5

Patient-Prosthesis Mismatch After Aortic Valve Replacement

Wauthy Pierre and Malekzadeh-Milani Sophie G.
Brugmann University Hospital,Brussels, Belgium

1. Introduction

Aortic valve replacement (AVR) is the treatment of choice for the majority of symptomatic adults with aortic valve stenosis. Despite improvements in bioprosthesis durability and reduction of complication rate (both thrombotic and hemorrhagic) of mechanical prosthesis, the ideal valve prosthesis is still elusive.

The hemodynamic performance of the native cardiac valve still outrivals that of prosthesis. In a way, any implanted cardiac prosthesis valve is stenotic compared to its native counterpart.

The concept of patient-prosthesis mismatch (PPM) was first described by Rahimtoola in 1978. According to this author, PPM exists whenever the effective orifice area (EOA) of an implanted prosthesis is inferior to the normal human valve (Rahimtoola, 1978). It can thus be said that, in this situation, the implanted prosthesis is stenotic compared to the normal native valve. On echocardiographic evaluation, those patients show a high transprothetic gradient despite a normal prosthetic valve function. The smaller the prosthetic valve EOA and the larger the patients body surface area, the more severe will be the mismatch and the observed gradient. Thus, the most useful definition and quantification of PPM is the ratio EOA/body surface area (EOA indexed to body surface area).

The prevalence of moderate PPM varies in different studies from 20 to 70% of cases whereas severe PPM is present in 2 to 11% (Pibarot and Dumesnil, 2006). PPM is thus a frequently encountered hemodynamic problem after aortic valve replacement.

2. Definition of the patient-prosthesis mismatch

Theoretically, an observed high transprosthetic gradient can result from two distinct situations. First, a "pathologic" obstruction can result from malfunction of the prosthesis: the motion of a mechanical prosthesis can be hindered by thrombus or pannus while deterioration of a bioprosthesis can result in rigidification of its leaflets. Besides, endocarditis can cause obstructive vegetation masses limiting leaflet motion. Second, a "physiologic" obstruction exists when the normally functioning prosthetic valve has too small EOA to accommodate the cardiac output without generating too much of a gradient. In all cases, a component of perivalvular obstacle must be excluded before blaming the prosthesis.

Patient-prosthesis mismatch is present when the effective orifice area (EOA) of a prosthetic valve is too small in relation to the body size of the patient. The hemodynamic consequence is the higher than expected gradient observed through a normally functioning prosthetic valve.

The clinical significance of PPM is diversely appreciated in the literature. For some authors, the consequences are minimal whereas for others, more severe PPM can even affect postoperative survival. This discordance is due in fact to different ways of evaluating EOA. As a whole, studies based on an in vivo evaluation of the indexed EOA tend to report clinical implications (Blais et al., 2003, Kulik et al., 2006, Ruel et al., 2006, Ruel et al., 2004, Tasca et al., 2006). In the contrary, the in vitro evaluation of the indexed EOA tends to underestimate clinical implications of PPM (Koch et al., 2005).
The transvalvular gradient (TVG) is determined by the hydraulic equation:

$$TVG=Q^2/(kxEOA^2) \quad (1)$$

Q stands for flow and k is a constant.
This equation shows that the transvalvular gradient is directly related to the square of transvalvular flow and inversely related to the square to the valve EOA (Effective Orifice Area of the valve). The flow is dependent on cardiac output which is at rest related to body surface area (BSA).
Mismatch can occur in aortic position and in mitral position. We will focus on the aortic PPM.
There is a large body of evidence that the best variable to evaluate transvalvular gradient at rest and during exercise is the indexed EOA: EOA is divided by the body surface area (Dumesnil and Pibarot, 2011, Pibarot et al., 2000, Zoghbi et al., 2009, Bleiziffer et al., 2007). This indexed EOA is the key factor used to define mismatch. Pibarot showed that the relation between transvalvular gradient and indexed EOA is curvilinear and that the gradient increases exponentially when the indexed EOA is inferior to 0.8 to 0.9 cm^2/m^2 (Pibarot and Dumesnil, 2000). The relation of the transvalvular gradient and indexed EOA are curvilinear at rest (Figure 1) and in stress conditions (Figure 2).

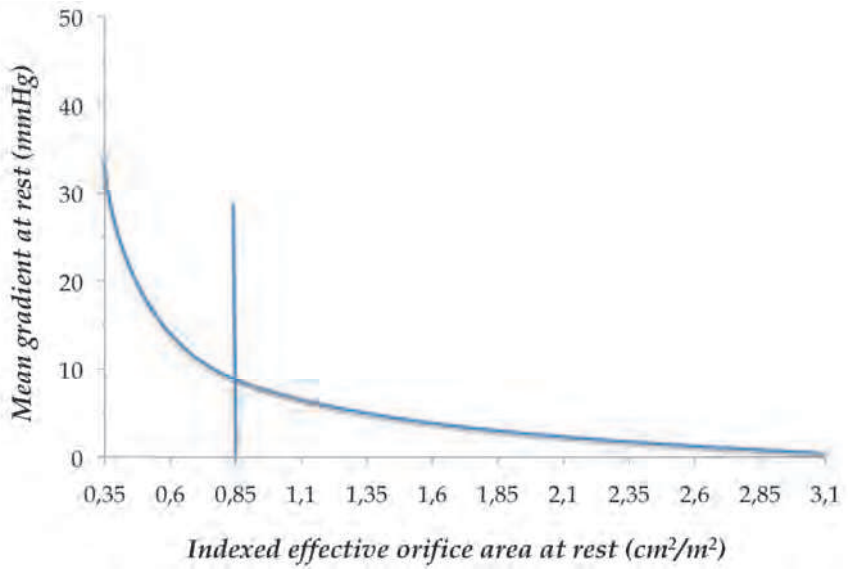


Fig. 1. Curvilinear relation of the gradient and indexed EOA at rest.

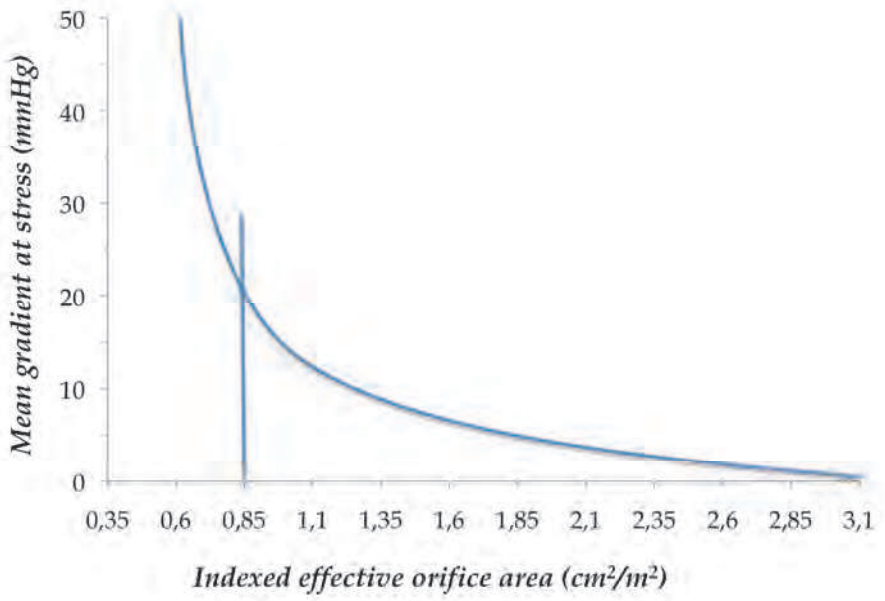


Fig. 2. Curvilinear relation of the gradient and indexed EOA at stress.

Based in this chart, PPM is considered present if indexed EOA (iEOA) is < 0,85 cm^2/m^2. It is graded moderate if the iEOA stands between 0,65 and 0,85 cm^2/m^2 and severe if less than 0,65 cm2/m2 (Pibarot and Dumesnil, 2000, Pibarot and Dumesnil, 2006).

3. Identification of PPM

Patient-prosthesis mismatch has several major clinical impacts described below and these impacts increase proportionally with the severity of PPM (Blais et al., 2003, Milano et al., 2002). It is thus important to quantify the severity of this hemodynamic situation.

PPM can be diagnosed and quantified on echocardiography when iEOA is measured. It can also be predicted or estimated at the time of surgery by using the projected EOA derived from in vivo studies and available for each type and size of prosthetic valve as illustrated in Table 1.

Echocardiography is the gold standard for the non invasive evaluation of prosthetic valve function. It is more demanding to perform and interpret data from a prosthetic valve compared to native valve. However, EOA can be calculated on echocardiography and with some other useful measurements lead to the diagnosis of PPM.

The degree of obstruction, the start point of the valve assessment, varies with the type and the size of the valve. To some extend every prosthetic valve is at least partly restrictive resulting in a mild acceleration though the prosthetic orifice. It may be difficult to differentiate obstructive hemodynamic conditions due to valve design from those of mild obstruction due to prosthetic dysfunction and from PPM.

A full echocardiography study is mandatory. The report should include height, weight, BSA, blood pressure, age, gender and the type of prosthetic valve implanted.

	Medtronic Freestyle®					
Prosthesis size	**19**	**21**	**23**	**25**	**27**	**29**
EOA (cm^2/m^2)	1,15	1,35	1,48	2	2,32	
BSA (m^2)						
1	1,15	1,35	1,48	2,00	2,32	
1,1	1,04	1,23	1,34	1,82	2,11	
1,2	0,96	1,12	1,23	1,67	1,93	
1,3	0,88	1,04	1,14	1,54	1,78	
1,4	0,82	0,96	1,06	1,43	1,66	
1,5	0,77	0,90	0,99	1,33	1,55	
1,6	0,72	0,84	0,92	1,25	1,45	
1,7	0,68	0,79	0,87	1,18	1,36	
1,8	0,64	0,75	0,82	1,11	1,29	
1,9	0,60	0,71	0,78	1,05	1,22	
2	0,57	0,67	0,74	1,00	1,16	
2,1	0,55	0,64	0,70	0,95	1,10	
2,2	0,52	0,61	0,67	0,91	1,05	
2,3	0,50	0,59	0,64	0,87	1,01	
2,4	0,48	0,56	0,62	0,83	0,97	
2,5	0,46	0,54	0,59	0,80	0,93	

Table 1. Indexed EOA by prosthesis sizes. Data from the literature (Blais et al., 2003).

3.1 2D echocardiography

The valve should be carefully imaged in 2D (presence of calcification, thrombus, leaflets motion). This can be difficult due to the artifacts created by the valve itself and due to the sometimes calcified aorta. Cardiac chambers have to be evaluated with a specific attention to the left ventricle (LV). Indeed LV mass, thickness, systolic and diastolic function need to be assessed. The aortic root and ascending aorta have to be measured as well as the left ventricle outflow (LVO) tract. This measure is important because it is used in the EOA measurement. It should be measured in parasternal long axis view or in a modified lower parasternal location to avoid the artifacts of the prosthesis. In EOA evaluation, artifacts induced by the prosthesis structure are the most frequent source of error.

3.2 Doppler echocardiography

The second part of the study is Doppler echocardiography. Several items need to be determined in order to rule out or diagnosed PPM:

1. Peak velocity, gradient and Velocity Time Integral (VTI) of the jet;
2. Effective Orifice Area;
3. Doppler Velocity Index;
4. Evaluation of the importance of pressure recovery phenomenon.

3.2.1 Peak velocity, gradient and VTI

The velocity resemble those of mild native aortic valve stenosis with a maximal velocity usually >2m/s. The shape of the velocity contour is triangular with occurrence of the maximal velocity in early systole. A different pattern of the flow velocity indicates the presence of valve dysfunction. A higher gradient than 3m/sec should prompt further investigations.

The VTI is the contour of the velocity through the valve and is a qualitative but valuable index. It is difficult as previously mentioned, to differentiate high flow status from obstruction from mismatch. Other indices are than used.

3.2.2 Effective orifice area

The aortic EOA is derived with the stroke volume at the LVO, according to the continuity equation. This equation shows that in a closed hydraulic system flow is the same at different points in the system:

$$EOA_{PrAV} = CSA_{LVO} x VTI_{LVO} / VTI_{PrAV} \quad (2)$$

CSA_{LVO} is the cross sectional area of the outflow tract just underneath the valve from the parastenal long axis view, assuming a circular geometry. Attention should be given to the measure. An error will be amplified by the fact that the radius derived of this measure is used in square.

The VTI_{LVO} is the VTI proximal to the valve using pulsed wave Doppler. The sample should be located 0,5 to 1 cm below the sewing ring to avoid subvalvular acceleration.

The VTI_{PrAV} is the VTI across the valve (PrVA: Prosthetic Aortic Valve) using continuous wave Doppler.

The calculated EOA is dependant of the valve size and should therefore be compared to the effective EOA available from in vivo measurements for each type and size of valve also called projected EOA.

If calculated EOA is different of 1DSA of the EOA, it is suggestive of dysfunction of the prosthesis.

3.2.3 Doppler velocity index

The Doppler Velocity Index (DVI) is the ratio of velocity proximal to the valve (V_{LVO}) and in the valve (V_{PrAV}). It is independent of the size of the LVO and the valve. It can be approximated by the ratio of the respective peak velocities.

$$DVI = V_{LVO} / V_{PrAV} \quad (3)$$

DVI is always less than one because flow always accelerates through the valve. If it is < 0,25 it highly suggestive of significant valve obstruction.

3.2.4 The pressure recovery phenomenon

The pressure recovery phenomenon should also be evaluated. The Bernouilli equation implies that conversion of pressure to velocity is reversible. When blood flows across a stenotic orifice, velocity rises and pressure drops with the lowest pressure and highest velocity at the narrowest portion of the jet. When flow widens, flow velocity diminishes and pressure increases. This is known as pressure recovery. It is always incomplete

because of energy loss due to viscosity and turbulences. The amount of energy lost varies with the shape and size of the conduit, and potentially reflect the severity of the stenosis (Garcia et al., 2000). The energy lost coefficient (ELC) can be quantified by the following equation:

$$ELC = EOA \times A_A / A_A - EOA \quad (4)$$

In this equation, A_A is the aortic cross-sectional area.

Pressure recovery can occur in 2 regions: downstream the valve and in the valve. Downstream the valve there is an inverse relationship between the size of aortic root and the amount of pressure recovery. The importance of the phenomenon is generally small except in aorta smaller than 3 cm where the gradient across the valve can be overestimated (Baumgartner et al., 1992, Baumgartner et al., 1999). Within the valve, in some cases (typically in bileaflet mechanical valves), due the specific design of the valve, this phenomenon occurs. The smaller orifice located centrally between the 2 leaflets may give rise to a high velocity jet corresponding in localized pressure drops that recovers one the central flow reunites with lateral flows. This high gradient can be interpreted and lead to overestimation of the gradient across the valve and underestimation of the EOA (Baumgartner et al., 1992). This is more frequent in smaller valves. Usually it is not a problem because normal gradients expected through each valve exist as for the EOA, and are reported in the literature (Zoghbi et al., 2009).

With all these data, PPM can be diagnosed. Some very clear algorithms exist in the literature guiding the clinician in his search for PPM (Pibarot and Dumesnil, 2006, Dumesnil and Pibarot, 2011, Zoghbi et al., 2009). Based on these observations, we here present in Figure 3 maybe the most accurate algorithm, used in our unit, from the Dumesnil and Pibarot observations (Dumesnil and Pibarot, 2011).

To summarise this algorithm and concentrate on mismatch, we could resume the sequence to infirm or confirm mismatch. If a high gradient is reported, calculation of the EOA should be compared to the projected EOA. If it is similar, the EOA should then be indexed to BSA. We can than grade the severity of mismatch with cut off points of 0.85 cm2/m2 for moderate mismatch and 0,65 cm2/m2 for severe mismatch bearing in mind the pressure recovery phenomenon for small aorta.

Of course one should bear in mind that PPM and prosthesis dysfunction can coexist and that evaluation can still be challenging. Other tests can help differentiating these conditions:

- Cinefluoroscopy by imaging the motion of the leaflets in mechanical valve;
- Transesophagial echocardiography to have better images of the valve including thrombus, endocarditis and leaflets;
- Computerized tomography to image pannus, calcifications and motion of the leaflets. Anatomic orifice area can be determined by CT. It is different than EOA, being too optimistic and cannot replace EOA;
- Exercise testing can be useful. Some patients are symptomatic but echocardiography is equivocal at rest. The presence of PPM or dysfunction of the valve is associated with marked increase in gradients and pulmonary artery pressure on exercise test. Although precise cut points are not available it is likely that a rise in mean gradient >15 mmHg is significant as for native valves (Pibarot et al., 1999). Stress test can be particularly helpful in elderly patients who may claim to be asymptomatic by self limitation.

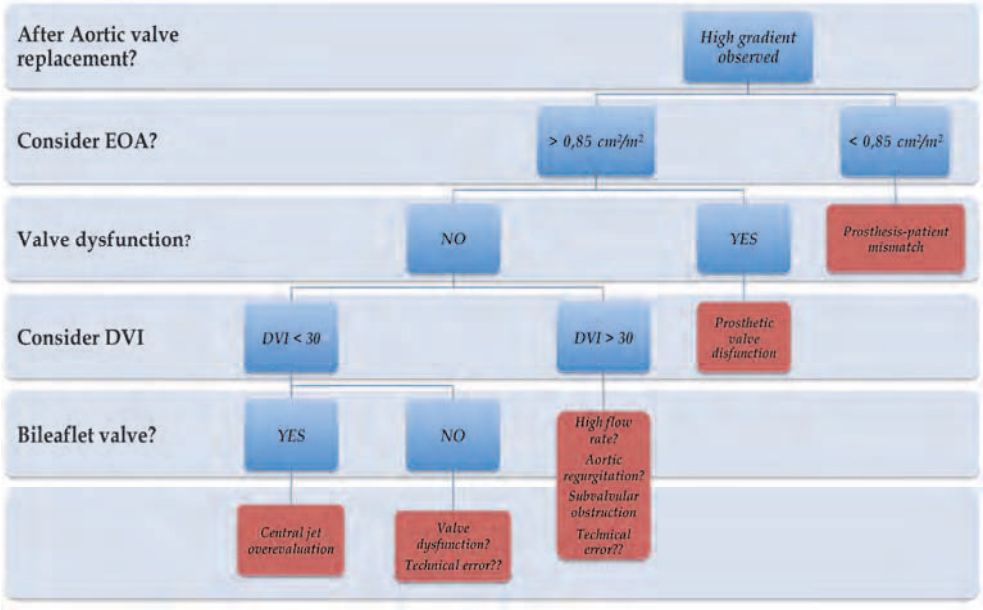


Fig. 3. Decisional algorithm to identify the origin of a abnormally hight transvalvular gradient.

4. Prediction of PPM

As previously said PPM can be estimated or predicted by using the projected EOA available for each valve type and size.

The predicted EOA measures coming from in vivo studies are well correlated with postoperative gradients and clinical outcomes (Pibarot and Dumesnil, 2006, Blackstone et al., 2003, Dumesnil and Pibarot, 2006, Koch et al., 2005).

At this stage it is important to point out that the indexed EOA derived from in vivo postoperative measures is the only parameter valid to predict PPM and postoperative gradients (Dumesnil and Pibarot, 2011, Zoghbi et al., 2009). It is thus the only one to be used.

The indexed geometric orifice area (GOA) a static manufacturing measure based on ex vivo measurements is considerably different than the iEOA. The way it is measured varies from one type of prosthesis to the other, it always overestimates the EOA being too optimistic. For similar values on indexed GOA, peak and mean gradients can double between pericardial valves and homograft's (Koch et al., 2005).

The same issue is raised by the EOA measured in vitro by manufacturers. It is also always too optimistic and overestimates the EOA derived from in vivo measurements.

Both GOA and in vitro indexes correlate poorly with postoperative gradients. Within the literature some authors are still using GOA and manufacturers data. This is one of the reasons why some detrimental effects of PPM remain partly controversial till today.

Using the indexed in vivo EOA, PPM in not infrequent. Prevalence of moderate PPM varies in the literature from 20 to 70% and severe PPM prevalence is estimated between 2

to 11% (Pibarot and Dumesnil, 2000, Blais et al., 2003, Milano et al., 2002, Tasca et al., 2005).
The PPM prediction at the time of surgery is a key issue. Indeed anticipated it can be avoided. Amongst all the risk factor of mortality in AVR, this is the only factor we can avoid.

5. Clinical implications

PPM has various adverse clinical effects. As for the native aortic valve stenosis, clinical impact of PPM increases proportionally with its severity. The consequences of PPM on clinical status depend both on severity of the mismatch and on patient characteristics. Numerous studies report PPM as a risk factor for postoperative mortality and morbidity.
As previously described PPM is not rarely encountered (prevalence of moderate PPM 20 to 70%, severe PPM 2 to 11%). It is noticeable that the frequency of severe PPM has decreased over the last couple of years due to the awareness of its detrimental effects, thanks to the useful prevention strategies at the time of surgery and thanks to the new generations of prosthetic valves with more favorable haemodynamics.
There is now a strong body of evidence that PPM has an impact on functional class, regression of left ventricular hypertrophy, left ventricular function, coronary flow reserve, rate of valve degeneration and more importantly, mortality (Tasca et al., 2005, Flameng et al., 2010). Over time it has become clear that the impact of PPM depends greatly on the clinical condition of the patients.

5.1 Mortality

Considering the most important outcome, mortality, we have to distinguish early and late mortality. The impact of PPM on early mortality is more important than on late mortality given that the left ventricle is more vulnerable during early postoperative period to any hemodynamic burden imposed. Early mortality is significantly increased if PPM is severe or if moderate PPM is associated with left ventricular dysfunction (left ventricular ejection fraction (LVEF) < 40%) (Blais et al., 2003, Pibarot and Dumesnil, 2006, Urso et al., 2009). Blais et al showed in a study in 1265 patients undergoing AVR that mortality was 5% in patients with moderate PPM and normal left ventricular function, was 16% in patients with moderate PPM and depressed left ventricular function and was 67% if PPM was severe and combined with left ventricular dysfunction (Blais et al., 2003).
There are still controversies regarding late mortality. Several studies reported that PPM is an independent factor of mortality after AVR (Blais et al., 2003, Tasca et al., 2006), other concluded that PPM did not affect mortality (Blackstone et al., 2003, Koch et al., 2005). The different conclusions may result from the heterogenous populations that have been studied and the way to predict PPM (GOA or in vitro EOA). Indeed PPM clinical relevance varies with the patient characteristics. Mohty et al summarizes the impacts of PPM on late mortality in different subgroups of patients: moderate PPM increases mortality if left ventricular function is reduced (LVEF <50%) but not with normal ventricular function. Severe PPM increases mortality in patients younger than 70 years old, with a reduced left ventricular function or BMI < 30 Kg/m^2 (Mohty et al., 2009).

Blackstone and Howell have used different parameters to define mismatch (GOA, in vitro EOA). Blakstone in a very large study showed no effect of PPM on mortality but population characteristic is not well defined (Blackstone et al., 2003).
Some other studies demonstrated that PPM has no impact on mortality in the elderly (Monin et al., 2007). The relationship between age and PPM can be explained by the cardiac index requirement varying with age. Indeed younger people are more active and have a higher basal metabolic state compared with older patients. Another potential explanation is the longer exposure to PPM for the younger patients. Finally if we consider patients implanted with a bioprosthetic valve, the deterioration of the valve is likely to appear faster in younger people who are more prone to calcifications. These patients will have less "EOA reserve" if PPM is present. Higher gradient and stenosis will tend to develop faster with the combination of degeneration and PPM (Flameng et al., 2010).
Interaction between PPM and BMI should be emphasized. PPM impact on patients with a BMI< 30 kg/m2 reflects more probably that EOA should not be indexed with BSA but with a fat-free index in these obese patients. iEOA overestimates the prevalence and severity of PPM in this subgroup of patients.
Logically patients with reduced left ventricular function will not tolerate the increased burden secondary to PPM regardless of its severity (Blais et al., 2003, Kulik et al., 2006, Ruel et al., 2006).

5.2 Left ventricular hypertrophy, function and coronary flow reserve

PPM has also an impact on the left ventricle. Controversies remain about the role of PPM on the regression of the left ventricular hypertrophy. After relief of the stenosis, reduction of the left ventricular hypertrophy will occur whatsoever and the impact of the PPM on the degree of regression of left ventricular mass remains unknown. It is know recognized that the presence of systemic hypertension, metabolic syndrome, decreased vascular compliance results in an increase of the afterload of the ventricle that will not be relieved after surgery. The degree of muscular hypertrophy and interstitial fibrosis (which is not reversible) does not depend only on residual gradient: left ventricular hypertrophy regression is multifactorial and not only related to PPM.
As described earlier PPM has a significant impact on mortality if present with concomitant left ventricular dysfunction. The improvement of LV function is correlated with the increased EOA after surgery. This has been shown for surgery but also for percutaneously implanted aortic valve. Indeed recently LV function has been compared in patients surgically implanted and percutaneously implanted. LV function improved faster after transcatheter implantation mainly to the larger iEOA observed after transcatheter implantation leading to smaller gradient and better haemodynamic (Jilaihawi et al., 2010, Clavel et al., 2009).
One of the main goals of aortic valve replacement is restoration of the myocardial reserve. A persistent significant gradient across the valve affects coronary reserve recovery. Independently of the regression of the left ventricular mass, postoperative coronary vasodilatory reserve varies proportionally to the iEOA and thus to PPM (Rajappan et al., 2003).

5.3 Miscelaneous

PPM is also associated with a number of other adverse outcomes with variable clinical importance: reduced quality of life, reduced exercise capacity (Bleiziffer et al., 2008), more important residual mitral regurgitation (Unger et al., 2010), the risk of early degeneration of bioprosthetic valve with stenotic lesions (Flameng et al., 2010) and increased risk of hemorrhagic complication due to the acquired abnormalities of the Von Willebrand factor (Vincentelli et al., 2003).

6. Prevention of patient-prosthesis mismatch

Aortic valve replacement has become a simple and safe procedure through the time. Nowadays, this procedure can be accomplished with a low mortality and morbidity rate. However, there is no zero risk aortic valve replacement surgery nowadays. In this particular setting, it appears that patient-prosthesis mismatch emerges as a prominent risk factor for postoperative mortality and morbidity, and one of the few that can be acted upon. A strategy of prevention of PPM is thus of the upmost importance. Severe PPM (EOA<0,65 cm^2/m^2) must be avoided in all patients. Moderate PPM only justifies an aggressive prevention strategy in the most susceptible patients:

1. Patients younger than 65 years of age;
2. Athletes;
3. Patients with preexistent systolic dysfunction of the left ventricle with left ventricular ejection fraction less than 40%;
4. Patients with severe left ventricular muscle hypertrophy.

To the contrary, moderate PPM could be neglected in low exposed patients including:

1. Obese patient where the cardiac output is not directly proportional to the BSA;
2. Older patients.

The EOA of the prosthesis to be implanted must thus be more than 0,85 cm^2/m^2 (compilation of the body surface area of the patients is prerequired).

6.1 The choice of the prosthesis

Compared to a bioprosthesis, mechanical valves present a better EOA at the same prosthesis size. Intraoperatively, it is important to consider the EOA of the prosthesis that can fit the aortic root. A type of prosthesis with the largest EOA for a given nominal diameter should be chosen. Not all available models of prostheses for a given aortic root configuration have the same size: a size 23 model of one manufacturer may fit the same aortic root configuration as a size 21 model of another. Stentless bioprostheses claim better hemodynamic parameters than their stented counterparts. Also, recent generation bileaflet mechanical prostheses offer better EOA for a given nominal external diameter. On Table 2 and Table 3, the EOA and iEOA of o bioprosthesis and a mechanical valve are reported. We can see that mechanical valves presents better hemodynamic parameters than bioprosthesis.

6.2 The surgical technique

Surgical implantation technique also allows implantation of a larger prosthesis. The simplest way to achieve this goal is to choose a supraannular rather than annular technique (Fig. 4).

	Carpentier-Edwards Perimount®					
Prosthesis size	**19**	**21**	**23**	**25**	**27**	**29**
EOA (cm^2/m^2)	1,1	1,3	1,5	1,8	1,8	
BSA (m^2)						
1	1,10	1,30	1,50	1,80	1,80	
1,1	1,00	1,18	1,36	1,64	1,64	
1,2	0,92	1,08	1,25	1,50	1,50	
1,3	0,85	1,00	1,15	1,38	1,38	
1,4	0,79	93,00	1,07	1,38	1,38	
1,5	0,73	0,87	1,00	1,20	1,20	
1,6	0,69	0,81	0,94	1,12	1,12	
1,7	0,65	0,76	0,88	1,06	1,06	
1,8	0,61	0,72	0,83	1,00	1,00	
1,9	0,58	0,68	0,79	0,95	0,95	
2	0,55	0,65	0,75	0,90	0,90	
2,1	0,52	0,62	0,71	0,86	0,86	
2,2	0,50	0,59	0,68	0,82	0,82	
2,3	0,48	0,56	0,65	0,78	0,78	
2,4	0,46	0,54	0,62	0,75	0,75	
2,5	0,44	0,52	0,60	0,72	0,72	

Table 2. Eoa and iEOA of a performant bioprosthesis (Blais et al., 2003).

	St Jude Medical Regent®					
Prosthesis size	**19**	**21**	**23**	**25**	**27**	**29**
EOA (cm^2/m^2)	1,5	2	2,4	2,5	3,6	4,8
BSA (m^2)						
1	1,50	2,00	2,40	2,50	3,60	4,80
1,1	1,36	1,82	2,18	2,27	3,27	4,36
1,2	1,25	1,67	2,00	2,08	3,00	4,00
1,3	1,15	1,54	1,85	1,92	2,77	3,69
1,4	1,07	1,43	1,71	1,78	2,57	3,43
1,5	1,00	1,33	1,60	1,67	2,40	3,20
1,6	0,94	1,25	1,50	1,56	2,25	3,00
1,7	0,88	1,18	1,41	1,47	2,12	2,82
1,8	0,83	1,11	1,33	1,39	2,00	2,67
1,9	0,79	1,05	1,26	1,32	1,89	2,53
2	0,75	1,00	1,20	1,25	1,80	2,40
2,1	0,71	0,95	1,14	1,16	1,71	2,29
2,2	0,68	0,91	1,09	1,14	1,64	2,18
2,3	0,65	0,87	1,04	1,09	1,56	2,09
2,4	0,62	0,83	1,00	1,04	1,50	2,00
2,5	0,60	0,80	0,96	1,00	1,44	1,92

Table 3. Eoa and iEOA of a performant bileaflet mechanical valve (Blais et al., 2003).

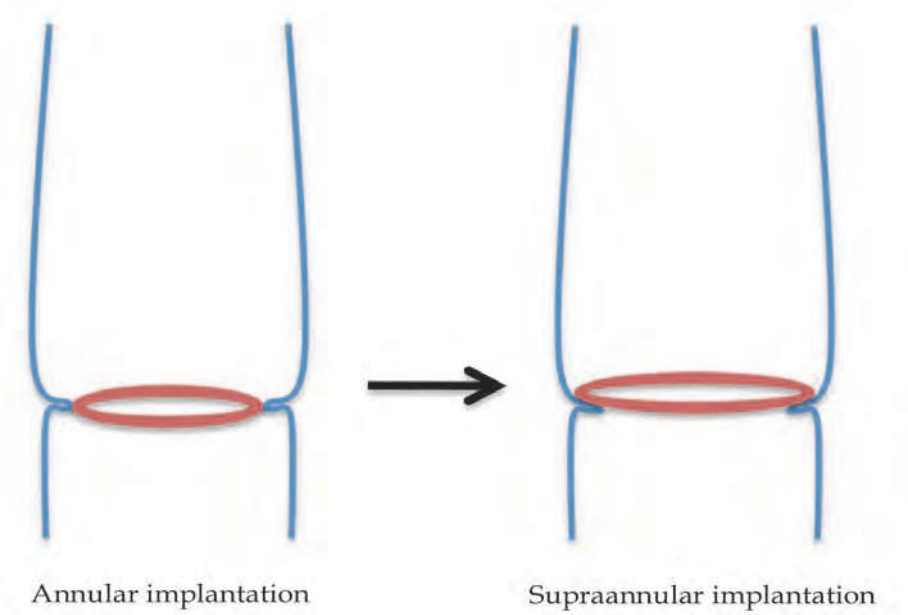


Fig. 4. Illustration of the benefit to implant the prosthesis in a supraannular technique.

A more aggressive, and more potentially beneficial technique, consist to associate aortic valve replacement and enlargement of the aortic root and annulus. The Manouguian technique inserts a widening patch in the left-non coronary commissure and allows implantation of a prosthesis one to two sizes larger (Manouguian and Seybold-Epting, 1979). Unfortunately, the presence of important aortic root calcifications limits the application of this technique. Briefly, an oblique aortotomy is performed and aimed to descend at the left-non coronary sinus, through the aorto-mitral transition (Figure 5).

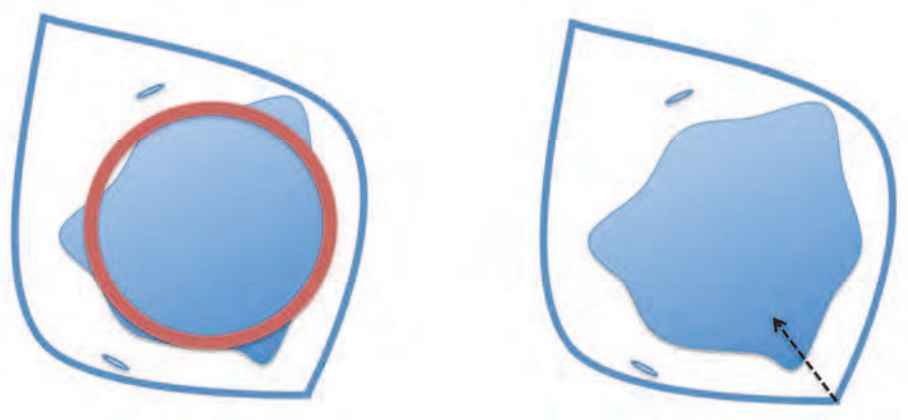


Fig. 5. Illustration of the transannular incision realized in the Manouguian technique.

A widening patch is then implanted to close this incision (Figure 6) and the prosthesis is thereafter sutured to the aortic annulus and to the reconstructive patch (Figure 7).

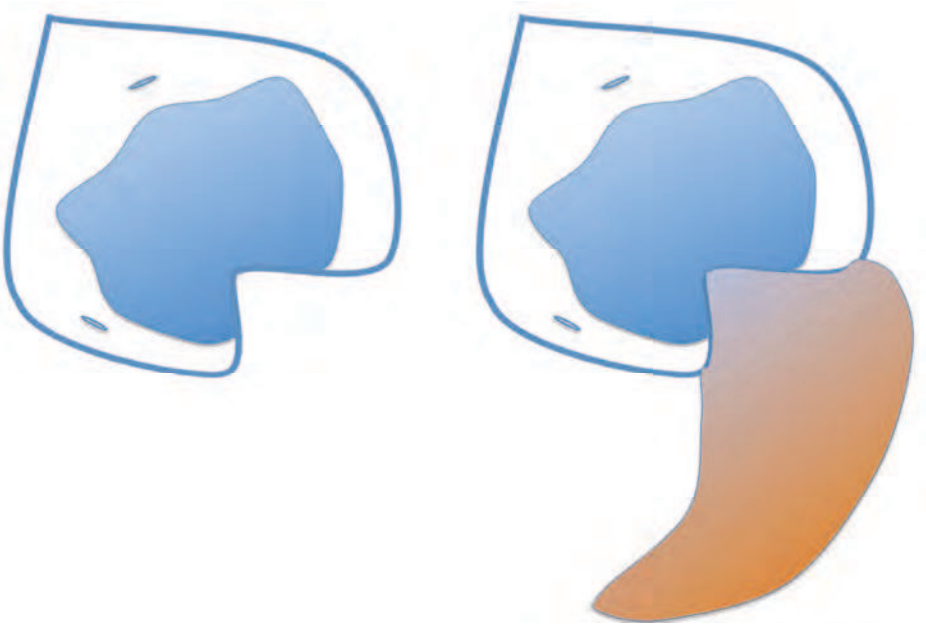


Fig. 6. Illustration of the enlarging patch reconstruction of the incision.

The Figure 7 shows the significant oversizing allowed by the technique compare to the initial prosthesis size matched to the initial annulus. The aortotomy is closed with the enlargement patch after the implantation of the aortic valve prosthesis.
During this procedure, the incision in the aortoventricular membrane must be carefully performed and not extended to deep in the mitral annulus, the anterior mitral leaflet and the left atrium. The reconstruction patch may in this particular setting interfere with the hinging portion of the anterior mitral leaflet.

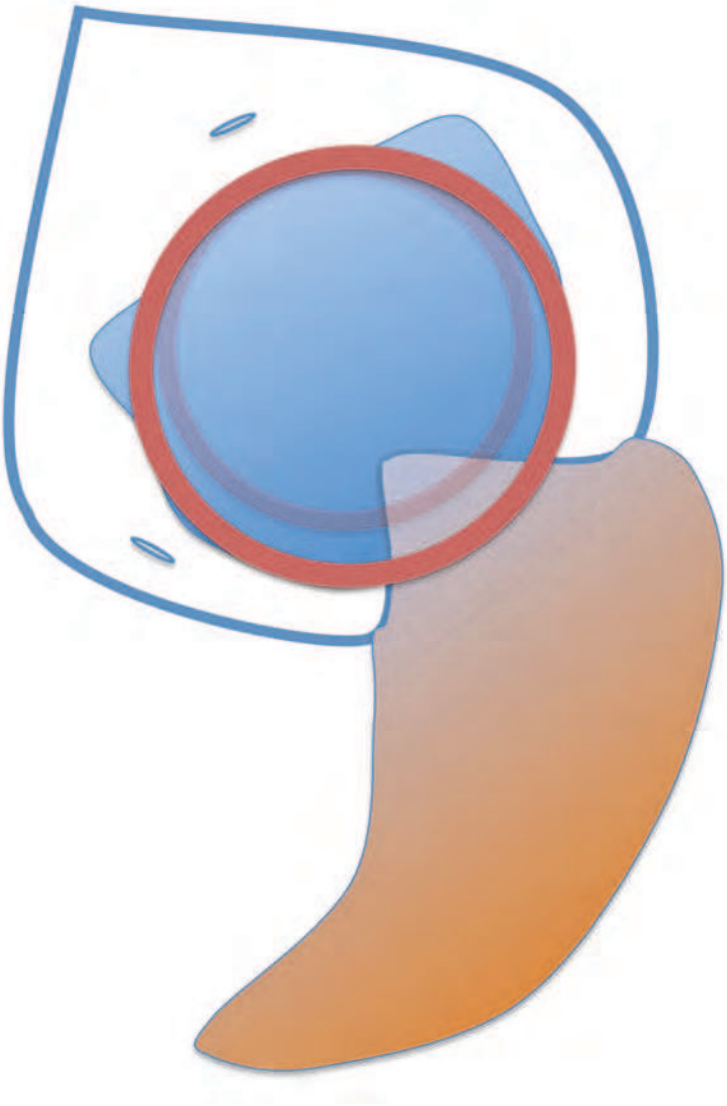


Fig. 7. Illustration of the realized oversizing allowed by the Manouguian technique.

The overall surgical strategy that we proposed is illustrated in the Figure 8. The first possibility to match the implanted valve to the patient is to realize a supraannular implantation. If this surgical technique is insufficient, we should consider an alternative second choice in the prosthesis strategy, ie a bileaflet new generation of mechanical prosthesis (an old patient with atrial fibrillation…). The last possibility is to realize a Manouguian enlargement of the aortic annulus, if possible.

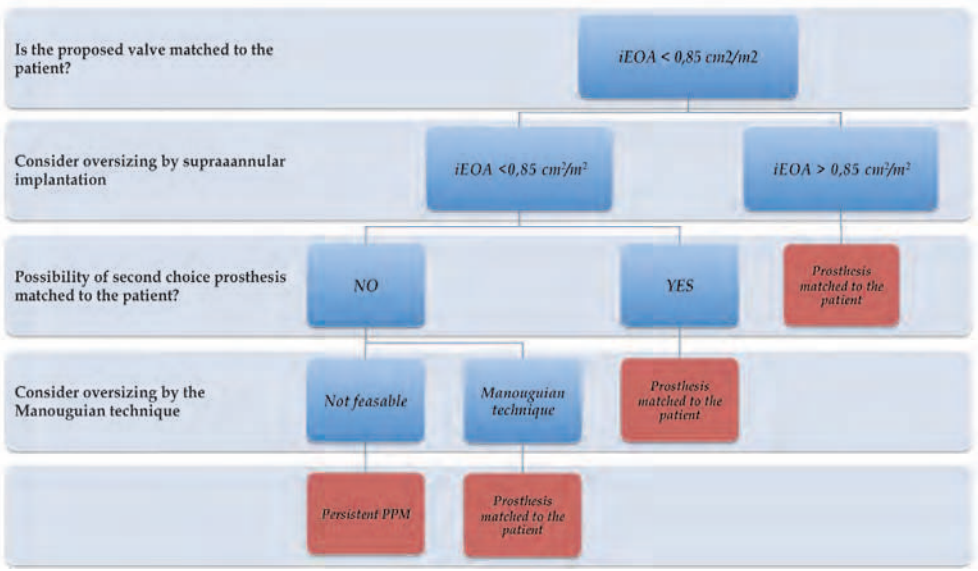


Fig. 8. Surgical strategy to avoid a patient-prosthesis mismatch.

It should be mentioned that some patients present with a hypoplastic aorto-ventricular junction. Most of them are referred to surgery during childhood. In such situation, a radical enlargement of both the aortic valve annulus and the left ventricular outflow tract should be performed. The anterior technique, first described by Konno in 1974 (Konno et al., 1975), consists in a wide opening of the aortic valve annulus and of the interventricular septum with an oblique incision at 5mm to the left side of the right coronary ostium. This technique is far more complex than the Manouguian technique and may lead to severe complications, particularly an iatrogenic ventricular septal defect or atrioventricular block.

7. Conclusions

Patient-prosthesis mismatch is probably the most frequently encountered hemodynamic problem after aortic valve replacement. All the patients are not equally exposed to this problem and clinical consequences may be variable from one to another. However, the consequences may lead to an increased mortality and worsen symptomatic improvements after the aortic valve replacement. Though, prevention of this mechanism is the key point in symptomatic patients that should be operated on. Indexed EOA of the implanted valve should be systematically calculated from reference values of the EOA of the prosthesis, and surgical strategies adapted to allow implantation of prosthesis with iEOA matched to the patient.

8. References

Baumgartner, H., Khan, S., DeRobertis, M., Czer, L. & Maurer, G. 1992. Effect of prosthetic aortic valve design on the Doppler-catheter gradient correlation: an in vitro study of normal St. Jude, Medtronic-Hall, Starr-Edwards and Hancock valves. *J Am Coll Cardiol,* 19, 324-32.

Baumgartner, H., Stefenelli, T., Niederberger, J., Schima, H. & Maurer, G. 1999. "Overestimation" of catheter gradients by Doppler ultrasound in patients with aortic stenosis: a predictable manifestation of pressure recovery. *J Am Coll Cardiol,* 33, 1655-61.

Blackstone, E. H., Cosgrove, D. M., Jamieson, W. R., Birkmeyer, N. J., Lemmer, J. H., Jr., Miller, D. C., Butchart, E. G., Rizzoli, G., Yacoub, M. & Chai, A. 2003. Prosthesis size and long-term survival after aortic valve replacement. *J Thorac Cardiovasc Surg,* 126, 783-96.

Blais, C., Dumesnil, J. G., Baillot, R., Simard, S., Doyle, D. & Pibarot, P. 2003. Impact of valve prosthesis-patient mismatch on short-term mortality after aortic valve replacement. *Circulation,* 108, 983-8.

Bleiziffer, S., Eichinger, W. B., Hettich, I., Guenzinger, R., Ruzicka, D., Bauernschmitt, R. & Lange, R. 2007. Prediction of valve prosthesis-patient mismatch prior to aortic valve replacement: which is the best method? *Heart,* 93, 615-20.

Bleiziffer, S., Eichinger, W. B., Hettich, I., Ruzicka, D., Wottke, M., Bauernschmitt, R. & Lange, R. 2008. Impact of patient-prosthesis mismatch on exercise capacity in patients after bioprosthetic aortic valve replacement. *Heart,* 94, 637-41.

Clavel, M. A., Webb, J. G., Pibarot, P., Altwegg, L., Dumont, E., Thompson, C., De Larochelliere, R., Doyle, D., Masson, J. B., Bergeron, S., Bertrand, O. F. & Rodes-Cabau, J. 2009. Comparison of the hemodynamic performance of percutaneous and surgical bioprostheses for the treatment of severe aortic stenosis. *J Am Coll Cardiol,* 53, 1883-91.

Dumesnil, J. G. & Pibarot, P. 2006. Prosthesis-patient mismatch and clinical outcomes: the evidence continues to accumulate. *J Thorac Cardiovasc Surg,* 131, 952-5.

Dumesnil, J. G. & Pibarot, P. 2011. Prosthesis-Patient Mismatch: An Update. *Curr Cardiol Rep*.

Flameng, W., Herregods, M. C., Vercalsteren, M., Herijgers, P., Bogaerts, K. & Meuris, B. 2010. Prosthesis-patient mismatch predicts structural valve degeneration in bioprosthetic heart valves. *Circulation,* 121, 2123-9.

Garcia, D., Pibarot, P., Dumesnil, J. G., Sakr, F. & Durand, L. G. 2000. Assessment of aortic valve stenosis severity: A new index based on the energy loss concept. *Circulation,* 101, 765-71.

Jilaihawi, H., Chin, D., Spyt, T., Jeilan, M., Vasa-Nicotera, M., Bence, J., Logtens, E. & Kovac, J. 2010. Prosthesis-patient mismatch after transcatheter aortic valve implantation with the Medtronic-Corevalve bioprosthesis. *Eur Heart J,* 31, 857-64.

Koch, C. G., Khandwala, F., Estafanous, F. G., Loop, F. D. & Blackstone, E. H. 2005. Impact of prosthesis-patient size on functional recovery after aortic valve replacement. *Circulation,* 111, 3221-9.

Konno, S., Imai, Y., Iida, Y., Nakajima, M. & Tatsuno, K. 1975. A new method for prosthetic valve replacement in congenital aortic stenosis associated with hypoplasia of the aortic valve ring. *J Thorac Cardiovasc Surg,* 70, 909-17.

Kulik, A., Burwash, I. G., Kapila, V., Mesana, T. G. & Ruel, M. 2006. Long-term outcomes after valve replacement for low-gradient aortic stenosis: impact of prosthesis-patient mismatch. *Circulation,* 114, I553-8.

Manouguian, S. & Seybold-Epting, W. 1979. Patch enlargement of the aortic valve ring by extending the aortic incision into the anterior mitral leaflet. New operative technique. *J Thorac Cardiovasc Surg,* 78, 402-12.

Milano, A. D., De Carlo, M., Mecozzi, G., D'Alfonso, A., Scioti, G., Nardi, C. & Bortolotti, U. 2002. Clinical outcome in patients with 19-mm and 21-mm St. Jude aortic prostheses: comparison at long-term follow-up. *Ann Thorac Surg,* 73, 37-43.

Mohty, D., Dumesnil, J. G., Echahidi, N., Mathieu, P., Dagenais, F., Voisine, P. & Pibarot, P. 2009. 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,* 53, 39-47.

Monin, J. L., Monchi, M., Kirsch, M. E., Petit-Eisenmann, H., Baleynaud, S., Chauvel, C., Metz, D., Adams, C., Quere, J. P., Gueret, P. & Tribouilloy, C. 2007. Low-gradient aortic stenosis: impact of prosthesis-patient mismatch on survival. *Eur Heart J,* 28, 2620-6.

Pibarot, P. & Dumesnil, J. G. 2000. Hemodynamic and clinical impact of prosthesis-patient mismatch in the aortic valve position and its prevention. *J Am Coll Cardiol,* 36, 1131-41.

Pibarot, P. & Dumesnil, J. G. 2006. Prosthesis-patient mismatch: definition, clinical impact, and prevention. *Heart,* 92, 1022-9.

Pibarot, P., Dumesnil, J. G., Briand, M., Laforest, I. & Cartier, P. 2000. Hemodynamic performance during maximum exercise in adult patients with the ross operation and comparison with normal controls and patients with aortic bioprostheses. *Am J Cardiol,* 86, 982-8.

Pibarot, P., Dumesnil, J. G., Jobin, J., Cartier, P., Honos, G. & Durand, L. G. 1999. Hemodynamic and physical performance during maximal exercise in patients with an aortic bioprosthetic valve: comparison of stentless versus stented bioprostheses. *J Am Coll Cardiol,* 34, 1609-17.

Rahimtoola, S. H. 1978. The problem of valve prosthesis-patient mismatch. *Circulation,* 58, 20-4.

Rajappan, K., Rimoldi, O. E., Camici, P. G., Bellenger, N. G., Pennell, D. J. & Sheridan, D. J. 2003. Functional changes in coronary microcirculation after valve replacement in patients with aortic stenosis. *Circulation,* 107, 3170-5.

Ruel, M., Al-Faleh, H., Kulik, A., Chan, K. L., Mesana, T. G. & Burwash, I. G. 2006. 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,* 131, 1036-44.

Ruel, M., Rubens, F. D., Masters, R. G., Pipe, A. L., Bedard, P., Hendry, P. J., Lam, B. K., Burwash, I. G., Goldstein, W. G., Brais, M. P., Keon, W. J. & Mesana, T. G. 2004. Late incidence and predictors of persistent or recurrent heart failure in patients with aortic prosthetic valves. *J Thorac Cardiovasc Surg,* 127, 149-59.

Tasca, G., Brunelli, F., Cirillo, M., DallaTomba, M., Mhagna, Z., Troise, G. & Quaini, E. 2005. Impact of valve prosthesis-patient mismatch on left ventricular mass regression following aortic valve replacement. *Ann Thorac Surg,* 79, 505-10.

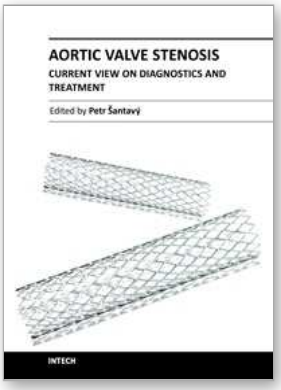
Tasca, G., Mhagna, Z., Perotti, S., Centurini, P. B., Sabatini, T., Amaducci, A., Brunelli, F., Cirillo, M., Dalla Tomba, M., Quaini, E., Troise, G. & Pibarot, P. 2006. Impact of prosthesis-patient mismatch on cardiac events and midterm mortality after aortic valve replacement in patients with pure aortic stenosis. *Circulation,* 113, 570-6.

Unger, P., Dedobbeleer, C., Van Camp, G., Plein, D., Cosyns, B. & Lancellotti, P. 2010. Mitral regurgitation in patients with aortic stenosis undergoing valve replacement. *Heart,* 96, 9-14.

Urso, S., Sadaba, R. & Aldamiz-Echevarria, G. 2009. 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,* 9, 510-8.

Vincentelli, A., Susen, S., Le Tourneau, T., Six, I., Fabre, O., Juthier, F., Bauters, A., Decoene, C., Goudemand, J., Prat, A. & Jude, B. 2003. Acquired von Willebrand syndrome in aortic stenosis. *N Engl J Med,* 349, 343-9.

Zoghbi, W. A., Chambers, J. B., Dumesnil, J. G., Foster, E., Gottdiener, J. S., Grayburn, P. A., Khandheria, B. K., Levine, R. A., Marx, G. R., Miller, F. A., Jr., Nakatani, S., Quinones, M. A., Rakowski, H., Rodriguez, L. L., Swaminathan, M., Waggoner, A. D., *et al.* 2009. 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,* 22, 975-1014; quiz 1082-4.



Aortic Valve Stenosis - Current View on Diagnostics and Treatment
Edited by Dr. Petr Santavy

ISBN 978-953-307-628-7
Hard cover, 146 pages
Publisher InTech
Published online 22, September, 2011
Published in print edition September, 2011

Currently, aortic stenosis is the most frequent heart valve disease in developed countries and its prevalence increases with the aging of the population. Affecting 3-5 percent of persons older than 65 years of age, it makes a large personal and economical impact. The increasing number of elderly patients with aortic stenosis brings advances in all medical specialties dealing with this clinical entity. Patients previously considered too old or ill are now indicated for aortic valve replacement procedures. This book tries to cover current issues of aortic valve stenosis management with stress on new trends in diagnostics and treatment.

How to reference
In order to correctly reference this scholarly work, feel free to copy and paste the following:

Pierre Wauthy and Sophie G. Malekzadeh-Milani (2011). Patient-Prosthesis Mismatch After Aortic Valve Replacement, Aortic Valve Stenosis - Current View on Diagnostics and Treatment, Dr. Petr Santavy (Ed.), ISBN: 978-953-307-628-7, InTech, Available from: http://www.intechopen.com/books/aortic-valve-stenosis-current-view-on-diagnostics-and-treatment/patient-prosthesis-mismatch-after-aortic-valve-replacement

INTECH
open science | open minds

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821