VALVULAR HEART DISEASE

Prediction of valve prosthesis–patient mismatch prior to aortic valve replacement: which is the best method?

Sabine Bleiziffer, Walter B Eichinger, Ina Hettich, Ralf Guenzinger, Daniel Ruzicka, Robert Bauernschmitt, Ruediger Lange

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See end of article for authors' affiliations

Correspondence to: Dr S Bleiziffer, Clinic for Cardiovascular Surgery, German Heart Center Munich, Lazarettstr. 36, Munich 80636, Germany; bleiziffer@dhm.mhn.de

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Background: To predict the occurrence of valve prosthesis–patient mismatch (VP–PM) after aortic valve replacement (AVR), the surgeon needs to estimate the postoperative effective orifice area index (EOAI).
Aim: To compare different methods of predicting VP–PM.
Methods: The effective orifice area (EOA) of 383 patients who had undergone AVR between July 2000 and January 2005 with various aortic valve prostheses was obtained echocardiographically 6 months after the operation. We tested the efficacy of (1) EOAI calculated from the echo data obtained in our own laboratory, (2) indexed geometric orifice area, (3) EOAI estimated from charts provided by prosthesis manufacturers (which are based either on in vitro or on echo data) and (4) EOAI estimated from reference echo data published in the literature to predict VP–PM.
Results: Sensitivity and specificity to predict VP–PM were 53% and 83% (method 1), 80% and 53% (charts based on echo data, parts of method 3) and 71% and 67% (method 4) using reference data derived from echocardiographic examinations. The sensitivity of method 2 and of charts based on in vitro data (parts of method 3) to predict VP–PM was 0–17%. The incidence of severe VP–PM could be reduced from 8.7% to 0.8% after the introduction of the systematic estimation of the EOAI at the time of operation ($p = 0.003$, method 1).
Conclusions: The best method of predicting VP–PM is the use of mean (SD) EOAs derived from echocardiographic examinations, whereas the use of in vitro data or the geometric orifice area is unreliable. After the surgeon's anticipation of VP–PM prior to AVR, the incidence of VP–PM could be reduced.

The clinical impact of valve prosthesis–patient mismatch (VP–PM)[1] after aortic valve replacement (AVR) is controversial. Several studies have shown that VP–PM is associated with lesser regression of left ventricular mass, more cardiac events and lower survival after AVR,[2–10] whereas other studies reported that VP–PM has no or minimal impact on postoperative outcomes.[11–15] The use of different methods to identify VP–PM might have contributed to the contradictory results in previous studies.

To predict the risk of occurrence of VP–PM at the time of operation, it is necessary to obtain a reliable estimate of the postoperative effective orifice area index (EOAI). This estimation is generally performed by indexing the normal reference value of the effective orifice area (EOA) for the type and size of prosthesis that is implanted by the patient's body surface area (BSA). The source of EOA reference values is therefore critical. In this regard, some authors have used the EOAs measured in vivo by Doppler echo in their own institution or reported in the literature.[2–10] Other authors have used EOAs measured in vitro by the manufacturer.[16 17] Some authors also attempted to identify VP–PM with the use of the internal geometric orifice area (GOA) derived from the internal prosthesis diameter.[11–14] In addition, prosthesis manufacturers have also provided charts to help the surgeon to assess the risk of VP–PM at the time of operation. However, these charts have been established with the use of different reference EOA sources and it remains uncertain whether or not these charts are reliable.

The objective of this study was thus to compare the performance of the different methods that have been previously proposed to predict VP–PM. An additional objective was to assess the impact of systematic estimation of the EOAI at the time of operation on the postoperative incidence of VP–PM.

METHODS

Between July 2000 and January 2005, 792 patients underwent primary AVR at our institution with stented or stentless bioprostheses or with mechanical prostheses, 468 of whom signed informed consent for echocardiographic follow-up. Ten patients died and 73 were lost to follow-up because of moving abroad or refusing echocardiography. Two patients had incomplete echo data due to bad echocardiographic conditions. Thus, echocardiographic data of 383 patients were available for the present study.

All echocardiographic examinations were performed by an experienced investigator. Echocardiography was carried out using an image Point Hx ultrasound system with a 2.5 MHz transducer (Hewlett Packard, Houston, Texas, USA). Routine follow-up echocardiography was performed under resting conditions 6 months postoperatively (mean (SD) follow-up time 6.1 ($\pm$2.1) months). Peak and mean pressure gradients in the left ventricular outflow tract (LVOT) 1 cm below the valve and across the valve were measured in an apical three- or five-chamber view using pulsed wave Doppler for the LVOT measurements and continuous wave Doppler for the valve measurements. Three of the best available signals were averaged in patients with sinus rhythm. If atrial fibrillation was present, a minimum of five measurements was averaged. EOA was obtained by using the continuity equation.[18] EOAI is the EOA per m^2 BSA, where BSA is derived from the Dubois formula. Due to the original describer, moderate VP–PM was

Abbreviations: AVR, aortic valve replacement; BSA, body surface area; CEP, Carpentier–Edwards prosthesis; EOA, effective orifice area; EOAI, effective orifice area index; GOA, geometric orifice area; LVOT, left ventricular outflow tract; VP–PM, valve prosthesis–patient mismatch

defined as an EOAI $\leqslant 0.9$ cm^2/m^2, and severe VP–PM as EOAI $\leqslant 0.6$ cm^2/m^2.[19]

We tested the reliability of four methods to predict VP–PM.

Method 1: We tested the accuracy of EOA values obtained from our institutional laboratory by Doppler examination 6 months postoperatively to predict VP–PM (table 1). This method has been used in conjunction with method 4 to identify VP–PM in a previous study.[4]

The numbers in brackets indicate the confidential interval.

Method 2: The indexed GOA was initially proposed in 1996 to predict VP–PM[14] and was used in large studies under the term ''prosthesis–patient size''.[12 13] In the present study, the GOA was calculated for each valve type and size from the internal valve diameter provided by the manufacturer. Thus, the GOA is a fixed geometric parameter for each valve type and size reflecting the internal valve orifice area.

Method 3: We also tested the quality of some commercially available charts when used on our patients to estimate the EOAI. The sources of such charts are either in vitro or echo data. Only charts of prostheses implanted in >45 patients and with a VP–PM incidence of >25% were included to avoid bias due to low pretest probability.

Method 4: The efficacy of predicting VP-PM when using EOA values published in the literature was tested. EOA values were available for the St Jude Regent, the Medtronic Mosaic, the Edwards Perimount and the Medtronic Advantage prosthesis.[3 20] This method has been used in previous studies.[21 22]

The second objective of our study was to assess the effect of systematic estimation of EOAI at the time of operation on the incidence of VP–PM. Taking the patient's BSA into account, the EOAI was calculated prospectively in 119 patients using method 1 since March 2004. In particular, we have had a list in the odds ratio (OR) with mean EOAs derived from our institutional follow-up echocardiographic investigations performed 6 months postoperatively for each valve type and each labelled valve size (table 1). The list was updated every 6 months, and is continuously in progress. Thus, the surgeons were able to choose a valve type and size large enough to prevent VP–PM. In all, 13 surgeons participated in the study during this time period. The decision whether a bioprosthesis or a mechanical prosthesis would be implanted was made preoperatively. Intraoperatively, all surgeons aimed to implant the valve with the largest EOA, especially in small aortic annuli. In general, we try to avoid the implantation of 19 mm prostheses. The incidence of VP–PM in this group of 119 patients (operated on between March 2004 and January 2005) was compared with the incidence of VP–PM in 264 patients (operated on between January 2000 and March 2004) before the introduction of intraoperative VP–PM prediction. Table 2 summarises the baseline characteristics and implanted valve types in the two groups.

RESULTS

Among 383 patients undergoing AVR, moderate VP–PM occurred in 128 (33.4%) and severe VP–PM occurred in 24 (6.3%) patients. Using four different predictive schemas, VP–PM was accurately predicted as follows.

Method 1: Table 3 lists the sensitivity, specificity, andpositive and negative predictive values using institutional mean EOA values to predict VP–PM. There is a significant linear correlation between the predicted and actual EOAI ($R = 0.620$, $p<0.001$; fig 1).

Method 2: Using the GOA instead of the echocardiographically obtained EOA, VP–PM could not be predicted in a single patient (sensitivity = 0%, table 3).

Method 3: When pooling all charts together, a sensitivity of 26% and a specificity of 92% was obtained to predict VP–PM. However, the performance varied extensively from one chart to another (table 3).

Method 4: Table 3 lists the sensitivity, specificity, and positive and negative predictive values for the EOA values published in the literature when used on our patients.

Regarding the subgroup of patients exhibiting severe mismatch, the proportion of patients who would have been detected by different methods is as follows: 22/24 (92%, method 1), 0/24 (0%, method 2), 13/22 (59%, method 3) and 19/22 (86%, method 4).

In our prospective cohort of 119 patients, VP–PM was predicted intraoperatively in 14 cases and was confirmed in 9 patients at follow-up echocardiography. In these cases, the surgeons accepted the risk of VP–PM, as it was not possible to implant a larger valve without enlarging the aortic annulus (fig 2). The incidence of moderate and severe VP–PM was 43.9% (116/264) before and was reduced to 30.3% (36/119) after calculating EOAI prior to AVR ($p = 0.011$). Severe VP–PM was reduced from 8.7% (23/264) to 0.8% (1/119; $p = 0.003$).

DISCUSSION

Assessment of VP–PM

The presence of VP–PM in each individual patient should be determined by measurement of the EOA. We used echocardiographic data obtained 6 months postoperatively, as immediate

Table 1 List of effective orifice area values derived from our institutional echocardiography data

	Labelled valve size					
Prosthesis	**19**	**21**	**23**	**25**	**27**	**29**
Edwards Perimount*	0.97 (0.81 to 1.14)	1.39 (1.22 to 1.57)	1.79 (1.58 to 1.99)	1.73 (1.34 to 2.11)		
Edwards Perimount Magna*	1.41 (1.01 to 1.81)	1.49 (1.40 to 1.59)	1.89 (1.78 to 2.00)	2.09 (1.87 to 3.32)		
St Jude Epic Supra†		1.6 (1.33 to 1.87)	2.38 (2.2 to 2.55)			
Medtronic Mosaic‡	0.74 (0.53 to 0.95)	1.18 (1.02 to 1.34)	1.54 (1.31 to 1.77)	1.94 (1.63 to 2.24)	2.14 (1.63 to 2.65)	
St Jude Toronto Root†			1.53 (1.32 to 1.73)	1.86 (1.48 to 2.24)	2.44 (2.05 to 2.83)	2.22 (1.71 to 2.52)
St Jude Regent†		1.99 (1.76 to 2.20)	2.4 (1.85 to 2.78)	2.8 (2.40 to 3.21)		
Medtronic Advantage‡		1.08 (0.89 to 1.27)	1.88 (1.62 to 2.13)	2.36 (2.11 to 2.61)	2.33 (1.98 to 2.68)	
Medtronic Advantage supra‡			2.13 (1.74 to 2.52)	2.81 (1.80 to 3.82)		
	Labelled valve size					
	18	**20**	**22**	**24**	**26**	**28**
Sorin Soprano§	1.25 (0.89 to 1.71)	1.56 (1.43 to 1.75)	1.78 (1.67 to 1.89)	1.91 (1.55 to 2.28)	2.5 (1.98 to 3.04)	

Values are represented as mean (95% CI).
*Edwards Lifesciences (Irvine, California, USA)
†St Jude Medical, Inc. (St Paul, Minnesota, USA)
‡Medtronic, Inc. (Minneapolis, Minnesota, USA)
§Sorin Biomedica (Saluggia, Italy)

Table 2 Patient characteristics and implanted valve prostheses

	All (n = 383)	Before introduction of method 1 (n = 264)	After using method 1 (n = 119)	p Value
Age (years)	69.6 (±9.9)	70.0 (±9.7)	68.5 (±10.2)	0.168
Female)	39.2%	39.4%	38.7%	0.891
BSA (m^2)	1.85 (±0.19)	1.85 (±0.19)	1.87 (±0.19)	0.158
Prevalence of CAD	55.5%	52.3%	64.2%	0.075
Valvular lesion				
Stenosis	94.7%	94.4%	95.8%	0.639
Regurgitation	5.3%	5.6%	4.2%	
Labelled valve size	23.0 (±2.1)	23.1 (±2.1)	22.9 (±1.9)	0.270
EOAI (cm^2/m^2) at FU	0.99 (±0.29)	0.97 (±0.30)	1.04 (±0.25)	0.016
NYHA at FU	1.4 (±0.5)	1.4 (±0.5)	1.5 (±0.6)	0.103
Valve prosthesis (n)				<0.001
Medtronic Advantage*	54	54	0	
sizes 21/23/25/27	2/18/21/13	2/18/21/13	0/0/0/0	
Edwards Perimount†	53	49	4	
sizes 19/21/23/25	4/19/24/6	4/16/23/6	0/3/1/0	
Edwards Perimount Magna†	123	64	59	
sizes 19/21/23/25	6/40/50/27	3/14/30/17	3/26/20/10	
Medtronic Mosaic*	49	49	0	
sizes 19/21/23/25/27	2/13/22/10/2	2/13/22/10/2	0/0/0/0/0	
Medtronic Advantage supra*	12	0	12	
sizes 21/23/25	1/6/5	0/0/0	1/6/5	
St Jude Epic supra‡	5	0	5	
sizes 21/23	3/2	0/0	3/2	
Sorin Soprano§	52	35	17	
sizes 18/20/22/24/26/28	3/14/16/15/3/1	3/14/6/11/1/0	0/0/10/4/2/1	
St Jude Regent‡	16	0	16	
sizes 19/21/23/25	1/3/3/9	0/0/0/0	1/3/3/9	
St Jude Toronto Root‡	19	13	6	
sizes 23/25/27/29	2/8/6/3	1/7/3/2	1/1/3/1	

BSA, body surface area; CAD, coronary artery disease; EOAI, effective orifice area index; FU, follow-up; NYHA, New York Heart Association.
For this study, we used data from our institutional database containing follow-up and echocardiographic data. We had obtained approval from the ethics committee for data collection (625/02, 1029/04, 00101, 922/03, 711/02). Data are presented as mean (SD) and as percentages. Differences in groups were tested with the Student's t test or the χ^2 test as appropriate. Statistical significance was achieved at $p<0.05$.
*Medtronic, (Minneapolis, Minnesota, USA).
†Edwards Lifesciences (Irvine, California, USA).
‡St Jude Medical, (St Paul, Minnesota, USA).
§Sorin Biomedica (Saluggia, Italy).

postoperative measurements are not representative because haemodynamics are not well stabilised at the pre-discharge examination. Patients could be in a hyperdynamic state or exhibit tachycardia, which makes the assessment of the EOA difficult. Additionally, the acoustic window is often of poor quality in the early postoperative period.

It should be kept in mind that, unlike the label prosthesis size or the GOA, the EOA is not necessarily a fixed parameter but rather a physiological parameter that may vary with flow rate.[22] In mechanical aortic valve prostheses, the EOA may depend on the orientation of the valve, as different orientations may cause different flow patterns.[25] In bioprosthesis, EOA variation may depend on patient hemodynamics, such as cardiac output and ventricular function, and also on variations due to the biological tissue, as shown by in vitro measurements.[23]

The purpose of the intraoperative methods used to predict VP–PM is to provide an estimate of the average EOA that a given type and size of prosthesis can generally achieve at normal resting flow-rate conditions.

Assessment of VP–PM in previous publications

The four methods evaluated in the present study have already been used in previous publications to identify VP–PM. Our results might contribute to reconcile the discrepancies between previous studies. Indeed, the vast majority of the studies that have used method 4 to project the postoperative EOAI at the time of operation,[2–4 6 7 9 10] or that have measured the EOAI directly by Doppler examination in the postoperative period,[5 8] have concluded that VP–PM is a common phenomenon and that it has a significant negative impact on postoperative outcomes. On the other hand, authors who have used method 2[11–14] or the indexed in vitro EOAs provided by the manufacturer[17] (as in some charts of method 3) concluded that VP–PM is rare and has little or no impact on outcomes. This is not surprising as, in the light of the results of the present study, the latter methods have very poor performance, and in particular very low sensitivity to predict VP–PM. Consequently, to accurately predict the risk of VP–PM at the time of operation it is crucial to use reliable sources of reference EOAs to project the postoperative EOAI.

In addition, some authors have used a combination of several methods. Tasca *et al*[4] used method 4 except for one prosthesis model. As EOA data were not available in the literature for this model, they used EOA data from their own laboratory (method 1). The conclusion of this study was that VP–PM is an independent risk factor for postoperative morbidity and mortality. Walther *et al*[9] predominantly used method 4 except for one model for which they used method 3 (in vitro data provided by the manufacturer). They found that VP–PM has a significant impact on postoperative mortality.

Some authors have assessed the predictive value of both indexed EOA and indexed GOA.[4 5] Interestingly, the indexed EOA was a strong independent predictor of postoperative morbidity and mortality, whereas indexed GOA was not. This finding is consistent with the results of the present study.

In conclusion, our results may contribute to reconcile the discrepancies of previous studies, as the performances of the methods to predict VP–PM vary extensively.

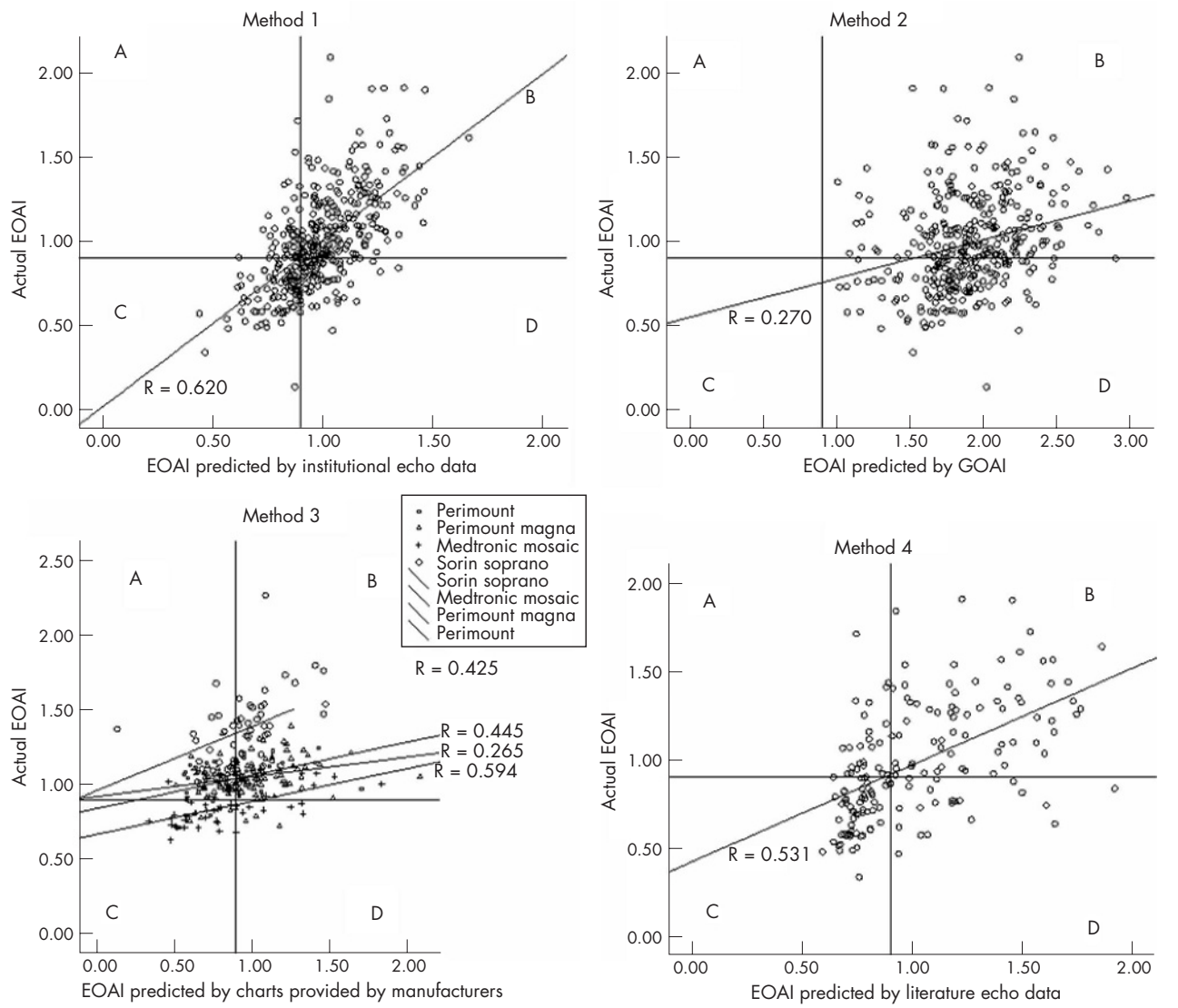

Figure 1 Scatter plots of the predicted versus actual indexed effective orifice area values (cm^2/m^2) for the four tested methods. The horizontal and vertical lines in each diagram mark the valve prosthesis–patient mismatch threshold of 0.9 cm^2/m^2. Good prediction methods are characterised by a correlation line through the crossing of those two lines (method 1, and parts of methods 3 and 4). A, false positive predictions; B, correct negative predictions; C, correct positive predictions; and D false negative predictions. EOAI, effective orifice area index; GOAI, geometric orifice area index.

Which is the best method?

When method 1 is used, data must be interpreted with caution. Flameng *et al*[26] used method 1 to predict VP–PM in a cohort of patients who underwent AVR with a Carpentier–Edwards prosthesis (CEP). Surprisingly, unlike previous studies,[10 27 28] they found that VP–PM was rare in their series and had no significant impact on outcomes. However, it should be noted that the EOA reference values measured in their laboratory and used as the data source to predict VP–PM were substantially higher than those reported in previous studies[10 27 28] and in the present study, and were in fact equivalent to the in vitro data reported by the manufacturer. The results of the present study, however, show that the manufacturer's chart for the CEP, which is based on those in vitro data, has a low sensitivity to predict VP–PM. If method 1 is used, one should ensure that the reference EOA values are derived from a large cohort of patients with sufficient numbers in each valve type and size to receive a reliable estimation of the EOA. In addition, care must be taken to adequately measure the LVOT diameter and EOA. In particular, failure to record the highest jet velocity will result in an overestimation of EOAs, which will, in turn, reduce the sensitivity to predict VP–PM. These technical pitfalls might also have affected the performance of method 1 in the present study. For example, as table 1 shows, the EOA of the 23 mm CEP valve is surprisingly higher than that of the 25 mm CEP valve. The performance of method 1 will improve with the addition of more patients as our data are continuously updated.

Our results show that the use of the GOA is insufficient to predict VP–PM, as the sensitivity was 0%. In addition, the indexed GOA has been reported to be unrelated to post-operative outcomes.[4 5]

When using charts provided by prosthesis manufacturers, one must be aware of the origin of the data, as some charts are based on in vitro data, which might not reflect the complexity of haemodynamics. Those charts may overestimate the in-vivo EOA by 10–15%,[21] or even more, as the in vitro EOA provided by the manufacturer may be more optimistic. For example, the two charts provided by Edwards Lifesciences for the Perimount

Table 3 Test characteristics

	n	EOA values based on	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Method 1						
Institutional data	380	Echo data	0.54	0.83	0.67	0.75
Method 2						
GOA	364	GOA	0	1	0	0.60
Method 3						
Charts provided by companies	271	Mixed	0.26	0.92	0.73	0.60
Edwards Perimount	47	In vitro data[23]	0.17	1	1	0.66
Edwards Perimount Magna	123	In vitro data*	0.09	0.96	0.57	0.63
Medtronic Mosaic	49	Echo data[24]	0.80	0.53	0.73	0.63
Sorin Soprano	52	In vitro data†	0	1	0	0.65
Method 4						
Literature data	172	Echo data[3 20]	0.71	0.67	0.64	0.74

EOA, effective orifice area; GOA, geometric orifice area.
Values are mean.
*In vitro data from Edwards Lifesciences.
†In vitro data from Sorin Biomedica.

pericardial bioprosthesis and the Perimount Magna bioprosthesis show poor test characteristics for the prediction of VP–PM (table 3). For both charts, the number of patients in whom VP–PM would not have been predicted, but would have occurred, is fairly high, as shown by the poor negative predictive values. The Sorin Soprano chart even has a sensitivity of 0%, indicating that none of the patients with VP–PM could be predicted. This phenomenon might be due to the in-vitro-derived data. The chart available for the Medtronic Mosaic valve shows a better sensitivity and positive predictive value, as the data were derived from echocardiographic examinations. These observations, and the fact that the detection of patients who will exhibit severe VP–PM is superior with methods 1 and 4, strengthen our hypothesis that the EOA in the individual patient can be best estimated by mean values based on echocardiography in a large number of patients.

In summary, as the accuracy of the GOA and in vitro data was poor to predict VP–PM, the best method for the intraoperative assessment of the risk of VP–PM is the use of mean EOA values derived from large patient populations (method 4) as shown, in particular by superior sensitivity. If no data are available in the literature—for example in new valves—method 1 should be used.

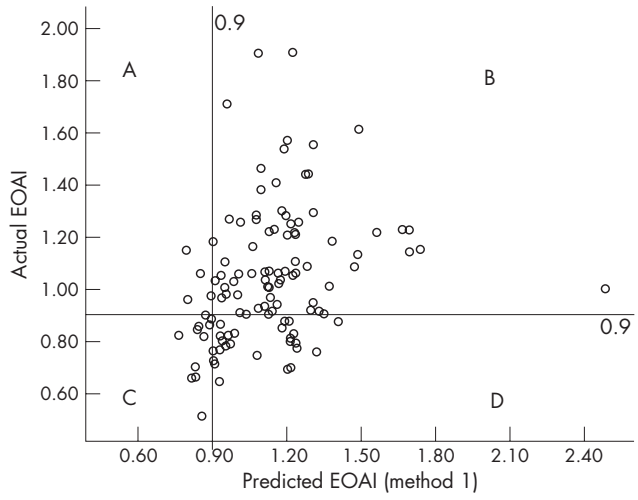

Figure 2 Scatter plot of (n=119) predicted effective orifice area index (EOAIs; method 1) and actual EOAI (cm^2/m^2) measured 6 months postoperatively (prospective data). A, false positive predictions; B, correct negative predictions; C, correct positive predictions; and D, false negative predictions.

VP–PM prevention

The present study contributes to develop a reasonable concept for VP–PM prevention. As a first step to prevent VP–PM, we suggest the intraoperative projection of the postoperative EOAI in every patient undergoing AVR using a method that provides an accurate estimate of the risk of VP–PM and of its severity (method 4 or 1, if no data are available in the literature). However, it is not always possible to implant a larger valve when VP–PM is predicted.[28] In the present study, VP–PM was anticipated in the OR in 14 cases, and 9 of those patients developed moderate VP–PM after postoperation. If VP–PM is predicted, they have several surgical options. The choice of another valve type—for example, a new generation supra-annular stented bioprosthesis or implantation of a mechanical or stentless prosthesis—might reduce the risk of occurrence of VP–PM. In the present study, after the introduction of the intraoperative anticipation of the EOA, a stented porcine valve was no longer used due to its inferior EOA values compared with other models (table 2). The 14 patients, for whom VP–PM was anticipated, received a biological stented valve with a labelled size of 21 or 22 mm. In all cases, the valve type with the largest possible EOA was chosen as a valve one size larger would not have fitted. Due to the American College of Cardiology/American Heart Association guidelines for the management of patients with valvular heart disease, ''a narrow LVOT […] may require enlargement of the annulus''.[29] The decision to extend the operative procedure from an isolated AVR to valve replacement plus root enlargement, which may lead to increased morbidity and mortality,[30] must be integrated into a differentiated assessment of the patient's comorbidities, age and lifestyle.[31] Alternatively, the implantation of a stentless valve could have been considered in these patients to improve the EOA, but would also have increased the length of the procedure. However, in the above-mentioned cases, the surgeons decided to accept the risk of VP–PM rather than extend the operation and bypass time. Thus, the preventive strategy must be individualised. Severe VP–PM should ideally be avoided in every patient. It may be reasonable to accept a moderate VP–PM in the elderly patient with a preserved left ventricular function. On the other hand, one should attempt to

avoid any degree of VP–PM in high-risk patients with poor left ventricular function and in young athlete patients.

Conclusion

It is critical to use a reliable source of EOA to adequately assess the risk of VP–PM at the time of operation. In this regard, the intraoperative estimation should be based on normal reference values of EOA measured in vivo by Doppler echocardiography. On the other hand, the GOA or the in vitro EOA provided by the manufacturer are not reliable to predict the EOAI, and thus the risk of VP–PM. The systematic estimation of the EOAI at the time of operation is associated with a significant reduction in the prevalence of VP–PM, especially of severe VP–PM. These results support the concept that being aware of the risk of VP–PM at the time of operation is useful to prevent this problem or, at least, reduce its severity.

Limitations of the study

In the present study, the EOA measured by Doppler examination 6 months postoperatively was used as the reference method to confirm the presence and severity of VP–PM. However, this reference method may also be affected by measurement errors. The classification errors are entirely attributed to the method used to predict the EOAI, whereas it may also be due to measurement errors related to the reference method used to measure the EOAI in the postoperative period.

Authors' affiliations

Sabine Bleiziffer, Walter B Eichinger, Ina Hettich, Ralf Guenzinger, Daniel Ruzicka, Robert Bauernschmitt, Ruediger Lange, Clinic for Cardiovascular Surgery, German Heart Center Munich, Munich, Germany

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