Hemodynamic and Clinical Impact of Prosthesis–Patient Mismatch in the Aortic Valve Position and Its Prevention

Philippe Pibarot, DVM, PhD, FACC, Jean G. Dumesnil, MD, FRCP(C), FACC
Sainte-Foy, Quebec, Canada

Prosthesis–patient mismatch is present when the effective orifice area of the inserted prosthetic valve is less than that of a normal human valve. This is a frequent problem in patients undergoing aortic valve replacement, and its main hemodynamic consequence is the generation of high transvalvular gradients through normally functioning prosthetic valves. The purposes of this report are to present an update on the concept of aortic prosthesis–patient mismatch and to review the present knowledge with regard to its impact on hemodynamic status, functional capacity, morbidity and mortality. Also, we propose a simple approach for the prevention and clinical management of this phenomenon because it can be largely avoided if certain simple factors are taken into consideration before the operation. (J Am Coll Cardiol 2000;36:1131–41) © 2000 by the American College of Cardiology

The problem of prosthesis–patient mismatch was first described by Rahimtoola in 1978 as follows: “Mismatch can be considered to be present when the effective prosthetic valve area, after insertion into the patient, is less than that of a normal human valve” (1). With the advent of Doppler echocardiography has come the observation that normally functioning valve prostheses can have relatively high postoperative transvalvular gradients, and that, in most instances, these high gradients are essentially due to a phenomenon akin to prosthesis–patient mismatch (2–6). Hence, prosthesis–patient mismatch is a more frequent problem than originally believed, and it may have a significant impact on the long-term results of valve surgery. The purposes of this report are to present an update on the concept of aortic prosthesis–patient mismatch and to review the present knowledge with regard to its impact on hemodynamic status, functional capacity, morbidity and mortality. We also propose a simple approach for the prevention and clinical management of this phenomenon because this phenomenon can be largely avoided if certain simple factors are taken into consideration before the operation.

DEFINITION OF PROSTHESIS–PATIENT MISMATCH

Several Doppler echocardiographic studies have shown that most prosthetic valves are at least mildly stenotic and that relatively high transprosthetic gradients can be observed.
became more widely recognized, the consequences of inserting a smaller prosthesis were largely ignored, and there was no particular effort to insert a larger prosthesis except in rare cases (3). Second, because the prosthesis is inserted within the aorta and has its own structural support, the EOA of the prosthesis is necessarily less than that which a normal native valve would have within the same aorta. Obviously, the support apparatus of stented bioprostheses or of mechanical valves creates a relative obstruction to flow, and it has been shown that the EOA available for blood flow represents only 40% to 70% of the total area occupied by the valve (23–26). Stentless bioprostheses were in part developed to alleviate this problem, and they generally provide a larger valve EOA in relation to the patient’s BSA, as compared with stented bioprostheses (20,27–43). Nonetheless, the EOA of stentless valves remains somewhat smaller than that of the corresponding native valve, because they are usually implanted using techniques requiring insertion of the prosthesis within the patient’s aorta (a cylinder within a cylinder).

Prosthesis–patient mismatch has been recognized by the American Society of Thoracic Surgeons and has been identified as a nonstructural dysfunction (44,45). Indeed, mismatch is a functional hemodynamic abnormality, rather than being due to an intrinsic defect of the prosthesis. Previous studies have demonstrated that mismatch is not necessarily a rare phenomenon. Hence, using a relatively conservative definition (i.e., indexed EOA ≤0.85 cm²/m²), it was observed in up to 52% of patients with a stented aortic bioprosthesis (5,6,46).

**DETERMINANTS OF MISMATCH**

Mismatch has been shown to be more likely to occur in patients with the following characteristics: larger BSA, older age, smaller prosthesis size and valvular stenosis as the predominant lesion before the operation (1,4–6,47). Larger patients are probably predisposed to mismatch because they have high cardiac output requirements, and the pathologic process possibly produced a greater narrowing of their valvular annulus in relation to their body size, as compared with smaller patients. Not surprisingly, the incidence of mismatch also increases with diminishing prosthesis size, and it is widely recognized that patients with a valve size ≤21 mm tend to have much higher gradients (9,14,48–50). Nonetheless, it must be emphasized that severe mismatch can also occur in patients receiving a prosthesis size >21 mm (5,6) and that, ultimately, it is always the relation between prosthesis size and body size, rather than each factor taken separately, that determines the final hemodynamic outcome. Thus, patients who received a smaller prosthesis probably had a smaller aortic annulus with regard to their body size. The fact that mismatch occurs more frequently in patients with stenotic native valves and in older patients is also consistent with this concept because patients with stenotic native valves generally have smaller valvular annuli than those with regurgitant valves (51) and calcific aortic stenosis is by far the most prevalent lesion in older patients undergoing aortic valve replacement.

**HEMODYNAMIC IMPACT OF MISMATCH**

The main consequence of prosthesis–patient mismatch is to generate high transvalvular gradients through normally functioning prosthetic valves. The transvalvular gradient is important to consider because an increased gradient will evidently result in increased LV work, thus jeopardizing the regression of LV hypertrophy. An expression of the potential severity of mismatch is given by the relation showing that the transvalvular gradient increases exponentially with a decrease in indexed EOA and that a small decrease in EOA will result in a relatively large increase in gradient (Fig. 1) (2,4,20). Accordingly, in a recent study (6), the average mean gradient of patients with mismatch, as defined by an indexed EOA ≤0.85 cm²/m², was found to be 22 ± 8 mm Hg, compared with 15 ± 6 mm Hg in patients without mismatch (Fig. 2A). In patients with an indexed EOA ≤0.65 cm²/m², it was 33 ± 2 mm Hg. Moreover, long-term follow-up showed a deterioration of hemodynamic measures only in patients with mismatch. Hence, cardiac index, which was similar in patients with and without mismatch up to three years after the operation, decreased significantly thereafter only in patients with mismatch (−0.54 ± 0.32 vs. −0.17 ± 0.49 liter/min per m²; p = 0.04) (Fig. 2B). Although the deterioration in valve...
EOA was similar in both groups, the mean gradient increased significantly (6.62 mm Hg vs. 1.61 mm Hg; \textit{p} = 0.0008) only in patients with mismatch during follow-up (Fig. 2A). Indeed, given the same absolute decrease in valve EOA, the gradient increases much more in patients with mismatch, given the curvilinear relation between gradients and indexed EOAs (Fig. 1). Not surprisingly, the greatest deteriorations in cardiac index and gradients were seen in the patients with the most severe mismatch (i.e., with an indexed EOA $\leq 0.65$ cm$^2$/m$^2$).

Figure 3A was obtained using data from our laboratory; it shows the relation between transvalvular gradient and indexed EOA 18–68 months after aortic valve replacement in 396 patients with either a stented bioprosthesis, a stentless bioprosthesis, an aortic homograft or a pulmonary autograft. As can be seen, these patients fall on different sections of the same exponential curve. The majority of patients with a stented bioprosthesis have an indexed EOA $\leq 0.85$ cm$^2$/m$^2$ and are therefore on the steep portion of the curve, where gradients are relatively high. In contrast, most patients with a stentless bioprosthesis and almost all patients with an aortic homograft or a pulmonary autograft have a larger indexed EOA and are therefore on the flat portion of the curve, where gradients are relatively low.

Recent studies from this laboratory also show the mean gradient, as well as the increase in mean gradient during maximal exercise, can also be directly related to the indexed EOA at rest ($19,20$). Indeed, in patients with an aortic bioprosthesis, we found a strong inverse relation between the mean gradient during maximal exercise and the indexed EOA at rest (Fig. 3B). Hence, in patients with mismatch, defined as an indexed EOA $\leq 0.85$ cm$^2$/m$^2$, the average mean gradient during exercise was 30 ± 10 mm Hg, which
is close to that observed in patients with mild to moderate aortic stenosis (37 ± 17 mm Hg) (52), whereas in patients without mismatch, the average mean gradient was 10 ± 5 mm Hg. These exercise studies also demonstrated that the EOA of a bioprosthesis has the potential to increase during exercise and that, as a consequence, the observed increase in gradient is substantially less (~25%) than what would have been observed if the EOA had remained constant during exercise (20). Nonetheless, it should be submitted that bioprostheses that have calcified over time or mechanical prostheses are probably less compliant and thus could have a greater increase in gradient during exercise. Indeed, recent studies performed in patients with mechanical valves suggest that the EOA of these patients does not increase during exercise, resulting in a relatively greater increase in gradient (41,53,54). Further studies are necessary to resolve these issues, but these results underline the necessity of evaluating the performance of the prosthesis not only at rest, but also during exercise.

Also, the impact of mismatch may be overestimated in patients with a smaller aortic root owing to the pressure recovery phenomenon. Indeed, recent studies have demonstrated that there is substantially more pressure recovery occurring downstream to native or prosthetic aortic valves in patients with a small aorta as compared with patients with a large aorta (55–58). Hence, given a similar indexed EOA, patients with a smaller aorta will have less energy loss and thus less burden on their LV than those with a larger aorta. We have recently proposed a new index of aortic stenosis severity, taking into account the influence of pressure recovery, but it remains to be validated in larger prospective series of patients with native atherosclerosis as well as with prostheses (58).

**IMPACT ON LV HYPERTROPHY**

A major consequence of high residual pressure gradients is the hindrance or delay of the regression of LV hypertrophy after the operation (59–61). Indeed, the extent of muscle mass regression has been shown to be highly dependent on the type and size of prosthesis used for valve replacement, as well as on their hemodynamic performance (61–63). Hence, Barner et al. (64) demonstrated that regression of LV hypertrophy after aortic valve replacement is better in patients with a prosthesis size >21 mm (~21%) than in patients with a prosthesis size ≤21 mm (~8%), and Nishimura et al. (60) found that the mean wall thickness of the LV was directly related to the pressure gradient across the aortic prosthetic valve. Other studies also demonstrate that aortic valve replacement with a stentless bioprosthesis is associated with a greater decrease in transvalvular gradient and LV wall stress, as well as with more complete regression of LV hypertrophy, compared with stented valves (20,61,65). In a recent study of 1,103 patients with a porcine bioprosthesis, Del Rizzo et al. (66) found a strong relation between the indexed EOA and the extent of LV mass regression. At three years after the operation, the LV mass index had decreased by 23%, on average, in patients whose indexed EOA was >0.8 cm²/m², as compared with 4.5% (p = 0.0001) in patients with an indexed EOA <0.8 cm²/m². In contrast, no difference was noted between the patients with an indexed EOA between 0.8 and 1.0 cm²/m² and those with an indexed EOA >1.0 cm²/m² (~24% vs. ~22%). These results are consistent with the relations shown in Figure 3, where the gradients are more important only when the indexed EOA is <0.8 to 0.9 cm²/m². Thereafter, the curve is relatively flat and the transvalvular gradients are low, regardless of the level of indexed valve EOA, thus resulting in an optimal regression of LV hypertrophy.

In this context, it should be emphasized that LV hypertrophy has long been recognized as an important risk factor and predictor of survival as well as a major determinant of systolic and diastolic function and exercise capacity (67–73). However, a caveat should be made that most studies showing long-term detrimental effects of LV hypertrophy were conducted in patients with hypertensive heart disease. It remains to be determined whether similar consequences are to be expected with respect to the hypertrophy due to valvular disease. Indeed, the hypertrophy associated with hypertension shows an important proportion of interstitial fibrosis in addition to muscle hypertrophy, and it has a neurohormonal component (73). The hypertrophy due to valvular disease could be different and more directly related to an increased hemodynamic burden, in which case it could be more physiologic, show less fibrosis and thus not have the same negative impact on long-term prognosis. Indeed, numerous studies have shown that the physiologic hypertrophy due to exercise is directly related to the increased burden related to the intensity of training and does not carry any long-term negative effects (74–76).

**CLINICAL IMPACT OF MISMATCH**

**Impact on physical capacity.** The postoperative improvement of the patient’s physical capacity is an important objective of valve replacement because it directly influences the patient’s symptomatic status, quality of life and rate of re-employment (77–80). Poor physical capacity is also associated with a higher rate of late mortality after valve replacement (81,82). Fernandez et al. (83) reported that the indexed EOA did not influence the New York Heart Association functional class in patients with a St. Jude bileaflet valve, whereas we (6) have found that the indexed EOA is an independent predictor of functional class in patients with a Medtronic Intact bioprosthetic valve. The discrepancy between the results of these two studies might be due to the fact that the indexed EOAs (range 0.74 to 2.86 cm²/m²) in the former study were larger than those in the latter study (range 0.57 to 1.59 cm²/m²), and few patients in the study by Fernandez et al. (83) had an indexed...
EOA <0.85 cm²/m², which is the generally accepted criteria for prosthesis–patient mismatch.

Conflicting results are also observed when exercise capacity is measured directly. On the one hand, there are some studies (6,83) demonstrating that prosthesis size is an independent predictor of exercise tolerance after aortic valve replacement. Moreover, de Carlo et al. (85) reported that among patients with a 21-mm St. Jude mechanical valve, those with a BSA =1.70 m² had significantly lower exercise tolerance than those with a BSA <1.70 m². Furthermore, the indexed valve EOA was an independent predictor of exercise tolerance variables. On the other hand, recent studies of patients with bioprosthetic valves show that maximal exercise capacity, as estimated by maximal workload, peak oxygen consumption or anaerobic threshold, is similar between patients with an indexed EOA ≤0.85 and >0.85 cm²/m² (19,20). As previously mentioned, the results of this study suggest that these bioprosthetic valves were compliant and these patients had the capacity to substantially increase their EOA during exercise, whereas this may not be the case with mechanical valves; as a result, the gradients measured during exercise are less than expected and probably not high enough to have a significant impact on maximal exercise capacity. Because patients with mismatch often have a small aortic root, it is also possible that some of these patients had substantial pressure recovery downstream to their prosthesis and thus better functional status than one would expect on the basis of the indexed EOAs and gradients measured by Doppler echocardiography.

Impact on postoperative morbidity. Two studies (6,83) have failed to demonstrate any association between the valve EOA indexed for BSA and the incidence of valve-related complications, such as thromboembolism, hemorrhage, structural valve deterioration and re-operation. However, a transversal study (5) in 61 patients with an aortic bioprosthesis showed that the occurrence of adverse clinical events not related to the valve was significantly higher in patients with an indexed EOA =0.85 cm²/m² (50% vs. 21%; p = 0.02). A longitudinal study (6) in patients with a stented bioprosthesis also suggested that mismatch could predispose them to the long-term development of heart failure, whether or not it is related to the prosthetic valve, but this was not confirmed as an independent predictor by multivariate analysis. It is therefore difficult to ascertain whether the difference between the two groups was related to the presence of mismatch or to other undetermined factors. Of the 392 patients followed for up to seven years, only two (0.5%) needed re-operation because they had severe mismatch and became symptomatic. Obviously, further long-term studies appear warranted. It is possible that the follow-up periods in these previous studies were not long enough to detect more significant differences. Nonetheless, medium-term prognosis with respect to postoperative morbidity appears relatively good. Future longitudinal studies may also be more difficult to perform or may even become obsolete, because awareness with regard to mismatch is increasing, and, as will be shown, this mismatch can largely be avoided.

Impact of mismatch on postoperative mortality. Several previous studies have demonstrated that mortality was higher in patients receiving a small (=21 mm) aortic prosthesis (86–88). For instance, Abdelnoor et al. (86) found that the five-year survival rate relative to a normal population was significantly lower in patients receiving a small aortic prosthesis (63%) than in the total cohort of patients (87%). Furthermore, Kratz et al. (89) reported that mortality 10 years after the operation was higher when a small (19 or 21 mm) St. Jude aortic valve prosthesis was implanted in a patient with a BSA >1.9 m². In this context, it should be remembered that a small valve size is more likely to be associated with significant prosthesis–patient mismatch (5), and it is highly possible that the patients reported in these studies were more extreme cases of mismatch, but this is difficult to ascertain given that the aforementioned studies often do not report values for indexed EOAs. Also, a smaller valve size may actually be an indicator of additional risk factors, such as a senescent aortic stenosis, a small calcified aortic root, marked LV hypertrophy and a smaller BSA (87). Finally, the implantation of a prosthesis in a small calcified aortic root can be technically more difficult and often requires a longer aortic cross-clamp time, which is a major risk factor in these patients (86,90). From these findings, it becomes evident that further longitudinal studies are necessary to determine whether the higher mortality associated with smaller valve sizes is due to prosthesis–patient mismatch or to the other aforementioned factors. To clarify this, it is important to report the results for the indexed EOA and to include them in the risk factor analysis.

Two previous studies in a relatively small number of patients failed to demonstrate a negative impact of mismatch (i.e., of the indexed valve EOA at operation) on short- and medium-term (up to eight years) mortality (6,83). However, in a recent study of 2,516 patients who underwent aortic valve replacement with a stented bioprosthetic valve, Rao et al. (91) showed that patient age (relative risk 1.07, 95% confidence interval 1.04 to 1.09) and indexed valve EOA (relative risk 1.46, 95% confidence interval 0.95 to 2.24), but not prosthesis size, were independent predictors of postoperative valve-related mortality. Mismatch had a significant impact on survival when the indexed EOA of the prosthetic valve at the time of the operation was <0.75 cm²/m² (75 ± 5% vs. 84 ± 2%; p = 0.004) at 12 years. This is somewhat in contrast with studies of native aortic valves showing that aortic stenosis is generally associated with higher morbidity and mortality rates when the indexed EOA is <0.60 cm²/m² (21,22). This apparent discrepancy could be due to the fact that Rao et al. used in vitro EOA values to calculate their indexed EOAs and that, although there is a good correlation between the two, in
vitro EOAs tend to overestimate in vivo EOAs by 10% to 15% (2,14,92,93).

**MANAGEMENT AND PREVENTION OF MISMATCH**

**Clinical management of mismatch.** The finding of a high transvalvular pressure gradient in a patient with a prosthetic valve is often a difficult diagnostic challenge. High transprosthetic pressure gradients may be present after aortic valve replacement, either because of intrinsic stenosis, a state of high cardiac output or prosthesis–patient mismatch. The most logical approach for assessing intrinsic prosthesis performance is to compare the EOA measured by Doppler echocardiography to the reference values measured either in vitro or in vivo for the same model and size of prosthesis. A value substantially lower than the reference values would certainly suggest an intrinsic stenotic process (e.g., tissue ingrowth, thrombus, calcification), and even more so if there had been a progressive decline of the EOA over time. In such cases, replacement of the prosthetic valve should be considered, because intrinsic stenosis processes are usually progressive. If the finding of a high transvalvular gradient is mainly due to prosthesis–patient mismatch, as indicated by an EOA consistent with normal reference values but an indexed EOA \(\leq 0.85\) cm\(^2\)/m\(^2\), there are no precise guidelines at present; however, given that these patients have a relatively good medium-term prognosis but may show hemodynamic deterioration and higher mortality in the long term (6,91), it would seem logical to follow them more closely. If, on the other hand, the patient develops the usual symptoms associated with aortic stenosis (i.e., angina, dyspnea or syncope) and has an indexed EOA compatible with severe stenosis (i.e., \(\leq 0.60\) cm\(^2\)/m\(^2\)), he or she should certainly be considered for re-operation, as is the case for native valves (22). In patients with mismatch undergoing re-operation, it would be very important to insert a prosthesis that will provide a larger indexed EOA, resulting in better hemodynamic performance. For this purpose, the surgeon could insert a prosthesis with a larger EOA either because it is of a larger size or a different type. If aortic root enlargement is contemplated, its risks must be weighed against the anticipated benefits.

**Prevention of mismatch.** To avoid prosthesis–patient mismatch, we would suggest a simple three-step algorithm that can easily be performed in the operating room (Table 1):

**Step 1**—Calculate the patient’s body surface (BSA) area using the formula:

\[
BSA = (\text{Weight}_{(kg)}^{0.425} \times \text{Height}_{(cm)}^{0.725}) \times 0.007184
\]

**Step II**—Determine the minimal requirement for prosthetic valve effective orifice area (EOA) to avoid prosthesis–patient mismatch.

**Step III**—Select the type and size of prosthesis that has reference values for EOA (2,3,13,14,26,39,93,95–102) greater or equal to the minimal EOA value obtained in step 2. Such values are provided in Table 2.

When examining the values in Table 2, the following points are important to remember:

1) Ideally, the reference values should be as representative as possible of the in vivo performance of the prostheses, and for this reason, we are giving the available in vivo values from the published data. Manufacturers also have in vitro values derived from premarketing studies; these values usually overestimate in vivo values by 10% to 15%, but otherwise correlate well with in vivo values (2,14,92,93). One notable exception is stentless valves, whose in vitro values for EOA grossly overestimate in vivo values and cannot be relied on (39). Our suggestion would therefore be that both in vitro and in vivo values for EOA be readily provided by the manufacturers, as they become available. Except in the case of stentless valves, in vitro values could temporarily be used as a reference until the in vivo values can be provided. Also,

<table>
<thead>
<tr>
<th>Patient BSA (m(^2))</th>
<th>Minimal Valve EOA (cm(^2)) for Indexed EOA &gt;0.85 cm(^2)/m(^2) (Ideal)</th>
<th>Minimal Valve EOA (cm(^2)) for Indexed EOA &gt;0.80 cm(^2)/m(^2)</th>
<th>Minimal Valve EOA (cm(^2)) for Indexed EOA &gt;0.75 cm(^2)/m(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.30</td>
<td>1.11</td>
<td>1.04</td>
<td>0.98</td>
</tr>
<tr>
<td>1.35</td>
<td>1.15</td>
<td>1.08</td>
<td>1.01</td>
</tr>
<tr>
<td>1.40</td>
<td>1.20</td>
<td>1.12</td>
<td>1.05</td>
</tr>
<tr>
<td>1.45</td>
<td>1.23</td>
<td>1.16</td>
<td>1.09</td>
</tr>
<tr>
<td>1.50</td>
<td>1.28</td>
<td>1.20</td>
<td>1.13</td>
</tr>
<tr>
<td>1.55</td>
<td>1.32</td>
<td>1.24</td>
<td>1.16</td>
</tr>
<tr>
<td>1.60</td>
<td>1.36</td>
<td>1.28</td>
<td>1.20</td>
</tr>
<tr>
<td>1.65</td>
<td>1.40</td>
<td>1.32</td>
<td>1.24</td>
</tr>
<tr>
<td>1.70</td>
<td>1.45</td>
<td>1.36</td>
<td>1.28</td>
</tr>
<tr>
<td>1.75</td>
<td>1.49</td>
<td>1.40</td>
<td>1.31</td>
</tr>
<tr>
<td>1.80</td>
<td>1.53</td>
<td>1.44</td>
<td>1.35</td>
</tr>
<tr>
<td>1.85</td>
<td>1.57</td>
<td>1.48</td>
<td>1.39</td>
</tr>
<tr>
<td>1.90</td>
<td>1.62</td>
<td>1.52</td>
<td>1.43</td>
</tr>
<tr>
<td>1.95</td>
<td>1.66</td>
<td>1.56</td>
<td>1.46</td>
</tr>
<tr>
<td>2.00</td>
<td>1.70</td>
<td>1.60</td>
<td>1.50</td>
</tr>
<tr>
<td>2.05</td>
<td>1.74</td>
<td>1.64</td>
<td>1.54</td>
</tr>
<tr>
<td>2.10</td>
<td>1.79</td>
<td>1.68</td>
<td>1.58</td>
</tr>
<tr>
<td>2.15</td>
<td>1.83</td>
<td>1.72</td>
<td>1.61</td>
</tr>
<tr>
<td>2.20</td>
<td>1.87</td>
<td>1.76</td>
<td>1.65</td>
</tr>
<tr>
<td>2.25</td>
<td>1.91</td>
<td>1.80</td>
<td>1.69</td>
</tr>
<tr>
<td>2.30</td>
<td>1.96</td>
<td>1.84</td>
<td>1.73</td>
</tr>
<tr>
<td>2.35</td>
<td>2.01</td>
<td>1.89</td>
<td>1.76</td>
</tr>
<tr>
<td>2.40</td>
<td>2.04</td>
<td>1.92</td>
<td>1.80</td>
</tr>
<tr>
<td>2.45</td>
<td>2.08</td>
<td>1.96</td>
<td>1.84</td>
</tr>
<tr>
<td>2.50</td>
<td>2.13</td>
<td>2.00</td>
<td>1.88</td>
</tr>
</tbody>
</table>
Table 2. Normal Effective Orifice Areas for the Most Currently Used Prosthetic Valves

<table>
<thead>
<tr>
<th>Prosthetic Valve Size (mm)</th>
<th>19</th>
<th>21</th>
<th>23</th>
<th>25</th>
<th>27</th>
<th>29</th>
<th>Reference no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stented Bioprosthetic valves</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medtronic Intact</td>
<td>0.85</td>
<td>1.02 ± 0.10</td>
<td>1.27 ± 0.11</td>
<td>1.40 ± 0.20</td>
<td>1.66 ± 0.16</td>
<td>2.04 ± 0.23</td>
<td>(2)</td>
</tr>
<tr>
<td>Medtronic Mosaic</td>
<td>—</td>
<td>1.22 ± 0.27</td>
<td>1.38 ± 0.23</td>
<td>1.65 ± 0.39</td>
<td>1.59 ± 0.33</td>
<td>1.65 ± 0.37</td>
<td>(95)</td>
</tr>
<tr>
<td>Hancock II</td>
<td>—</td>
<td>1.18 ± 0.11</td>
<td>1.33 ± 0.16</td>
<td>1.46 ± 0.15</td>
<td>1.55 ± 0.18</td>
<td>1.60 ± 0.15</td>
<td>(3)</td>
</tr>
<tr>
<td>Carpentier-Edwards SAV 2650</td>
<td>—</td>
<td>1.16 ± 0.14</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>(96)</td>
</tr>
<tr>
<td>Carpentier-Edwards Pericardial 2900</td>
<td>1.10</td>
<td>1.30</td>
<td>1.50</td>
<td>1.80</td>
<td>1.60</td>
<td>—</td>
<td>(97)</td>
</tr>
<tr>
<td>Stentless bioprosthetic valves</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medtronic Freestyle</td>
<td>1.15</td>
<td>1.35 ± 0.21</td>
<td>1.48 ± 0.33</td>
<td>2.00 ± 0.39</td>
<td>2.32 ± 0.48</td>
<td>—</td>
<td>(39)</td>
</tr>
<tr>
<td>Hancock II</td>
<td>1.29 ± 0.19</td>
<td>1.46 ± 0.32</td>
<td>1.79 ± 0.33</td>
<td>2.34 ± 0.69</td>
<td>2.67 ± 0.75</td>
<td>—</td>
<td>(98)</td>
</tr>
<tr>
<td>St. Jude Medical Toronto SPV</td>
<td>—</td>
<td>1.30</td>
<td>1.50</td>
<td>1.70</td>
<td>2.00</td>
<td>2.50</td>
<td>(SJM1)</td>
</tr>
<tr>
<td>Prima Edwards</td>
<td>0.80</td>
<td>1.10</td>
<td>1.50</td>
<td>1.80</td>
<td>2.30</td>
<td>2.80</td>
<td>(100)</td>
</tr>
<tr>
<td>Mechanical valves</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medtronic Hall</td>
<td>1.19 ± 0.21*</td>
<td>1.34 ± 0.15</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>(96)</td>
</tr>
<tr>
<td>Carbomedics Standard</td>
<td>1.00 ± 0.40</td>
<td>1.54 ± 0.31</td>
<td>1.63 ± 0.30</td>
<td>1.98 ± 0.41</td>
<td>2.41 ± 0.46</td>
<td>2.63 ± 0.38</td>
<td>(93)</td>
</tr>
<tr>
<td>St. Jude Medical Standard</td>
<td>1.11 ± 0.13</td>
<td>1.52 ± 0.22</td>
<td>1.84 ± 0.25</td>
<td>2.12 ± 0.31</td>
<td>2.65 ± 0.21</td>
<td>—</td>
<td>(14)</td>
</tr>
<tr>
<td>St. Jude Medical Hemodynamic Plus</td>
<td>1.04 ± 0.19</td>
<td>1.38 ± 0.22</td>
<td>1.52 ± 0.26</td>
<td>2.08 ± 0.41</td>
<td>2.65 ± 0.58</td>
<td>3.23 ± 0.30</td>
<td>(13)</td>
</tr>
</tbody>
</table>

*The label valve size of this valve is 20 mm. †Data provided by St. Jude Medical.

Effective orifice areas are expressed as the mean value ±SD cm². The effective orifice areas were measured by Doppler echocardiography using the continuity equation in patients with normally functioning prostheses. Some data appear conflicting or are based on limited series and may have to be revised as more data become available.

the in vivo values should ideally be taken at one year after the operation, because hemodynamic data may change during the first year (39,40,103,104). Finally, it should be remembered that in vivo EOA values for bileaflet valves may artificially be underestimated when evaluated by Doppler echocardiography owing to localized high velocity jets (105–108), and hence, in this case, a value for EOA lower than the reference value does not necessarily indicate prosthesis dysfunction.

2) For similar prosthesis size, the EOA can vary considerably depending on the type of prosthesis. Hence, in general, mechanical prostheses tend to have a larger EOA than bioprostheses, except for stentless bioprostheses, whose performance may be equivalent or better. Evidently, within the same category of prosthesis, performance can vary considerably from one manufacturer to the other.

3) Unfortunately, there are often important discrepancies between the actual prosthesis ring outer diameter and the manufacturer’s labeled valve size (109–111). Better standardization is desirable, but for the time being, sizing must be performed in the operating room using the sizer relevant to the prosthesis considered for insertion. The reference values for EOAs should therefore be readily available in the operating room to determine whether a particular prosthesis meets the requirements to avoid prosthesis–patient mismatch. If not, the insertion of a larger prosthesis size or that of a different type with a better hemodynamic performance should be considered.

The insertion of a larger prosthesis may require enlargement of the aortic root (112–114), in which case the increased operative risk must be weighed against the anticipated benefits. Other alternatives to avoid mismatch include performing a supra-annular implantation (49,112,115) or choosing a different type of prosthesis. In this context, the advent of stentless bioprostheses represents a major advance, because these prostheses generally have a much better hemodynamic performance than stented bioprostheses both at rest and during exercise (20,28–32,37–41,43). Indeed, stentless bioprostheses provide a larger EOA in relation to the patient’s BSA, resulting in a larger indexed EOA and a lower gradient at any given flow level (Fig. 3) (20). The superior hemodynamic performance of stentless valves is due to the fact that, size for size, their EOA is generally larger than that of stented valves. Moreover, for the stentless valves, a larger prosthesis can be inserted in a smaller annulus (32,36,39,42). Hence, Walther et al. (42) have recently shown in a randomized clinical trial that the prosthetic valve size is larger in the patients receiving a stentless bioprosthesis (25 ± 2 mm) than in those receiving a stented bioprosthesis (23 ± 2 mm), despite a similar size of the anatomic aortic annulus (24 ± 2 mm in both groups). Other attractive alternatives also include aortic homografts (116,117) or pulmonary autografts (Ross procedure) (118–121), which provide an indexed EOA similar to that of the normal native aortic valve (Fig. 3).

These alternative surgical techniques require a longer learning period and are frequently associated with longer aortic cross-clamp times and increased blood loss during the operation (112,122–124). In analyzing the different options,
it is therefore important to consider whether the benefits of avoiding prosthesis–patient mismatch overcome the drawbacks of using these techniques. First, mild degrees of prosthesis–patient mismatch may be acceptable when the surgical risk is high, whereas severe mismatch may not be acceptable. Second, the level of physical activity of the patient is an important variable to consider, because what would be acceptable for an older sedentary patient would not necessarily be adequate in a young active patient. In this context, the relations given in Figure 3 can be useful in predicting the rest and exercise postoperative gradients from the projected indexed EOA, which can easily be calculated in the operating room by dividing the reference value for the EOA of the prosthesis being implanted (Table 2) by the patient’s BSA. In making these choices, one must also realize that knowledge is still evolving and that newer data on the long-term impact of mismatch on mortality and morbidity are progressively becoming available. As discussed, there are presently limitations in determining the “critical” indexed EOA at which adverse events occur, and these new data should contribute to the refinement of strategy for mismatch prevention. Nonetheless, the knowledge of these variables provides the framework for making more enlightened decisions, because the hemodynamic outcome is no longer an unknown variable but can be forecasted before the operation. In addition, it provides the impetus for the development of better performing prostheses.

Conclusions. Patients with evidence of aortic prosthesis–patient mismatch have less symptomatic improvement and worse hemodynamic data after aortic valve replacement. Furthermore, the hemodynamic status progressively deteriorates during follow-up, and mismatch has negative impacts on the regression of LV hypertrophy, as well as on long-term survival. Nonetheless, aortic prosthesis–patient mismatch can largely be avoided by calculating, before the operation, the indexed EOA from reference values for the EOA of the prosthesis being implanted and the patient’s BSA, remembering that for optimal valve performance at rest and exercise, the indexed EOA at rest should ideally be no less than 0.85 to 0.90 cm²/m². However, achievement of such a goal must be evaluated in light of the anticipated risks and benefits for a given patient.

Acknowledgment

The authors thank Isabelle Laforest, MS, for her assistance in the preparation of the figures.

References


63. González-Juanatey JR, García-Ácuna JM, Fernandez MV, et al. Influence of the size of aortic valve prostheses on hemodynamic and...
110. Cochran RP, Konzelman KS. Discrepancies between labeled and