Review Article

Prosthesis-patient Mismatch - What Cardiac Anesthesiologists Need to Know?

Abstract

Prosthesis-patient Mismatch (PPM) is not uncommon with an incidence reported up to 70% after aortic valve (AV) replacement. Severe forms of PPM are less common (up to 20%); PPM can lead to increased short- and long-term morbidity and mortality. It is important to discriminate PPM from other forms of prosthetic valve dysfunction. Sometimes, prosthetic valve degenerative disease may coexist with PPM. Echocardiography plays an important role in the prevention and diagnosis of PPM. Preemptive strategies to prevent PPM include insertion of newer generation prosthetic valves with better hemodynamic characteristics, stentless prosthesis, aortic root enlargement to insert a larger prosthesis, aortic homograft, and transcutaneous AV implantation. We present an illustrative case and review the literature on PPM pertinent to anesthesiologists.

Keywords: *Aortic root enlargement, aortic valve, indexed effective orifice area, patient-prosthesis mismatch*

Kathirvel Subramaniam, Soheyla Nazarnia[1]

Department of Anesthesiology, University of Pittsburgh, Pittsburgh, PA, USA, [1]Board Certified Cardiac Anesthesiologist, Tabriz, Iran

Introduction

Valve prosthesis-patient mismatch (PPM) was originally described in 1978 by Rahimtoola as "when effective orifice area (EOA) of the prosthetic valve, after insertion into the patient, is less than that of a normal human valve."[1] Based on Rahimtoola's PPM definition, the majority of patients with implanted prosthetic aortic valves (AVs) will demonstrate some degree of PPM since the prosthesis structure (leaflets, sewing ring, and struts) will introduce some degree of obstruction to blood flow across the prosthetic valve. With more insights from the scientific literature on this subject, PPM is nowadays recognized by a spectrum of hemodynamic and functional derangements when EOA of the implanted prosthetic valve is too small for the body size, or more accurately, body surface area (BSA).[2] PPM has been described in both the mitral and aortic positions and is known to be associated with inferior functional recovery, significant reduction of cardiac index, and higher incidence of congestive heart failure, cardiac events, and mortality.[2,3] One of the major goals of AV replacement (AVR) is to achieve regression of left ventricular (LV) hypertrophy and improvement of LV performance.[4,5] By the same token, incomplete regression of LV hypertrophy following AVR has been demonstrated to significantly reduce 10-year survival.[6,7] Therefore, it is crucial to avoid PPM, especially in physically active, younger patients. In this review, we describe the definition, risk factors, echocardiographic features, and differential diagnosis of PPM. We address the effect of PPM on patient outcomes and present all available preventive strategies. We also describe an illustrative case with suspected post-AVR PPM and attempt to raise the awareness for the cardiac anesthesiology community.

Case Report

A 64-year-old male with a history of obesity, obstructive sleep apnea, hypertension, hyperlipidemia, noninsulin-dependent diabetes mellitus, coronary artery disease (history of one vessel bypass to obtuse marginal [OM_1] in 2013), and valvular heart disease (history of #21 mm bioprosthetic AVR in 2013) presented with progressive dyspnea and chest pain in November of 2015. Coronary angiogram showed 75% left anterior descending artery stenosis, which then was treated with drug-eluting stent placement in an outside

How to cite this article: Subramaniam K, Nazarnia S. Prosthesis-patient mismatch - what cardiac anesthesiologists need to know?. Ann Card Anaesth 2017;20:234-42.

Address for correspondence:
Dr. Kathirvel Subramaniam, Associate Professor, Department of Anesthesiology, University of Pittsburgh, Pittsburgh, PA, USA.
E-mail: subramaniamk@upmc.edu

Video available on: www.annals.in

Access this article online

Website: www.annals.in

DOI: 10.4103/aca.ACA_9_17

Quick Response Code:

facility. The saphenous vein graft to OM_1 was found to be patent at the time of angiography. This patient also had an echocardiogram following stent placement, which showed restricted and tethered mitral valve (MV) leaflets causing severe mitral regurgitation (MR), for which he was referred to our university medical center. After admission for MV surgery, a repeat transthoracic echocardiogram (TTE) was done, which showed elevated AV peak velocity and elevated AV mean gradient in addition to severe MR [Figure 1]. The patient was listed for a redo cardiac surgery to repair/replace the MV and further evaluation of AV by transesophageal echocardiography (TEE) was planned under anesthesia. Intraoperative TEE showed thickened and restricted bioprosthetic AV leaflets with moderate eccentric aortic incompetence [Video 1]. The AV mean gradient was found to be lower under anesthesia, and effective AV area was calculated by continuity equation as 1.3 cm^2 (valve area indexed to BSA, known as indexed effective orifice area (iEOA), was 0.6 cm^2/m^2) [Figure 2a and b]. The measured dynamic velocity index was 0.38 and acceleration time was >100 ms, with a rounded contour for AV ejection waveform. The aortic annulus measured 21 mm, confirming the patient's

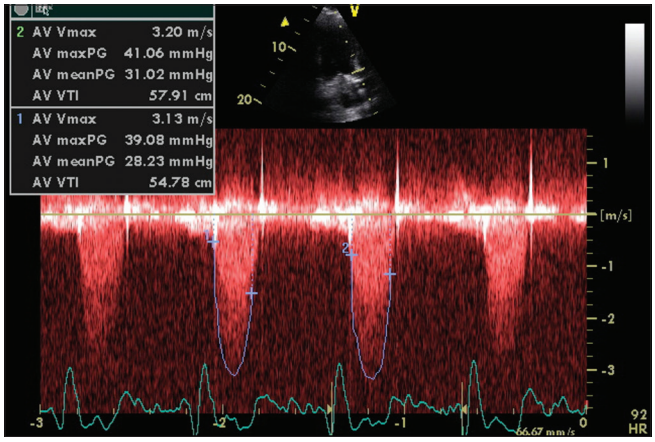

Figure 1: Continuous wave Doppler through aortic valve showing increased peak velocity (>3 m/s), rounded velocity contour, and increased mean gradients (preoperative transthoracic echocardiography)

medical records, indicating a 21 mm bioprosthesis implantation in the AV position during the previous cardiac surgery. The decision was made to proceed with AV and MV replacement. Intraoperative surgical findings confirmed 21 mm bioprosthesis and small aortic root. All preoperative and intraoperative echocardiographic findings were given in Table 1. Given the body mass index (BMI) of 34 kg/cm^2 and BSA of 2.2 cm^2, the surgical team felt that this was a case of PPM. Aortic root enlargement, 23 mm St. Jude Regent mechanical bileaflet valve implantation for aortic position, and MV replacement with 25 mm On-X valve were performed, and the patient was subsequently successfully weaned from cardiopulmonary bypass (CPB). Post-CPB, LV ejection fraction was 25%–30%, mean AV gradient was 16 mmHg, and the iEOA was calculated as 1.67 cm^2/m^2 [Figure 3]. Moderate right ventricle dysfunction following separation from CPB improved on inotropic therapy. The patient did well postoperatively and was discharged home.

Case Discussion

Echocardiographic protocols utilize peak AV velocity, iEOA, acceleration time, dynamic velocity index, and jet contour to diagnose PPM in patients with suspected prosthetic valve dysfunction (PVD).[8,9] Several echocardiographic protocols have been described to differentiate between prosthetic valve disease due to degenerative changes and PPM [Figure 4]. However, it is important to recognize that the two pathologies can coexist in any patient with PVD. Our patient, who had his AV replaced 2 years before the recent presentation, showed significant thickening and restriction of the implanted bioprosthesis on two-dimensional (2D) echocardiographic examination. iEOA <0.8 cm^2/m^2 with dynamic velocity index >0.25 suggested PPM. However, rounded contour of AV spectral waveform with acceleration time >100 ms confirmed the presence of stenotic prosthetic valve disease. It is important to note that improperly sized valves with PPM can hasten the development of early degenerative changes and premature PVD with varying degrees of

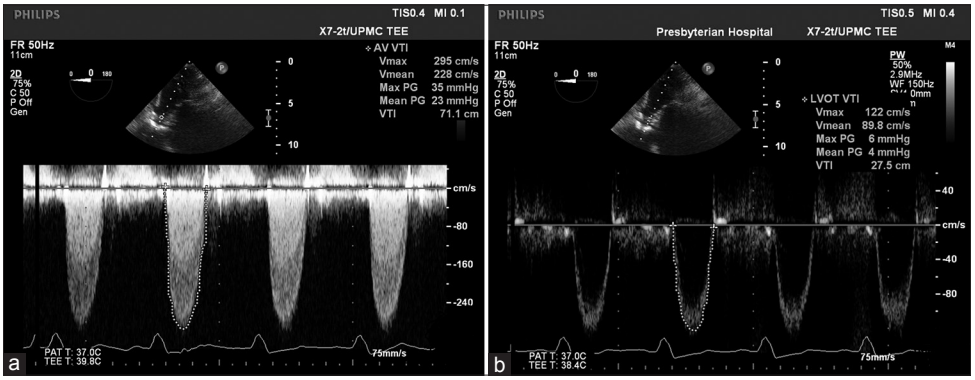

Figure 2: Transesophageal echocardiographic evaluation of aortic valve area using continuity equation and calculation of dynamic velocity index. (a) Aortic valve gradients and peak velocity by continuous wave Doppler and (b) left ventricular outflow velocity and gradients with pulse wave Doppler

Table 1: Echocardiographic findings over time in a patient with valvular heart disease

	AV peak velocity (m/s)	AV mean gradient (mmHg)	Mitral regurgitation	Aortic regurgitation	Systolic pulmonary artery pressure by echo	LV function (%)
March 2014 (routine screening TTE before total knee replacement)	2.53	17	Mild	None	46 mmHg	50-55
November 2015 (TTE during admission for left anterior descending artery stent) done outside university system	1.7	6	Severe		Severely elevated	40
January 20, 2016 (immediate preoperative TTE)	3.2	28	Moderate to severe	Mild	34 mmHg	42
February, 2016 (intraoperative prebypass TEE)	3	23	Moderate	Moderate	16 mmHg	40
February 2016 (postbypass intraoperative TEE)	2.83	16	No perivalve leak	No perivalve leak		35-40

TTE: Transthoracic echocardiogram, LV: Left ventricle, AV: Aortic valve

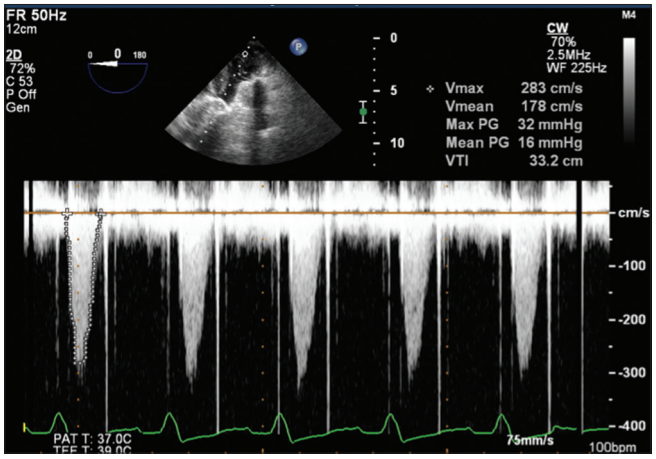

Figure 3: Continuous wave Doppler through newly implanted mechanical aortic valve showing mean gradients after cardiopulmonary bypass

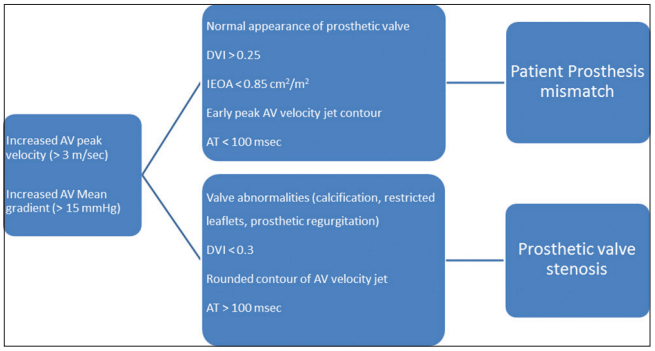

Figure 4: Echocardiographic algorithm to differentiate patient-prosthesis mismatch and prosthetic valve obstructive valve disease from progressive degeneration

prosthetic valve stenosis and regurgitation.[10] Therefore, it is crucial for clinicians to integrate the information derived from previous records, including annular size and valve size, with echocardiographic findings and patient parameters such as BSA at the time of redo cardiac surgery to prevent recurrence of similar problems in the future. Our patient presented with a higher BSA compared to that at his previous surgery, the AV annulus was small and the previously implanted 21 mm AV bioprosthesis manifested the stigmata of degenerative valve disease. Our perioperative and surgical teams concurred the diagnosis of both PPM and premature PVD and aortic root enlargement; implantation of 23 mm mechanical AV was performed to prevent future occurrence of the same problem.

Definition of PPM

The presence of PPM in post-AVR patients is best assessed using the iEOA of the prosthetic AV. Based on existing literature, PPM is defined as none or mild when iEOA of the prosthetic valve is more than 0.85 cm^2/m^2, moderate when it is between 0.65 cm^2/m^2 and 0.85 cm^2/m^2, and severe when it is less than 0.65 cm^2/m^2.[11,12] Valve PPM is not a rare occurrence, as the reported prevalence of PPM for AV position varies between 19%–70% and 2%–20% for moderate and severe PPM, respectively.[11,12]

Echocardiographic Measurements Relevant to PPM

The EOA of the prosthetic valve can be calculated using a simplified continuity equation with the information derived from Doppler echocardiography and then indexed to BSA to obtain iEOA.[13] Postoperative PPM is manifested by an unacceptably high transvalvular pressure gradient (TPG), which is mathematically expressed by hydraulic equation. As shown by hydraulic equation, pressure gradient has a direct relationship with the square of flow across the valve and an inverse relationship with square of the valvular orifice area.

$TPG = Q^2/[k6EOA^2]$

TPG = transvalvular pressure gradient, Q = transvalvular flow, EOA = effective orifice area.

Since the relation between the gradient across a stenotic valve to the valve area is curvilinear, transvalvular gradients can increase exponentially with decreased iEOA.[14] In other words, a small decrease in iEOA may potentially result in a large increase in TPG. Once EOA of a stenotic valve is reduced to less than 35% of normal, the gradient rises precipitously.[15] In Pibarot *et al*.'s study, patients with PPM (iEOA ≤0.85 cm^2/m^2) and those with no PPM had a TPG of 22 ± 8 mmHg and 15 ± 6 mmHg, respectively.[16] The greatest reduction in cardiac index and increase in TPG are seen in patients with severe PPM (iEOA ≤0.65 cm^2/m^2). TPG can be greatly impacted by hemodynamics, ventricular function, and other valvular pathologies. In our patient, measured iEOA was indicative of severe PPM and the unexpectedly low mean gradients across the prosthetic valve were attributed to the presence of severe MR and LV ejection fraction, resulting in low flow status.

iEOA is a reliable indicator of PPM and it is important to document iEOA immediately after AVR in the operating room, and in the postoperative period and reference, those values for follow-up. As for our patient, we could not obtain a postoperative iEOA documented following the first AVR performed in an outside hospital, but our post-redo-AVR iEOA was calculated as 1.67 cm^2/m^2. Bleiziffer *et al*.[17] in a prospective study predicted PPM in 11.7% (14/119) of AVR patients using intraoperative TEE. Sixty-five percent of patients (9/14) who were predicted to develop PPM actually manifested the stigmata of mismatch during follow-up.

Prediction of postoperative iEOA will help guide the surgeon's decision on proper prosthesis size or alternate procedures (described below) to prevent the development of PPM. Bleiziffer *et al*.[17] studied several methods to predict post-AVR iEOA in their cohort of 383 patients. The study compared four different methods. The first used their hospital database of patients who underwent AVR and had a follow-up echocardiographic examination 6 months later to calculate post-AVR iEOA; the second method used indexed geometric orifice area (IGA); the third method used commercially available charts from various sources, including manufacturers; and the fourth method used iEOA published in the literature. Indexed IGA used in the second method is defined as fixed internal diameter of the valve prosthesis (provided by the manufacturer) divided by the BSA of the patient. Severe PPM developed in 24 (6.3%) of the patients. The proportion of patients predicted to have PPM by the four study methods were as follows; 22/24 (92%) by method 1, 0/24 (0%) by method 2, 13/22 (56%) by method 3, and 19/22 (86%) by method 4. The second method using IGA performed worse, and the performance of method 3 varied significantly based on the chart used. In contrast to iEOA, IGA has not been shown to have any relationship whatsoever with postoperative TPG and hemodynamic improvements. According to Koch *et al*., there is no correlation between IGA and postoperative recovery and outcomes in patients undergoing AVR.[18] Furthermore, IGA varies from one prosthesis to the other and in general, overestimates EOA. Similarly, values from charts that use *in vitro* methods to calculate iEOA will result in higher values (by 10%–15%); this must be avoided to prevent undersizing of the prosthetic valve.[17]

Joshi *et al*. investigated the incidence of PPM in an Indian population (n = 668).[19] The incidence of any PPM was 9% and severe PPM occurred only in 0.5% of patients. Indexed aortic annulus size <16 mm/m^2 was predictive of PPM. It may be useful to index annulus size to BSA to decide on the best therapeutic options before AVR. Accurate estimation of aortic annular diameter is crucial, and 2D measurements may underestimate annular diameter.[20] Three-dimensional (3D) measurement of the longest annular diameter using off-line multiplanar reconstruction method or biplane method should be used to measure aortic annular diameter.[20] da Silva *et al*.[21] showed that patients who had 3D TEE measurements of the aortic annulus had a significantly (P = 0.03) lower incidence of severe PPM after transcatheter AV implantation (TAVI) when compared to those who had annulus measured using 2D TTE (0% with 3D TEE vs. 20% with 2D TTE). While computerized tomography is the most accurate methodology for measuring aortic annulus preoperatively before TAVI, 3D TEE is the most practical method in the operating room for measurement of annular diameter during surgical AVR (SAVR).[20,22,23]

Risk Factors for Development of PPM

Reduction of iEOA to mild and moderate degrees often leads to no negative symptomatic or hemodynamic impact. Nevertheless, endothelialization, tissue growth, and pannus and thrombus formation with time will further decrease the EOA to produce symptoms. The presence of PPM at the time of implantation can accelerate structural degeneration of bioprosthetic valves. We feel that PPM following the first AVR in our patient led to an early degeneration of prosthetic AV within a shorter period following implantation.

Several other factors influence the development and impact of PPM in any individual patient. The combination of small aortic annulus and higher BSA in a functionally active individual should alert the echocardiographer and surgeon to undertake the necessary steps to prevent PPM.[24,25] It is important to note that an EOA, which is considered acceptable in a small elderly patient with a sedentary lifestyle, is often inadequate for a younger active individual.[24-28] However, the frequency of PPM may be higher in elderly patients with AS.[28] In contrast to the larger annuli seen in younger patients presenting with aortic insufficiency, older patients with AS often present

with annular fibrosis and calcification, resulting in a small annular size. This in turn will limit the size of the prosthesis implanted in older patients with AS, predisposing them to PPM.[28]

Other risk factors reported to be associated with PPM include female gender, hypertension, diabetes, and elevated serum creatinine.[10] The most susceptible patients include the ones with coronary artery disease requiring revascularization, depressed LV systolic function, severe LV hypertrophy, significant diastolic dysfunction, concomitant MR, and paradoxical low-flow low-gradient aortic stenosis (PLF-LG AS).[29-32] It is also known that patients with a bioprosthesis have a three-time higher risk of developing PPM compared to those with mechanical AVs.[10]

Effects of PPM on Short-term and Long-term Outcomes

PPM can be associated with less improvement in symptoms and functional class after AVR, worse hemodynamics at rest and during exercise, less improvement in coronary flow reserve, less regression of LV hypertrophy, more frequent postoperative cardiac events, increased short-term mortality, and ultimately, poor long-term outcomes.[10,11] As far as the impact of PPM on patient outcomes is concerned, study results are conflicting. Failure to demonstrate an effect on mortality could be explained by the low incidence of significant PPM. Few studies have used IGA and charts from *in vitro* resources for iEOA calculation, which could have contributed to overestimation of iEOA and wrong classification of patient groups.[33-35] Furthermore, it is very difficult to differentiate the effects of PPM on patient outcomes from other coexisting cardiac pathologies (LV systolic dysfunction, diastolic dysfunction, and PLF-LG AS), as the risk factors for these pathologies overlap and their impact can be additive in increasing mortality. The effect of PPM may also vary according to the patient population studied; PPM has been shown to have negligible effects on patients of Asian (Japanese and Indian) descent compared to their Caucasian counterparts.[19,36]

In this context of the low incidence of significant PPM, systematic reviews of pooled data were found to be useful in examining the effects of PPM. These reviews have consistently shown increased mortality with significant PPM.[10,30,37] A recently published review by Dayan *et al*.[10] assessed predictors of PPM and their association with perioperative and overall mortality. Data from 382 studies published between 1965 and 2014 were assessed, of which 58 studies (40,381 patients) were found to be suitable for analysis. PPM was present in 43% of patients. Perioperative and overall mortality was increased in patients with severe PPM, whereas moderate PPM was associated with higher incidence of perioperative mortality but not overall mortality. The impact of PPM on mortality was found to be higher in studies, in which the mean age was <70 years, and also in studies with a higher number of patients undergoing combined AVR and coronary revascularization. They also found that the impact of PPM was less pronounced in patients with larger BMIs (>28 kg/m^2). This is most likely due to the fact that cardiac output requirement may not increase in direct proportion to BMI. This could also be because of different formulae used to calculate BSA and BMI in those studies. Physiologically, inferior hemodynamic and symptomatic status of the patients with diagnosed PPM is attributed to the hemodynamic consequences of increased afterload to LV output across the implanted valve, as well as the persistence of abnormal coronary flow reserve. Persistence of untreated mitral and tricuspid regurgitation may also contribute to poor outcomes.

The effect of LV systolic function on PPM and mortality has been the subject of several studies. Blais *et al*.[38] conducted a prospective study analyzing baseline risk factors and subsequent short-term mortality in 1265 consecutive patients undergoing AVR between 1992 and 2001. Moderate-to-severe PPM was diagnosed in 38% of the patients. Thirty-day mortality was 4.6% and PPM and LV ejection fraction <40% independently predicted short-term mortality. Risk of mortality was higher with LV ejection fraction <40% compared to patients with LV ejection fraction >40% for mild, moderate, and severe degrees of PPM. In a best evidence paper, Urso *et al*.[30] reviewed the effect of PPM on 30-day and short-term mortality and found that moderate PPM was well tolerated, except for in patients with poor LV function, but severe PPM was a predictor of overall mortality irrespective of LV function. Ruel *et al*.[39] reported that patients with PPM (iEOA <0.85 cm^2/m^2) after AVR in the presence of impaired LV function had decreased late survival and decreased freedom from heart failure symptoms compared to patients with PPM and normal LV function. Impaired LV function leads to low gradients in patients with AS. The impact of PPM on patients (n = 664) with low-gradient AS was evaluated by Kulik *et al*.[40] When compared with patients without low-gradient AS, patients with low-gradient AS had significantly lower mortality freedom from congestive heart failure at 10 years and any PPM was independently associated with increased rates of congestive heart failure and impaired LV mass regression. Several other authors reported similar findings in patients with impaired LV function; we can conclude that it is important to avoid any degree of PPM in the presence of preoperative impaired LV function. Our patient had borderline LV function, justifying the strategy of aortic root enlargement to implant a bigger prosthesis. Paradoxical low flow aortic stenosis (PLF-AS) is another factor that has been shown to interact with PPM and affect

outcomes. PLF-AS is characterized by normal ventricular function, severe AS, and low stroke volume and gradients. Mohty *et al*. studied 677 patients with severe AS; 15% of them had preoperative PLF-AS and developed PPM after surgery. Ten-year survival was significantly affected in patients with PLF-AS and PPM compared with patients with no PPM or no PLF-AS.[32]

Preemptive Strategies and PPM

The most appropriate way to preempt PPM is to predict it at the time of surgery and implement a validated strategy. It is only prudent to avoid moderate-to-severe PPM in physically active younger individuals and in patients with systolic dysfunction. It might be reasonable to accept some degree of PPM to avoid complications of prolonged CPB in elderly patients with a sedentary lifestyle who have adequate LV function. Insertion of a small prosthesis also does not appear to result in PPM in patients with small body size.

Pibarot and Dumesnil described a stepwise strategy to avoid PPM in the aortic position.[41] If the originally intended prosthesis is projected to cause PPM (i.e., if predicted postoperative is ≤0.85 cm^2/m^2), the surgeon must decide whether to pursue alternative options or stay the course. Further decision-making must be based on a calculated judgment for the individual patient with specific hemodynamics, physical condition, and lifestyle, taking into consideration the risk–benefit ratio of revision surgery. Reference values n5for EOA should be derived from *in vivo* echocardiographic studies as described in the previous sections. These reference values for EOA must be readily available in the operating room to determine whether the considered prosthetic valve meets the requirements to avoid PPM.

Despite the obvious benefits of insertion of stented bioprosthesis with the lack of requirement for anticoagulation, the obstructive nature of the stent in conventional stented bioprosthesis is a major disadvantage because they lead to a nonphysiological flow pattern, residual TPG, and ultimately PPM. Alternate options include a selection of a newer generation bioprosthetic valve (e.g., St. Jude Trifecta pericardial bioprosthetic valve), aortic homograft, Ross operation, stentless bioprosthesis, mechanical prosthesis (i.e., St. Jude Regent mechanical bileaflet valve), and TAVI.[42]

In general, the hemodynamic performance of newer generations of prosthetic valves is better than older generations, and the supra-annular stented bioprosthesis has been proven to be superior to the annular ones, providing a larger iEOA.[43,44] The incidence of PPM with the newer generation of mechanical valves also appears to be lower, and they are well tolerated in patients with small annuli.[45,46] Superior hemodynamic performance of stentless bioprosthesis can be observed both at rest and during exercise.[47,48] This is not to overlook the complexity of inserting stentless valves, which requires longer CPB and ischemic times and superior surgical skills. None of these procedures completely preclude the occurrence of postoperative PPM, as a considerable number of patients undergoing Freestyle stentless bioprosthesis implantation following full root replacement have been diagnosed with mismatch. The lack of success in these cases might be attributed to the technical difficulties involving stentless valve implantation.[49]

Insertion of a larger prosthesis can be done along with aortic root enlargement to avoid PPM.[50-52] Castro *et al*.[53] conducted a study including 657 consecutive patients undergoing AVR. In this series of patients, aortic root enlargement was performed routinely whenever iEOA was projected to be ≤0.85 cm^2/m^2. The overall incidence of PPM in Castor's study was 2.5%. The authors concluded that aortic root enlargement could be used to successfully preempt PPM without any increase in operative risk. Penaranda *et al*.[54] reviewed the records of 117 octogenarian patients who underwent AVR for AV pathology. In this study, 87 patients received a 19 mm prosthetic valve in the aortic position and 30 patients underwent aortic root enlargement, followed by a 21 mm prosthetic AV. The study results suggested that aortic root enlargement in octogenarian patients allows for insertion of larger prostheses with better hemodynamic performance and less PPM without any increase in operative morbidity and mortality. Obviously, undertaking each of these options or conversely carrying on with the primary planned surgery must depend on patient's physical status and the surgeon's skill level.

More recently, TAVI has resulted in improved hemodynamic performance although application of this intervention is limited to small size devices due to concerns about vascular access and its related potential complications. In a randomized study, Pibarot *et al*. assessed the incidence of PPM in two groups of patients undergoing TAVI or SAVR. The results showed that in patients with severe AS and high surgical risk, PPM appears to be more frequent and more severe following SAVR than TAVI.[55] Several other studies have shown that the prevalence of PPM is significantly reduced in TAVI patients when compared to SAVR.[56-58] This can be attributed to the lack of a sewing rim in the TAVI device, resulting in a larger EOA. Nevertheless, in the context of absent long-term results and evidence of immediate postprocedural complications of TAVI (paravalvular leakage, cardiac conduction disturbance, vascular injury, and annulus rupture), the risks may outweigh the hemodynamic advantages achieved by transcatheter intervention.[59] Hence, the role of TAVI in preventing

PPM in patients with small aortic root has yet to be determined with further studies that include a larger number of patients and longer follow-up periods.[59]

Therefore, in patients undergoing AVR for aortic stenosis, improvement of hemodynamic performance, as well as the extent of LV mass regression, is directly related to the type and size of implanted prosthetic valve. Therefore, for PPM to be avoided, extreme care is needed when choosing the most appropriate prosthetic valve, while balancing the risk–benefit ratio at each step of the procedure.

Acknowledgment

Authors acknowledge the help with language and grammar from Christine Heiner, Scientific Writer from Department of Anesthesiology, University of Pittsburgh, Pittsburgh, PA, USA.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Rahimtoola SH. The problem of valve prosthesis-patient mismatch. Circulation 1978;58:20-4.
2. Dahou A, Mahjoub H, Pibarot P. Prosthesis-patient mismatch after aortic valve replacement. Curr Treat Options Cardiovasc Med 2016;18:67. [Doi: 10.1093/icvts/ivw397].
3. Zhang JF, Wu YC, Shen WF, Kong Y. Impact of prosthesis-patient mismatch on survival after mitral valve replacement: A systematic review. Chin Med J (Engl) 2013;126:3762-6.
4. Kennedy JW, Doces J, Stewart DK. Left ventricular function before and following aortic valve replacement. Circulation 1977;56:944-50.
5. Pantely G, Morton M, Rahimtoola SH. Effects of successful, uncomplicated valve replacement on ventricular hypertrophy, volume, and performance in aortic stenosis and in aortic incompetence. J Thorac Cardiovasc Surg 1978;75:383-91.
6. Bortolotti U, Milano A, Mossuto E, Mazzaro E, Thiene G, Casarotto D. Early and late outcome after reoperation for prosthetic valve dysfunction: Analysis of 549 patients during a 26-year period. J Heart Valve Dis 1994;3:81-7.
7. Lund O, Kristensen LH, Baandrup U, Hansen OK, Nielsen TT, Emmertsen K, *et al.* Myocardial structure as a determinant of pre- and postoperative ventricular function and long-term prognosis after valve replacement for aortic stenosis. Eur Heart J 1998;19:1099-108.
8. Blauwet LA, Miller FA Jr. Echocardiographic assessment of prosthetic heart valves. Prog Cardiovasc Dis 2014;57:100-10.
9. Zoghbi WA, Chambers JB, Dumesnil JG, Foster E, Gottdiener JS, Grayburn PA, *et al.* Recommendations for evaluation of prosthetic valves with echocardiography and Doppler ultrasound: A report From the American Society of Echocardiography's Guidelines and Standards Committee and the Task Force on Prosthetic Valves, developed in conjunction with the American College of Cardiology Cardiovascular Imaging Committee, Cardiac Imaging Committee of the American Heart Association, the European Association of Echocardiography, a registered branch of the European Society of Cardiology, the Japanese Society of Echocardiography and the Canadian Society of Echocardiography, endorsed by the American College of Cardiology Foundation, American Heart Association, European Association of Echocardiography, a registered branch of the European Society of Cardiology, the Japanese Society of Echocardiography, and Canadian Society of Echocardiography. J Am Soc Echocardiogr 2009;22:975-1014.
10. Dayan V, Vignolo G, Soca G, Paganini JJ, Brusich D, Pibarot P. Predictors and outcomes of prosthesis-patient mismatch after aortic valve replacement. JACC Cardiovasc Imaging 2016;9:924-33.
11. Pibarot P, Dumesnil JG. Hemodynamic and clinical impact of prosthesis-patient mismatch in the aortic valve position and its prevention. J Am Coll Cardiol 2000;36:1131-41.
12. Kohsaka S, Mohan S, Virani S, Lee VV, Contreras A, Reul GJ, *et al.* Prosthesis-patient mismatch affects long-term survival after mechanical valve replacement. J Thorac Cardiovasc Surg 2008;135:1076-80.
13. Otto CM, Pearlman AS, Gardner CL, Kraft CD, Fujioka MC. Simplification of the Doppler continuity equation for calculating stenotic aortic valve area. J Am Soc Echocardiogr 1988;1:155-7.
14. Gorlin R, Gorlin SG. Hydraulic formula for calculation of the area of the stenotic mitral valve, other cardiac valves, and central circulatory shunts. I. Am Heart J 1951;41:1-29.
15. Frank S, Johnson A, Ross J Jr. Natural history of valvular aortic stenosis. Br Heart J 1973;35:41-6.
16. Pibarot P, Dumesnil JG, Lemieux M, Cartier P, Métras J, Durand LG. Impact of prosthesis-patient mismatch on hemodynamic and symptomatic status, morbidity and mortality after aortic valve replacement with a bioprosthetic heart valve. J Heart Valve Dis 1998;7:211-8.
17. Bleiziffer S, Eichinger WB, Hettich I, Guenzinger R, Ruzicka D, Bauernschmitt R, *et al.* Prediction of valve prosthesis-patient mismatch prior to aortic valve replacement: Which is the best method? Heart 2007;93:615-20.
18. Koch CG, Khandwala F, Estafanous FG, Loop FD, Blackstone EH. Impact of prosthesis-patient size on functional recovery after aortic valve replacement. Circulation 2005;111:3221-9.
19. Joshi SS, Ashwini T, George A, Jagadeesh AM. Patient prosthesis mismatch after aortic valve replacement: An Indian perspective. Ann Card Anaesth 2016;19:84-8.
20. Cerillo AG, Mariani M, Berti S, Glauber M. Sizing the aortic annulus. Ann Cardiothorac Surg 2012;1:245-56.
21. da Silva C, Sahlen A, Winter R, Bäck M, Rück A, Settergren M, *et al*. Prosthesis-patient mismatch after transcatheter aortic valve implantation: Impact of 2D-transthoracic echocardiography versus 3D-transesophageal echocardiography. Int J Cardiovasc Imaging 2014;30:1549-57.
22. Kenny C, Monaghan M. How to assess aortic annular size before transcatheter aortic valve implantation (TAVI): The role of echocardiography compared with other imaging modalities. Heart 2015;101:727-36.
23. Kempfert J, Van Linden A, Lehmkuhl L, Rastan AJ, Holzhey D, Blumenstein J, *et al.* Aortic annulus sizing: Echocardiographic versus computed tomography derived measurements in comparison with direct surgical sizing. Eur J Cardiothorac Surg 2012;42:627-33.
24. Hernández-Vaquero D, Llosa JC, Díaz R, Khalpey Z, Morales C,

Álvarez R, *et al.* Impact of patient-prosthesis mismatch on 30-day outcomes in young and middle-aged patients undergoing aortic valve replacement. J Cardiothorac Surg 2012;7:46.

25. Hernandez-Vaquero D, Garcia JM, Diaz R, Calvo D, Khalpey Z, Hernández E, *et al.* Moderate patient-prosthesis mismatch predicts cardiac events and advanced functional class in young and middle-aged patients undergoing surgery due to severe aortic stenosis. J Card Surg 2014;29:127-33.
26. Moon MR, Pasque MK, Munfakh NA, Melby SJ, Lawton JS, Moazami N, *et al.* Prosthesis-patient mismatch after aortic valve replacement: Impact of age and body size on late survival. Ann Thorac Surg 2006;81:481-8.
27. Mohty D, Dumesnil JG, Echahidi N, Mathieu P, Dagenais F, Voisine P, *et al.* Impact of prosthesis-patient mismatch on long-term survival after aortic valve replacement: Influence of age, obesity, and left ventricular dysfunction. J Am Coll Cardiol 2009;53:39-47.
28. Hernandez-Vaquero D, Calvo D, Garcia JM, Lozano I, Morales C, Naya JL, *et al.* Influence of patient-prosthesis mismatch in the octogenarian undergoing surgery for aortic valve replacement due to severe stenosis. Rev Esp Cardiol 2011;64:774-9.
29. Tully PJ, Aty W, Rice GD, Bennetts JS, Knight JL, Baker RA. Aortic valve prosthesis-patient mismatch and long-term outcomes: 19-year single-center experience. Ann Thorac Surg 2013;96:844-50.
30. Urso S, Sadaba R, Aldamiz-Echevarria G. Is patient-prosthesis mismatch an independent risk factor for early and mid-term overall mortality in adult patients undergoing aortic valve replacement? Interact Cardiovasc Thorac Surg 2009;9:510-8.
31. Boulogne C, Mohty D. Aortic prosthesis-patient mismatch in patients with paradoxical low flow severe aortic stenosis: A dreadful combination. Arch Cardiovasc Dis 2015;108:1-4.
32. Mohty D, Boulogne C, Magne J, Pibarot P, Echahidi N, Cornu E, *et al.* Prevalence and long-term outcome of aortic prosthesis-patient mismatch in patients with paradoxical low-flow severe aortic stenosis. Circulation 2014;130 11 Suppl 1:S25-31.
33. House CM, Nelson WB, Kroshus TJ, Dahiya R, Pibarot P. Manufacturer-provided effective orifice area index charts and the prevention of prosthesis-patient mismatch. J Heart Valve Dis 2012;21:107-11.
34. House CM, Nelson WB, Raikar GV, Ahmed I, Dahiya R. How reliable is an effective orifice area indexed chart? J Heart Valve Dis 2009;18:530-4.
35. Swinkels BM, de Mol BA, Kelder JC, Vermeulen FE, ten Berg JM. Prosthesis-patient mismatch after aortic valve replacement: Effect on long-term survival. Ann Thorac Surg 2016;101:1388-94.
36. Sakamoto Y, Hashimoto K. Update on aortic valve prosthesis-patient mismatch in Japan. Gen Thorac Cardiovasc Surg 2013;61:669-75.
37. Head SJ, Mokhles MM, Osnabrugge RL, Pibarot P, Mack MJ, Takkenberg JJ, *et al.* The impact of prosthesis-patient mismatch on long-term survival after aortic valve replacement: A systematic review and meta-analysis of 34 observational studies comprising 27 186 patients with 133 141 patient-years. Eur Heart J 2012;33:1518-29.
38. Blais C, Dumesnil JG, Baillot R, Simard S, Doyle D, Pibarot P. Impact of valve prosthesis-patient mismatch on short-term mortality after aortic valve replacement. Circulation 2003;108:983-8.
39. Ruel M, Al-Faleh H, Kulik A, Chan KL, Mesana TG, Burwash IG. Prosthesis-patient mismatch after aortic valve replacement predominantly affects patients with preexisting left ventricular dysfunction: Effect on survival, freedom from heart failure, and left ventricular mass regression. J Thorac Cardiovasc Surg 2006;131:1036-44.
40. Kulik A, Burwash IG, Kapila V, Mesana TG, Ruel M. Long-term outcomes after valve replacement for low-gradient aortic stenosis: Impact of prosthesis-patient mismatch. Circulation 2006;114 1 Suppl:I553-8.
41. Pibarot P, Dumesnil JG. Prosthesis-patient mismatch: Definition, clinical impact, and prevention. Heart 2006;92:1022-9.
42. Barratt-Boyes BG, Christie GW. What is the best bioprosthetic operation for the small aortic root? allograft, autograft, porcine, pericardial? Stented or unstented? J Card Surg 1994;9 2 Suppl: 158-64.
43. Yadlapati A, Diep J, Barnes MJ, Grogan T, Bethencourt DM, Vorobiof G. Comprehensive hemodynamic performance and frequency of patient-prosthesis mismatch of the St. Jude Medical Trifecta bioprosthetic aortic valve. J Heart Valve Dis 2014;23:516-23.
44. Deutsch MA, Prinzing A, Fiegl K, Wottke M, Badiu CC, Krane M, *et al.* Early haemodynamic performance of a latest generation supra-annular aortic bioprosthesis: Experience from a large single-centre series. Eur J Cardiothorac Surg 2016;49:1691-8.
45. Liu Y, Zhang EY, Fang Z, Gu J, Qian H. Mid-long-term clinical results and hemodynamic evaluation of aortic valve replacement with 17 mm St. Jude medical regent valve. Sichuan Da Xue Xue Bao Yi Xue Ban 2013;44:316-8, 326.
46. Mizoguchi H, Sakaki M, Inoue K, Iwata T, Tei K, Miura T. Mid-term results of small-sized St. Jude Medical Regent prosthetic valves (21 mm or less) for small aortic annulus. Heart Vessels 2013;28:769-74.
47. David TE, Feindel CM, Bos J, Sun Z, Scully HE, Rakowski H. Aortic valve replacement with a stentless porcine aortic valve. A six-year experience. J Thorac Cardiovasc Surg 1994;108:1030-6.
48. Dumesnil JG, LeBlanc MH, Cartier PC, Métras J, Desaulniers D, Doyle DP, *et al.* Hemodynamic features of the freestyle aortic bioprosthesis compared with stented bioprosthesis. Ann Thorac Surg 1998;66 6 Suppl: S130-3.
49. Albert A, Florath I, Rosendahl U, Hassanein W, Hodenberg EV, Bauer S, *et al.* Effect of surgeon on transprosthetic gradients after aortic valve replacement with freestyle stentless bioprosthesis and its consequences: A follow-up study in 587 patients. J Cardiothorac Surg 2007;2:40.
50. Nakano S, Matsuda H, Shimazaki Y, Taniguchi K, Kaneko M, Ueda T, *et al*. An appraisal of patch enlargement of the small aortic annulus in 33 patients undergoing aortic valve replacement. Eur J Cardiothorac Surg 1992;6:347-9.
51. Sommers KE, David TE. Aortic valve replacement with patch enlargement of the aortic annulus. Ann Thorac Surg 1997;63:1608-12.
52. Bortolotti U, Celiento M, Milano AD. Enlargement of the aortic annulus during aortic valve replacement: A review. J Heart Valve Dis 2014;23:31-9.
53. Castro LJ, Arcidi JM Jr., Fisher AL, Gaudiani VA. Routine enlargement of the small aortic root: A preventive strategy to minimize mismatch. Ann Thorac Surg 2002;74:31-6.
54. Penaranda JG, Greason KL, Pislaru S, Schaff HV, Daly RC, Park SJ, *et al*. Aortic root enlargement in octogenarian patients results in less patient-prosthesis mismatch. Ann Thorac Surg 2014;97:1533-8.
55. Pibarot P, Weissman NJ, Stewart WJ, Hahn RT,

Lindman BR, McAndrew T, *et al*. Incidence and sequelae of prosthesis-patient mismatch in transcatheter versus surgical valve replacement in high-risk patients with severe aortic stenosis: A PARTNER trial cohort – A analysis. J Am Coll Cardiol 2014;64:1323-34.

56. Giannini C, Petronio AS, Nardi C, De Carlo M, Guarracino F, Delle Donne MG, *et al*. Left ventricular reverse remodeling in percutaneous and surgical aortic bioprostheses: An echocardiographic study. J Am Soc Echocardiogr 2011;24:28-36.
57. Kamperidis V, van Rosendael PJ, de Weger A, Katsanos S, Regeer M, van der Kley F, *et al.* Surgical sutureless and transcatheter aortic valves: Hemodynamic performance and clinical outcomes in propensity score-matched high-risk populations with severe aortic stenosis. JACC Cardiovasc Interv 2015;8:670-7.
58. Takagi H, Umemoto T; ALICE (All-Literature Investigation of Cardiovascular Evidence) Group. Prosthesis-patient mismatch after transcatheter aortic valve implantation. Ann Thorac Surg 2016;101:872-80.
59. Morita S. Aortic valve replacement and prosthesis-patient mismatch in the era of trans-catheter aortic valve implantation. Gen Thorac Cardiovasc Surg 2016;64:435-40.