

# Durability and Prevalence of Aortic Regurgitation Nine Years after Aortic Valve Replacement with the Toronto SPV Stentless Bioprosthesis

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**Background and aim of the study:** Stentless aortic bioprostheses have excellent hemodynamics. Previous investigations of the Toronto SPV valve described a correlation between the occurrence of significant aortic regurgitation (AR) and dilation of the sinotubular junction. The study aim was to determine the long-term durability and determinants of AR at nine years in a large, multicenter study of the Toronto SPV valve.

**Methods:** The study included 447 patients from six centers. Clinical outcomes and echocardiographic data (gradients, effective orifice area index (EOA-I), left ventricular mass, aortic root dimensions, and presence and severity of AR) were collected prospectively. A multivariable logistic regression model was used to evaluate clinical and echocardiographic variables for impact on the occurrence of AR.

**Results:** Total follow up was 2,660 patient-years (mean  $6.0 \pm 2.5$  years; range: 0 to 11.1 years). Mean gradient and EOA-I remained unchanged through nine years. There were 17 cases of structural deterior-

ation, of which 15 underwent explantation. The mechanism of failure was predominantly leaflet tear in the setting of sinotubular dilation. Freedom from explant for structural failure was 90.1% at nine years (100% for patients aged  $\geq 65$  years). Freedom from hemodynamically significant AR was 96.9% at five years and 82.5% at nine years. Determinants of AR were longer duration of follow up, larger valve size, and increase in the ratio of sinotubular junction to the size of valve implanted.

**Conclusion:** At nine years after implantation of the Toronto SPV valve, hemodynamics remained excellent. There was good freedom from structural deterioration through nine years, and structural failure occurred due to aortic root dilation and leaflet tear, without significant valve calcification. AR tends to occur with longer follow up, larger valve sizes, and dilation of the sinotubular junction.

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Stentless aortic bioprostheses are associated with low transvalvular gradients and large effective orifice area (EOA), with early regression of abnormally increased left ventricular (LV) mass after aortic valve replacement. As a bioprosthesis, the utility of a stentless aortic valve will be determined by its durability. The Toronto SPV<sup>®</sup> valve is a stentless porcine aortic valve that is scalloped for consistent subcoronary implantation. Data describing its hemodynamic performance and durability through eight years have been reported previously (1-4).

Because the Toronto valve is implanted using a com-

plete subcoronary technique, coaptation of the cusps and preservation of valve geometry during diastole are influenced by the geometry of the ascending aorta. Previous investigation of the Toronto SPV valve at a single center described a strong correlation between dilation of the sinotubular junction and the occurrence of aortic regurgitation (AR) at 5.8 years (5). Specifically, increases over time in the size of the sinotubular junction and in the ratio of the sinotubular junction to the size of valve implanted were significantly associated with the presence of grade  $\geq 1+$  AR. A multicenter analysis of explants of the Toronto SPV valve demonstrated a high prevalence of aortic root and sinotubular dilation among patients who underwent reoperation for severe AR (6). In the same study, there was an association between native bicuspid aortic valve and late AR, likely related to a greater tendency for aortic root dilation among patients with a native bicuspid aortic valve (7). The durability of the Toronto SPV valve has

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not been previously described in a multicenter cohort beyond eight years. The aim of the present study was to determine the ongoing durability and freedom from AR in a large, multicenter study of the Toronto SPV valve, and to describe the relationship between aortic root geometry and late AR.

## Clinical material and methods

### Study group

The multicenter clinical evaluation of the Toronto SPV® stentless aortic valve (St. Jude Medical, Inc., St. Paul, MN, USA) began at 12 investigational centers in July 1991. The long-term study cohort included 447 consecutive patients from six of the original investigative centers in the United States and Canada, selected for patient volume and protocol adherence. Details of the study sites and clinical investigators are listed in Appendix I. For the present study, data were collected for 447 patients in the long-term study group between July 1991 and October 2002. At the time of surgery the group included 293 men and 154 women (mean age  $64.9 \pm 11.5$  years; range: 33 to 90 years). Other demographic features have been previously described (4). Valve sizes implanted were 20 to 22 mm (n = 20; 4.5%), 23 mm (n = 58; 13.0%); 25 mm (n = 106; 23.7%); 27 mm (n = 146; 32.7%) and 29 mm (n = 117; 26.2%). Concomitant coronary artery bypass grafting was performed in 185 patients (41.4%); mitral valve repair and aortoplasty were each performed in three patients (0.7%).

### Clinical follow up

Patients were prospectively monitored at the respective investigative centers throughout the postoperative follow up period for adverse events, following guidelines of the Society of Thoracic Surgeons and the American Association of Thoracic Surgeons (8). Clinical data were obtained prior to hospital discharge,

at six months and one year postoperatively, and yearly thereafter.

### Echocardiography

Transthoracic echocardiographic imaging with Doppler was performed at specified time periods, including prior to hospital discharge, at six months and one year postoperatively, and yearly thereafter. Imaging included two-dimensional (2-D) and M-mode echocardiography, pulsed- and continuous-wave spectral Doppler, and color flow Doppler imaging. The aortic valve prosthesis was interrogated for regurgitation using parasternal and apical windows. Echocardiographic images were recorded on standard or super VHS videotape or standard digital (DICOM) format for subsequent analysis.

Echocardiograms were obtained at the respective investigational centers and underwent analysis at a central, core echocardiography laboratory by observers highly trained in qualitative and quantitative echocardiography and Doppler. Observers were blinded to the clinical information at the time of echocardiographic analysis. Analysis included 2-D echocardiographic measurement of LV dimensions, and ejection fraction. Aortic root dimensions at the valve annulus, sinuses of Valsalva, sinotubular junction and tubular ascending aorta were measured from inner edge to inner edge. Aortic root measurements were made prospectively after 2001; echocardiograms performed prior to this were reanalyzed in retrospect for aortic root dimensions. Mean and peak transvalvular gradients were calculated using the modified Bernoulli equation ( $\Delta P = 4(V_2^2 - V_1^2)$ , correcting for proximal velocity in the LV outflow tract (9). EOA was calculated using the continuity equation (9). The LV mass index was calculated as the ratio of LV mass to body surface area; LV mass was calculated using the method of Devereux (10):

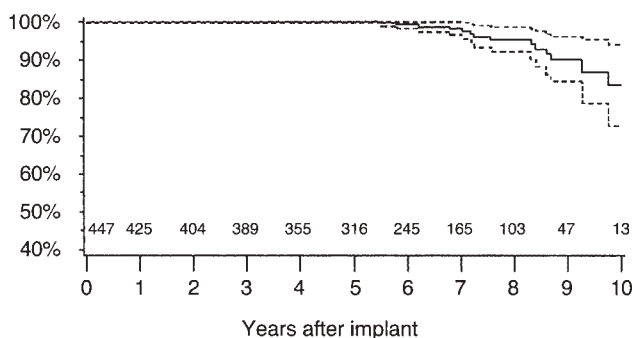


Figure 1: Freedom from explant for structural deterioration. Numbers above the x-axis indicate patients at risk.

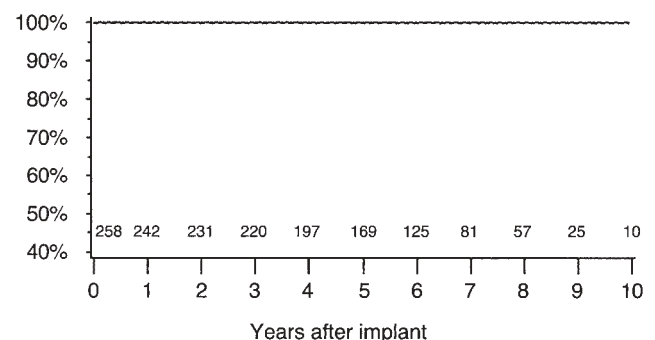


Figure 2: Freedom from explant for structural deterioration in patients aged  $\geq 65$  years at the time of implantation. Numbers above the x-axis indicate patients at risk.

$$\text{LV mass index (g/m}^2\text{)} = \{1.04 \times ([\text{IVS} + \text{LVIDD} + \text{PW}]^3 - [\text{LVIDD}]^3) - 13.6\} / \text{BSA}$$

where IVS, LVIDD and PW are standard measures (cm) of the interventricular septum thickness, LV internal diameter in diastole, and posterior wall thickness, respectively, and BSA is body surface area. If present, AR was quantified as either trivial, mild, moderate or severe based on the measurement of regurgitant jet diameter relative to the diameter of the LV outflow tract (11), assessment of AR deceleration slope on spectral continuous-wave Doppler, and assessment of diastolic flow reversal in the descending thoracic aorta.

### Statistical analysis

Continuous data were presented as mean  $\pm$  SD. Because only few implantations were performed using the smallest valve sizes, data were pooled for patients with valve sizes 20, 21 and 22 mm. Changes over time in transvalvular gradients, EOA and LV mass index were compared using paired Student's *t*-tests. Change over time in the prevalence of AR was compared using Fisher's exact test. Determinants of AR, including aortic root size, were investigated using a multivariable logistic regression model. Clinical (age at implant (years), gender, native bicuspid aortic valve, time since implant (months), valve size implanted (mm), and interactions between age and time since implant) and echocardiographic aortic root dimensions (including ratio of sinotubular junction to valve size implanted) were investigated in a multivariate model for correlation with mild or greater AR. Statistical analyses were performed using SAS<sup>®</sup> System statistical software (SAS Institute, Inc., Cary, NC, USA). A *p*-value <0.05 was considered to be statistically significant.

## Results

### Clinical follow up

The aggregate follow up for the group was 2,660 patient-years (pt-yr); mean follow up per patient was 6.0  $\pm$  2.5 years (range: 0 to 11.1 years). Follow up was available for 59 (87%) of 68 eligible patients at nine years, and for 19 (83%) of 23 eligible patients at 10 years. There were four early deaths (<30 days postoperative) (0.9%), and 76 late deaths (2.9% per pt-yr). Among valve-related deaths, two were early (0.4%) and 12 late (0.5% per pt-yr).

There were 17 cases of structural deterioration, of which 15 underwent explantation. Of the valves explanted, 14 had evidence of leaflet tear, and 10 of these occurred in the setting of dilation of the aortic sinotubular junction (25% increase versus size of valve implanted). Calcification was evident in five (29%) of 17 valves (one on echocardiographic imaging, one on operative inspection, and three on radiographic evaluation after explant); no calcification was evident in the remaining 12 valves (71%). Of the valves that were subjected to pathological inspection, calcium was described as more than focal in only one case. Freedom from explant due to structural valve dysfunction is shown in Figure 1; freedom from explant for structural failure at five years was 100%, and at nine years was 90.1% (95% CI: 84.3 to 96.0%). The average age of patients with structural deterioration was 52  $\pm$  12 years (range: 36 to 74 years; median 50 years). Freedom from explant for structural deterioration for patients aged  $\geq$ 65 years at the time of implantation is shown in Figure 2, and was 100% at both five and nine years. The late rate for bleeding was 0.2% per pt-y, thrombosis 0.0% per pt-yr, thromboembolism 1.2% per pt-yr, and endocarditis 0.4% per pt-yr.

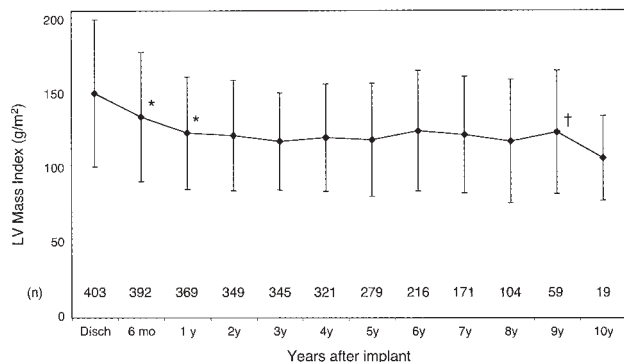


Figure 3: Left ventricular mass index decreased significantly from discharge to six months, and from six months to one year (\**p* <0.0001), with a small but significant increase from three years to nine years (†*p* = 0.002).

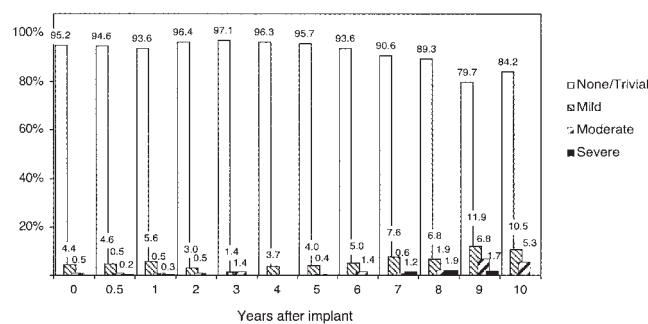


Figure 4: Prevalence and severity of aortic regurgitation increased from three years to nine years after surgery (*p* = 0.03).

Table I: Hemodynamic variables in relation to size of valve implanted, at one year and nine years after surgery.

Parameter	Valve size (mm)					
	21	23	25	27	29	All
<i>One year postoperative</i>						
No. of valves	17	46	95	131	105	394
Mean gradient (mmHg)	11.7±7.8	7.9±5.3	7.1±5.3	5.6±3.3	4.4±2.7	6.2±4.5
Peak gradient (mmHg)	23.5±15.5	15.8±9.4	13.9±9.8	11.0±6.5	8.5±5.1	12.1±8.7
EOA (cm <sup>2</sup> )	1.4±0.5	1.6±0.5	1.8±0.4	2.0±0.5	2.4±0.6	2.0±0.6
EOA-I (cm <sup>2</sup> /m <sup>2</sup> )	0.84±0.31	0.94±0.29	0.98±0.26	1.07±0.26	1.23±0.33	1.06±0.31
<i>Nine years postoperative</i>						
No. of valves	1	3	6	25	17	52
Mean gradient (mmHg)	7.0	5.3±4.6	4.1±2.1	5.9±5.0	3.7±3.0	5.0±4.1
Peak gradient (mmHg)	16.3	10.8±9.7	9.9±3.9	12.4±10.0	8.3±6.5	10.7±8.3
EOA (cm <sup>2</sup> )	1.2	1.8±1.0	2.3±0.5	2.3±0.7	2.6±0.8	2.4±0.7
EOA-I (cm <sup>2</sup> /m <sup>2</sup> )	0.72	1.08±0.58	1.30±0.30	1.22±0.38	1.34±0.42	1.25±0.39

Values are mean ± SD.

EOA: Effective orifice area; EOA-I: Effective orifice area index.

### Hemodynamics and LV mass index

Data reflecting hemodynamics through the eight-year postoperative period have been previously reported (4). Values for mean pressure gradient, peak pressure gradient, EOA and indexed EOA by valve size at one year and at nine years are listed in Table I. After initial decreases in gradients and an increase in EOA early after surgery, there was no significant change in mean gradient ( $\Delta = +0.7$  mmHg;  $p = 0.28$ ), EOA ( $\Delta = +0.1$  cm<sup>2</sup>;  $p = 0.32$ ), or indexed EOA ( $\Delta = +0.05$  cm<sup>2</sup>/m<sup>2</sup>;  $p = 0.36$ ) between one year and nine years postoperatively. There was a small, but statistically significant, increase in peak gradient between one year and nine years after surgery ( $\Delta = +3.1$  mmHg; 95% CI: 1.1 to 5.2 mmHg;  $p = 0.004$ ), though this was of doubtful clinical importance.

The LV mass index from discharge through 10-year

follow up is shown in Figure 3. As previously described (2), the LV mass index decreased significantly early after surgery. At three years postoperatively, the LV mass index had decreased to within the range of normal in 307 of 445 patients (69.0%) for whom data were available. Specifically, at three years, 200 (68.7%) of 291 men had a LV mass index >130 g/m<sup>2</sup>, and 107 (69.5%) of 154 women had a LV mass index >110 g/m<sup>2</sup>. From three to nine years postoperatively, there was a small but statistically significant increase in LV mass index for the group ( $\Delta = 13.8$  g/m<sup>2</sup>; 95% CI: 5.4 to 22.1;  $p = 0.002$ ).

### Aortic regurgitation

The presence and severity of AR through 10 years is shown in Figure 4. There was a statistically significant increase in AR at nine years compared with one year

Table II: Prevalence and location of mild (or greater) aortic regurgitation over time.

Time	n	Valvular	Paravalvular
Discharge	435	13 (3.0)	6 (1.4)
6 months	411	11 (2.7)	9 (2.2)
1 year	393	11 (2.8)	12 (3.1)
2 years	365	4 (1.1)	6 (1.6)
3 years	346	7 (2.0)	3 (0.9)
4 years	321	3 (0.9)	3 (0.9)
5 years	277	6 (2.2)	5 (1.8)
6 years	220	9 (4.1)	3 (1.4)
7 years	171	9 (5.3)	3 (1.8)
8 years	103	8 (7.8)	2 (1.9)
9 years	59	9 (15.3)	0 (0)
10 years	19	1 (5.3)	1 (5.3)

Values in parentheses are percentages.

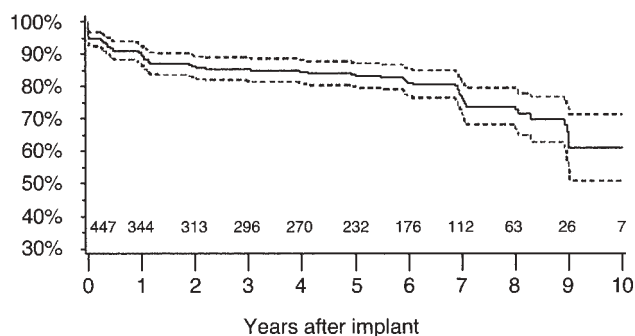


Figure 5: Freedom from mild (or greater) aortic regurgitation at five years was 83.6% (95% CI: 79.9 to 87.4%), and at nine years was 60.9% (95% CI: 50.7 to 71.1%). Numbers above the x-axis indicate patients at risk.

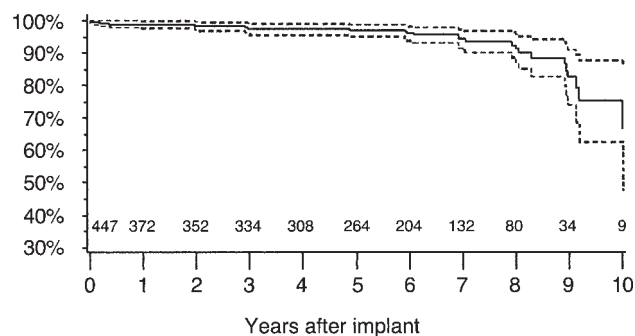


Figure 6: Freedom from hemodynamically significant ( $\geq$  moderate) aortic regurgitation at five years (96.9%; 95% CI: 95.1 to 98.7%) and nine years (82.5%; 95% CI: 74.1 to 91.0%). Numbers above the x-axis indicate patients at risk.

postoperatively ( $p = 0.03$ ). Freedom from AR (mild or greater), and freedom from hemodynamically significant AR (moderate or severe) are shown in Figures 5 and 6, respectively. Freedom from mild or greater AR at five years was 83.6% (95% CI: 79.9 to 87.4%), and at nine years was 60.9% (95% CI: 50.7 to 71.1%). Freedom from hemodynamically significant ( $\geq$  moderate) AR at five years was 96.9% (95% CI: 95.1 to 98.7%), and at nine years was 82.5% (95% CI: 74.1 to 91.0%).

The distribution of valvular versus paravalvular AR over time is shown in Table II. Although the prevalence of mild or greater paravalvular AR did not change over time, the prevalence of mild or greater valvular AR began to increase six years after surgery, and continued to increase through nine years.

Echocardiographically determined dimensions of the aortic root over the follow up period are shown in Table III. Using multivariable logistic regression analysis, significant predictors of mild or greater AR were valve size, months from implant, and ratio of sinotubular junction to valve size. The results of multivari-

able analysis for association with mild or greater AR are listed in Table IV. From the multivariate model, the odds of having mild or greater AR increase 8% for each year after implant, increase 40% for each 2 mm increase in the size of valve implanted, and increase 17% for each 0.1 increase in the ratio of sinotubular junction diameter to size of valve implanted. Progressive change in sinotubular junction diameter was not a significant predictor of AR in the multivariate model.

## Discussion

Stentless aortic bioprostheses represent an important alternative to mechanical and stented bioprosthetic valves for patients who require aortic valve replacement. They combine near-normal hemodynamics with features that may allow for improved durability compared with stented tissue valves. The Toronto SPV valve is a stentless porcine bioprosthesis that is scalloped for consistent subcoronary implantation. This

Table III: Aortic root dimensions over time after surgery.

Time	Annulus (mm)	Sinuses (mm)	STJ (mm)	Tubular (mm)
Discharge	23.2 $\pm$ 2.5 (110)	28.8 $\pm$ 4.4 (110)	28.2 $\pm$ 5.0 (106)	30.9 $\pm$ 5.7 (92)
6 months	23.5 $\pm$ 2.5 (86)	29.8 $\pm$ 4.5 (86)	29.0 $\pm$ 4.9 (86)	31.9 $\pm$ 6.3 (80)
1 year	23.5 $\pm$ 2.4 (69)	29.9 $\pm$ 4.8 (69)	28.4 $\pm$ 4.8 (68)	31.9 $\pm$ 5.7 (65)
2 years	23.4 $\pm$ 2.4 (94)	29.2 $\pm$ 4.2 (94)	27.5 $\pm$ 4.5 (93)	31.1 $\pm$ 5.3 (79)
3 years	23.6 $\pm$ 2.3 (122)	29.5 $\pm$ 4.4 (121)	27.6 $\pm$ 4.4 (119)	31.5 $\pm$ 6.1 (106)
4 years	23.6 $\pm$ 2.3 (166)	29.5 $\pm$ 4.0 (166)	27.5 $\pm$ 4.3 (162)	32.1 $\pm$ 5.5 (149)
5 years	23.6 $\pm$ 2.4 (190)	30.0 $\pm$ 3.9 (190)	28.3 $\pm$ 3.9 (185)	31.9 $\pm$ 5.8 (172)
6 years	23.7 $\pm$ 2.1 (186)	30.5 $\pm$ 3.9 (186)	28.9 $\pm$ 3.8 (182)	32.9 $\pm$ 5.5 (168)
7 years	23.8 $\pm$ 2.5 (160)	31.0 $\pm$ 4.5 (160)	29.3 $\pm$ 4.6 (154)	33.7 $\pm$ 6.0 (144)
8 years	22.9 $\pm$ 2.3 (93)	29.2 $\pm$ 4.5 (93)	27.4 $\pm$ 4.8 (87)	32.1 $\pm$ 6.4 (81)
9 years	22.8 $\pm$ 1.9 (54)	30.4 $\pm$ 3.8 (54)	28.5 $\pm$ 3.9 (54)	34.8 $\pm$ 5.5 (50)
10 years	22.3 $\pm$ 2.1 (18)	27.9 $\pm$ 4.1 (18)	26.7 $\pm$ 3.7 (18)	33.2 $\pm$ 6.2 (17)

Values in parentheses are numbers of measurements available.  
STJ: Sinotubular junction.

Table IV: Significant predictors of aortic regurgitation in multivariate analysis.

Parameter	Estimate	Odds Ratio	p-value
Months after implant	0.00641	1.006	0.02
Valve size	0.1684	1.183	<0.0001
Ratio of STJ (mm) to valve size implanted (mm)	1.5965	4.936	0.03

STJ: Sinotubular junction.

allows for replacement of only the diseased tissue among patients with isolated valve disease. However, like the native aortic valve, the Toronto SPV valve is reliant on the native aortic root for structural support, and may become incompetent if there is significant aortic root dilation, especially at the sinotubular junction. At one implantation center, sinotubular dilation has been described as a major determinant of late AR and structural valve failure (5). Because favorable hemodynamics have already been well established for the Toronto SPV valve, its utility now will be largely influenced by measures of durability.

#### Late hemodynamics and LV mass regression

The Toronto SPV valve has excellent hemodynamics (1-4). In the present study, there were no significant changes in mean gradient or EOA through nine years. Although peak pressure gradient increased late after surgery, the increase was small and of doubtful clinical relevance. Because peak gradient is especially susceptible to LV contractility and cardiac output, previously described on-going improvement in LV performance late after surgery (4) could be responsible for the observed small increase in peak gradient without significant changes in mean gradient or EOA. Rapid and often complete regression of LV hypertrophy has been previously reported with the Toronto SPV valve - evidence of effective relief of LV outflow obstruction and favorable LV remodeling after aortic valve replacement. A small but statistically significant late increase in LV mass index was observed in the present study, though in light of the preserved low gradients and large EOA it is likely that other factors may have contributed to this. Specifically, hypertension could have contributed to a late increase in LV mass index, independent of valve hemodynamics.

#### Valve durability

In the present study, freedom from explant for structural valve deterioration was 90.1% (95% CI: 84.3 to 96.0%) at nine years. This compared favorably with data for the Hancock-MO valve (79 ± 3% (SEM) freedom from structural deterioration and 77 ± 2% freedom from explant at 10 years) (12). The data were also similar to reported data for the Carpentier-Edwards

Pericardial valve (94% freedom from explant for structural deterioration at 10 years) (13) and the Hancock-II (97 ± 1% freedom from structural deterioration and 94 ± 1% freedom from reoperation at 10 years) (14). Similar to reported findings with other bioprostheses (12,13), freedom from structural failure in the present study was better among older patients (100% freedom from reoperation for structural valve deterioration at nine years among patients aged ≥65 years at the time of implantation).

Bioprostheses typically fail due to leaflet calcification (15). However, a previous report suggested that mechanisms of structural failure other than calcification are associated with the Toronto SPV valve. A review of structural failure of the Toronto SPV valve at seven years found that five of eight cases occurred due to aortic root dilation, and the remaining three to commissural tears (6). At the time of writing, symptomatic calcific stenosis of the Toronto SPV valve remains rare (16). In the present study, the absence of tissue calcification as a cause of failure continued through nine years. Specifically, only five of 17 (29%) valves with structural deterioration at nine years had any evidence of calcification, and calcification was regarded as more than focal in only one valve among 17 (6%). As in the previous report (6), the preponderance of structural failure in the present study was due to leaflet tear, typically in the setting of sinotubular dilation. Although stentless bioprostheses may eventually fail due to leaflet calcification, this does not appear to pose a significant problem through a nine-year follow up.

#### Determinants of AR

A previously published report demonstrated a strong association between dilation of the ascending aorta (and especially the sinotubular junction) and the development of moderate or greater AR (5). In the present study, there were few cases of moderate or severe AR, and attempts to correlate these cases with aortic root dilation were unsuccessful. However, mild AR was more prevalent among patients late after Toronto aortic valve replacement. Factors that correlated in multivariate logistic regression analysis with the presence of mild or greater AR included duration after implantation, larger valve size, and an increase in the

ratio of sinotubular junction to size of valve implanted.

Increasing prevalence of AR is anticipated with increasing duration after surgery. Although one study reported a trend toward greater structural failure associated with small-sized stented aortic bioprostheses (13), structural failure is usually greater for the larger mitral than the smaller aortic bioprostheses (14,17), possibly related to increased tissue fatigue. However, it is not clear whether the mechanism of failure is the same for larger stentless as for larger stented bioprostheses. Although there may be greater tissue fatigue associated with both types of bioprosthesis, stentless - but not stented - bioprostheses are also affected by the geometry of the aortic root, which apparently serves as a competing mechanism of failure. Preoperative measurements of the sinotubular junction and tubular ascending aorta are not available for analysis in the present study. However, in keeping with sizing practices for the Toronto SPV valve in the early 1990s (sizing the valve to the sinotubular junction), it is possible that larger valve sizes were implanted in patients with a larger aortic root (and not just a larger aortic valve annulus). As such, it is possible that the higher prevalence of AR among patients implanted with a larger Toronto SPV valve is a marker for intrinsic aortic root disease rather than a measure of valve dysfunction. That progressive change in diameter of the sinotubular junction was not an independent predictor of AR may be a reflection of the variety of causes of AR in this multicenter study.

### Study limitations

The present study was an observational analysis of data acquired from a large patient cohort prospectively followed in a multicenter study. Data reflecting the presence of hypertension and adequacy of antihypertensive therapy during follow up were not collected, and the impact of blood pressure on late LV mass index was not known. Aortic root measurements were performed retrospectively for echocardiograms performed before 2001, after the impact of aortic root dilation had been reported (5). Because not all echocardiograms were submitted for re-analysis, the data were less complete than for prospectively measured variables of gradients, EOA, LV mass index and AR. Patients who underwent reoperation (including those in whom the valve was explanted due to structural valve failure) did not all undergo echocardiographic imaging for aortic root measurements performed prior to explant. As such, an analysis of the interaction between aortic root geometry and AR may have excluded some patients with the most significant AR. The low prevalence of hemodynamically significant AR among the study population may have contributed to the inability to correlate moderate or severe

AR with aortic root geometry. Finally, native bicuspid valve was not significantly related to the presence of late AR, although progressive root dilation would be expected to be more significant in this patient subset (7). It is possible that reliable identification of functional bicuspid valves was difficult at the time of surgical intervention owing to a high prevalence of heavy native valve calcification.

*In conclusion*, in a multicenter study with follow up of 2,660 pt-yr, the Toronto SPV valve showed excellent preservation of hemodynamics at nine years. Measures of durability were good, with 90% overall freedom from explant for structural failure at nine years, and 100% freedom from explant for structural failure at nine years among patients aged  $\geq 65$  years at the time of implantation. Freedom from hemodynamically significant ( $\geq$  moderate) AR was 96.9% at five years and 82.5% at nine years; the presence of more than mild AR was associated with time since implant, a larger valve size, and dilation of the sinotubular junction relative to the size of valve implanted.

### References

1. David TE, Feindel CM, Scully HE, Bos J, Rakowski H. Aortic valve replacement with stentless porcine aortic valves: A ten-year experience. *J Heart Valve Dis* 1998;7:250-254
2. Bach DS, David T, Yacoub M, et al. Hemodynamics and left ventricular mass regression following implantation of the Toronto SPV valve. *Am J Cardiol* 1998;82:1214-1219
3. Goldman B, Christakis G, David T, et al. Will stentless valves be durable? The Toronto valve (TSPV) at 5 to 6 years. *Semin Thorac Cardiovasc Surg* 1999;11(suppl.1):42-49
4. Bach DS, Goldman B, Verrier E, et al. Eight year hemodynamic follow-up after aortic valve replacement with the Toronto SPV stentless aortic valve. *Semin Thorac Cardiovasc Surg* 2001;13(suppl.1):173-179
5. David TE, Ivanov J, Eriksson MJ, Bos J, Feindel CM, Rakowski H. Dilation of the sinotubular junction causes aortic insufficiency after aortic valve replacement with the Toronto SPV bioprosthesis. *J Thorac Cardiovasc Surg* 2001;122:929-934
6. Shargall Y, Goldman B, Christakis G, David T. Analysis of explants and causes of mortality during long-term follow-up of the Toronto stentless porcine valve. *Semin Thorac Cardiovasc Surg* 2001;13(suppl.1):106-112
7. Keane MG, Wiegers SE, Plappert T, Pochettino A, Bavaria JE, St. John Sutton MG. Bicuspid aortic valves are associated with aortic dilatation out of proportion to coexistent valvular lesions.

- Circulation 2000;102(suppl.III):III-35-III-39
8. Edmunds LH, Clark RE, Cohn LH, et al. Guidelines for reporting morbidity and mortality after cardiac valvular operations. *Ann Thorac Surg* 1988;46:257-259
  9. Feigenbaum H. Hemodynamic information derived from echocardiography. In: Feigenbaum H, ed. *Echocardiography*, 5th edition. Lea & Febiger, Philadelphia, 1994:181-215
  10. Devereux RB, Alonso DR, Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Am J Cardiol* 1986;57:450-458
  11. Perry GJ, Helmcke F, Nanda NC, et al. Evaluation of aortic insufficiency by Doppler color flow mapping. *J Am Coll Cardiol* 1987;9:952-959
  12. Cohn LH, Collins JJ, Jr., Rizzo RJ, Adams DH, Cooper GS, Avanki SF. Twenty-year follow-up of the Hancock modified orifice porcine aortic valve. *Ann Thorac Surg* 1998;66:S30-S34
  13. Banbury MK, Cosgrove DM, III, White JA, Blackstone EH, Frater RWM, Okies JE. Age and valve size effect on the long-term durability of the Carpentier-Edwards aortic pericardial prosthesis. *Ann Thorac Surg* 2001;72:753-757
  14. David TE, Ivanor J, Armstrong S, Feindel CM, Cohen G. Late results of heart valve replacement with the Hancock II bioprosthesis. *J Thorac Cardiovasc Surg* 2001;121:268-278
  15. Schoen FJ, Levy RJ. Bioprosthetic heart valve failure: Pathology and pathogenesis. *Cardiol Clin* 1984;2:717-739
  16. Tansley PDT, Sheppard MN, Pepper J. Symptomatic calcific stenosis of a Toronto stentless porcine valve. *Eur J Cardiothorac Surg* 2000;17:763-765
  17. Glower DD, White WD, Hatton AC, et al. Determinants of reoperation after 960 valve replacements with Carpentier-Edwards prostheses. *J Thorac Cardiovasc Surg* 1994;107:381-392

*Appendix I: Details of the study sites and clinical investigators.*

Study site Principal investigator	Location
Toronto General Hospital Canada	Toronto, Ontario, Tirone David MD
Sunnybrook Medical Center Canada	Toronto, Ontario, Bernard Goldman MD
University of Washington USA	Seattle, Washington, Edward Verrier MD
Medical Center St. Thomas Hospital USA	Nashville, Tennessee, Michael Petracek MD
Queen Elizabeth II Health Canada	Halifax, Nova Scotia, Jeremy Wood MD
Sciences Centre Lankenau Hospital Pennsylvania,	Wynnewood, Scott Goldman MD USA

## Meeting discussion

**SIR MAGDI YACOUB (UK):** These are very interesting, provocative data which seem to suggest that the sinotubular junction was absolutely stable with time. So the relationship between the sinotubular junction and size of valve implanted was present at the time of implantation. Am I right or wrong?

**DR. DAVID S. BACH (Ann Arbor, Michigan, USA):** I apologize if that was not clear. The increase in ratio of sinotubular junction to size of valve implanted did occur over time, but only in a small number of people.

**MR. YACOUB:** It didn't affect the statistics?

**DR BACH:** No. it did not.

**DR. TIRONE E. DAVID (Toronto, Canada):** In our sample size, this phenomenon occurred in 11% of 197 patients who were studied for up to 10 years.

**MR. YACOUB:** And it was masked completely?

**DR. DAVID:** By the sample size, but I think it remains a problem.

**MR. YACOUB:** Can you show it to us in the small subset of patients and can you define the predictors for this to happen? How often to start with - 1 or 2%? If it was higher than that we would have seen it in the statistics, but we haven't.

**DR. BACH:** Correct. It was in a relatively small percentage or it would have shown up in the whole group. I did not analyze a threshold of how many dilated by 'x' amount. We treated the ratio as a contin-

uous variable. One disadvantage of a multicenter trial may be that it washes out the clinical experience of a single center. One of the most active surgeons in the group routinely banded the sinotubular junction.

**MR. YACOUB:** At the time, was it a continuous dilation or a quick dilation early or late. Do you have these data - and are they reliable?

**DR. BACH:** I will quote Dr. David's more reliable data which show that it is a progressive, slow change over time, not a precipitous one.

**MR. YACOUB:** Continuous, and as time goes by, it will be huge?

**DR. BACH:** As time goes by, it will reach a threshold so that it causes AI and structural valve failure. It doesn't happen in all patients, and when it does - unlike aortic dissection - it is not a precipitous event.

**MR. YACOUB:** It might be that we don't see it because we don't look for it - but we are not aware of it.

**DR. DAVID:** But, Mr. Yacoub, would you agree that when you replace an aortic valve, and follow the patients longitudinally, a number do develop ascending aortic aneurysm - in our hands, 4%. We now have about 2,000 patients on the database. We are specifically interested in bicuspid aortic valve, for which 14% of patients returned for replacement of the ascending aorta within 20 years. These patients develop an ascending aortic aneurysm - the wall tension ultimately goes down the sinotubular junction, which might be dilated. But I am not saying that bicuspid aortic valve is a marker.

**MR. YACOUB:** When we looked at bicuspid aortic valves, it was not a marker - and you confirmed that. It is only the patient who has had a coarctation repaired in the presence of a bicuspid valve that is a risk factor.

**DR. BACH:** We included bicuspid aortic valve as one of the variables to try to predict progression of AI, but it was not a marker for this.

**DR. THANOS SIORIS (Canada):** Did you see any long-term effects if the aortotomy incision was curved towards the annulus in the non-coronary sinus and you perhaps used autologous pericardium, especially in very small roots to facilitate exposure?

**DR. BACH:** That surgical detail was not within the database. How the aortotomy was performed was not an evaluated variable because I didn't have access to that data.

**DR. W. R. ERIC JAMIESON (Canada):** If you use one of the three root formulations and keep the non-coronary sinus intact, can you prevent this problem?

**DR. BACH:** Perhaps.