of PPM, particularly in the critical perioperative period. These data are also consistent with recent studies suggesting that, in patients with severe LV dysfunction, the implantation of stentless bioprostheses warrants a larger indexed EOA and leads to enhanced recovery of LVEF compared with that achieved with stented bioprostheses [21,22].

How to prevent mismatch

At present, there is a general agreement that the postoperative indexed EOA of the prosthesis being implanted should not be <0.85–0.90 cm²/m². To achieve this goal, it is suggested that the following algorithm is performed in the operating room (Fig. 9) [6]:

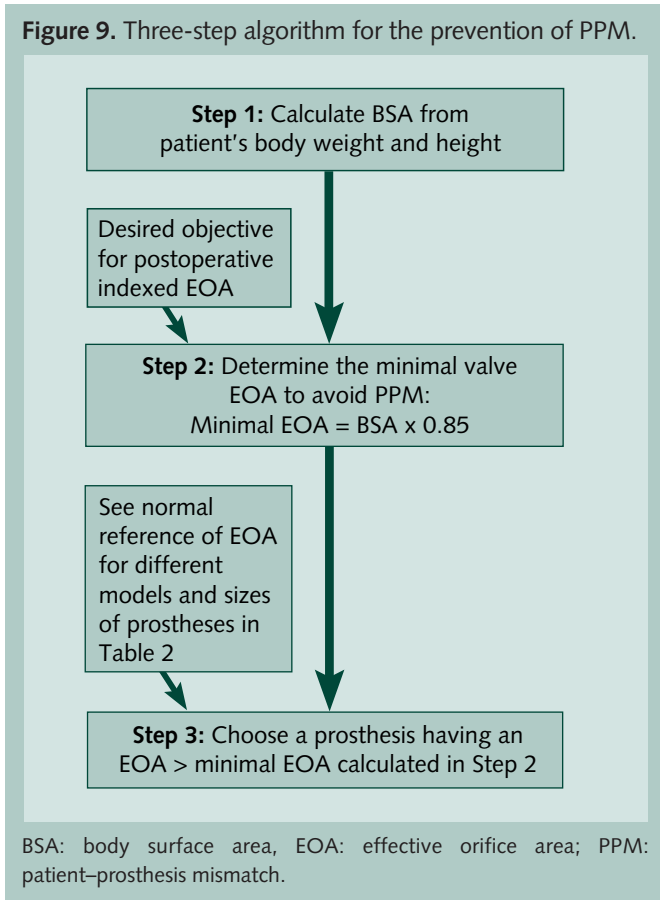
- Calculate the BSA from the patient's body height (in cm) and weight (in kg) using the DuBois and DuBois formula (below) [23], or the chart derived from that formula:

$$BSA = \text{Weight}^{0.425} \times \text{Height}^{0.725} \times 0.007184$$

- Determine the minimal EOA that the prosthesis being implanted must have in order to avoid PPM, by multiplying the desired postoperative indexed EOA (e.g. 0.85 cm²/m²) by the patient's BSA. Hence, if the patient's BSA is 1.6 m², the minimal EOA that the prosthesis being implanted should have in order to avoid PPM is 1.6 m² multiplied by 0.85 cm²/m², i.e. 1.36 cm².
- The prosthesis is then chosen using the published reference values for EOAs of different types and sizes of prostheses (Table 2). To follow the aforementioned example, if one had chosen to insert a Mosaic bioprosthesis (Medtronic, Inc., Minneapolis, MN, USA) the minimal size that should be utilized to yield the desired objective of 1.36 cm² should be a size 23. Hence, if the patient's annulus accepted only a size 21, as may be the case in patients with a small aortic annulus associated with calcific aortic stenosis, the available options to avoid PPM would be either to perform an aortic root enlargement to accommodate the size 23 prosthesis, or to use another type of prosthesis with a better hemodynamic profile (e.g. a stentless bioprosthesis or a mechanical valve).

Castro et al. prospectively used this strategy in 657 patients [17]. An aortic root enlargement was performed whenever the indexed EOA, calculated using steps 1 and 2 of the algorithm, was projected to be <0.85 cm²/m². As a result, the overall incidence of PPM in their population was only 2.5%, as opposed to the 17% that would have occurred had this prospective strategy not been utilized. Moreover, operative mortality was not increased as a result of aortic root enlargement (3.6% overall mortality) [17]. It should be noted that aortic root enlargement was performed using a novel technique consisting of the insertion of a patch made of polyethylene terephthalate.

These results demonstrate that this prospective strategy to avoid PPM can easily be applied with success. It should be emphasized that the information necessary to perform the calculation is readily available, since it requires only the patient's height and weight, and the EOA reference values for the different types and sizes of prosthesis being contemplated for operation. The latter information should readily be provided by the manufacturers and can easily be found in the literature (Table 2). In this regard, there are two caveats worth mentioning [14,24]:



- The values should be derived from *in vivo* rather than *in vitro* data, since the latter are usually too optimistic, particularly in the case of stentless valves.
- Values derived from geometric measurements (e.g. internal diameters or geometric areas) are totally inadequate, since they do not predict postoperative TPGs.

Despite these considerations, and as endorsed by the Canadian Consensus Conference on Heart Valve Surgery, the calculation of the projected indexed EOA should become an integral part of the decision-making process leading to the choice of a particular type and size of prosthesis. In this context, it should ideally be performed in the operating room, since only at that time can the aortic annulus diameter be accurately measured. As previously stated, the ideal objective is for the prosthesis to have an indexed EOA of $>0.85 \text{ cm}^2/\text{m}^2$ after operation, but lower values are probably acceptable in the less active and/or older population.

Conclusion

PPM is a highly relevant issue in valve replacement today. It is associated with inferior hemodynamics, less regression of LV hypertrophy, more cardiac events, and higher mortality

rates after AVR. The greatest impact of PPM with regards to mortality is the early postoperative period, especially in patients with depressed LV function. Contrary to other risk factors for postoperative morbidity and mortality, PPM can be largely prevented by implementing a simple prospective strategy in the operating room.

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Disclosures

The authors have no relevant financial interests to disclose.

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