Dilation of the sinotubular junction causes aortic insufficiency after aortic valve replacement with the Toronto SPV bioprosthesis

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Objective: This study was undertaken to examine the causes of late aortic insufficiency in patients who had aortic valve replacement with the Toronto SPV bioprosthesis (St Jude Medical, Inc, St Paul, Minn).

Methods: From 1991 to 1996, 174 patients with a mean age of 63 ± 11 years underwent aortic valve replacement with the Toronto SPV bioprosthesis and were evaluated annually by Doppler echocardiographic studies to assess valve function. The diameters of the aortic root were retrospectively measured in all patients who had aortic insufficiency and also in a random sample of 23 patients without aortic insufficiency. The mean follow-up was 5.8 years (range 4 to 9 years).

Results: Aortic insufficiency greater than 1+ developed in 19 patients. The diameter of the sinotubular junction increased in these patients and did not change in those without aortic insufficiency. The ratio between the diameter of the sinotubular junction and the size of the Toronto SPV bioprosthesis increased in patients who had aortic insufficiency and did not change in those without aortic insufficiency. Both 2-way analysis of covariance and analysis by a mixed linear model demonstrated a significant difference in slopes between the patients with aortic insufficiency greater than 1+ and in those without insufficiency for the ratio of the diameter of the sinotubular junction/diameter of the Toronto SPV relationships over time (aortic insufficiency $\cdot$ Year; $P < .001$). Structural valve deterioration was observed in 5 valves, and in 4 of them the sinotubular junction of the aortic root had dilated. The freedom from structural valve deterioration was $99\% \pm 1\%$ for patients without aortic insufficiency and $82\% \pm 12\%$ for those with aortic insufficiency of more than 1+ at 8 years ($P = .004$). One patient had moderate aortic insufficiency without structural valve deterioration and dilation of the sinotubular junction.

Conclusions: Dilation of the sinotubular junction causes aortic insufficiency after aortic valve replacement with the Toronto SPV bioprosthesis and increases the risk of structural valve deterioration. Banding the sinotubular junction may prevent dilation and enhance the durability of this valve.

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Dilation of the sinotubular junction causes aortic insufficiency (AI) in patients with normal aortic cusps.^{1,2} This is the mechanism of AI in patients with ascending aortic aneurysm and anatomically normal aortic cusps and the reason replacement of the ascending aorta with proper reduction in the diameter of the sinotubular junction corrects AI in these patients.^{1,3,4} Dilation of the sino-

tubular junction occurs in patients with diseased media of the ascending aorta such as those with bicuspid aortic valve, systemic hypertension, and in some older patients.^{5,6}

Aortic valve replacement (AVR) with stentless biologic valves (pulmonary autograft, aortic valve homograft, and porcine aortic valves) is performed by implanting the donor valve in the subcoronary position of the recipient's aortic root or by using it as an aortic root either inside the native aortic root (inclusion technique) or as a full aortic root replacement.¹ When the technique of subcoronary implantation is used, it is extremely important that the diameter of the sinotubular junction of the recipient's aortic root be similar to that of the donor valve at the end of the procedure.¹ Although intraoperative correction of the diameter of the sinotubular junction of the recipient's aortic root allows for proper coaptation of the cusps and valve competence, there is no guarantee that the diameter of the sinotubular junction will remain unchanged for the life of the stentless biologic valve. Progressive dilation of the sinotubular junction pulls the cusps apart, increases the mechanical stress, and may cause premature failure of the stentless valve.

This study examines the correlation between dilation of the sinotubular junction and AI after AVR with the Toronto SPV bioprosthesis (St Jude Medical, Inc, St Paul, Minn).

Patients and Methods

From July 1991 to June 1996, 174 patients underwent isolated AVR with the Toronto SPV bioprosthesis at Toronto General Hospital as part of a multicenter, observational trial sponsored by St Jude Medical to obtain Food and Drug Administration approval for this valve. The Toronto SPV bioprosthesis was approved late in 1997, but all patients have continued to be monitored annually with clinical evaluations and Doppler echocardiographic studies.

The selection of size of the Toronto SPV bioprosthesis was based on the diameter of the sinotubular junction. If the diameter of the sinotubular junction was more than 2 mm larger than the diameter of aortic anulus, this valve was not used in this trial.

Bioprosthetic aortic valve function was assessed by transthoracic Doppler echocardiography 1 week after the operation, 3 to 6 months postoperatively, and annually thereafter. Peak and mean systolic flow velocity across the aortic valve was recorded with continuous-wave Doppler ultrasonography and also proximal to the aortic valve with pulsed-wave Doppler ultrasonography. The diameter of the left ventricular outflow tract was measured in midsystole from the parasternal long-axis view to calculate the effective aortic valve area and cardiac output. AI was assessed by color flow Doppler, continuous-wave Doppler, and pulsed-wave Doppler ultrasonography. The echocardiographic grading of AI was as follows: 0 = none; 1+ = trace; 2+ = mild; 3+ = moderate; and 4+ = severe. All studies were recorded on individual tapes in chronologic sequence. The diameters of the sinotubular junction and aortic anulus were measured retrospectively in all studies of all patients in whom AI of more than 1+ developed. A random sample of patients without AI with follow-up of the same duration or longer had those diameters measured for comparison.

Follow-up extended from 4 to 9 years, mean of 5.8 years, and was complete.

Statistical Analysis

The primary end point for the study was the development of AI of more than 1+. Secondary end points included late survival and freedom from adverse events such as structural valve deterioration, reoperation, thromboembolic events, and prosthetic valve endocarditis.

SAS 6.12 for Windows was used for all statistical analyses (SAS Institute, Inc, Cary, NC). Continuous variables are reported as the mean $\pm$ 1 SD in tables and text and as the mean $\pm$ 1 SE in figures unless otherwise noted.

Baseline and operative comparisons between groups (AI>1+ vs no AI) for continuous variables were performed with the use of unpaired *t* tests and the χ^2 or Fisher exact test (where appropriate) for categorical variables. Late survival and freedom from adverse events were estimated by the nonparametric Kaplan-Meier method. The log-rank test was used for statistical comparisons of the Kaplan-Meier curves. There were too few events to develop a stable multivariable Cox model for predictors of AI more than 1+.

Evaluations of echocardiographic diameters were made by analysis of covariance testing the main effects, AI group, year, and the interaction of AI group times year. For statistical corroboration we also used the SAS procedure PROC MIXED to test the same main effects. A 1-way analysis of variance was used to test differences in echocardiographic diameters between AI grades.

Results

Table 1 shows the clinical profile and operative data of the patients in this study.

Aortic Insufficiency

AI of more than 1+ developed in 19 (11%) of 174 patients during the follow-up period. Mild AI (2+) developed in 13 patients, moderate AI (3+) developed in 4, and severe AI (4+) developed in 2 patients. In 80% of patients in whom AI of more than 1+ developed, it did so during the first 4 years of follow-up. Ninety-four percent of the entire patient sample has been followed up for longer than 4 years. Table 2 shows the clinical profile of patients with AI of more than 1+ and of the random patients without AI used for comparison. Table 3 depicts the mean $\pm$ SD of the various diameter ratios associated with each AI grade. There was a significant difference between the AI grades by 1-way analysis for all diameters and ratios with the exception of the aortic anulus diameter.

Figure 1 shows the changes in the diameter of the sinotubular junction in patients with AI of more than 1+ and in those without AI. This relationship showed a significant difference in slopes by analysis of covariance ($P < .001$). Analysis of covariance also revealed a significant difference in slope between the groups with AI of more than 1+ and the non-AI groups over time for sinotubular junction/aortic anulus (AI $\cdot$ Year, $P = .031$) and for sinotubular junction/valve

TABLE 1. Clinical profile of patients with the Toronto SPV bioprosthesis

No. of patients	174
Age (y)	
Mean $\pm$ SD	63 $\pm$ 11
Range	32-80
Sex	
Male	120 (69)
Female	54 (31)
NYHA functional class	
I	14 (8)
II	77 (44)
III	66 (38)
IV	17 (9)
Associated diseases	
Hypertension	66 (38)
Diabetes mellitus	21 (12)
Peripheral vascular disease	7 (4)
Infective endocarditis	
Active	0
Healed	4 (2)
Previous aortic valve surgery	6 (3)
Aortic valve lesion	
Stenosis	135 (78)
Insufficiency	13 (9)
Mixed	24 (14)
Left ventricular ejection fraction	
>59%	76 (44)
40%-59%	73 (42)
20%-39%	23 (13)
<20%	2 (1)
Coronary artery disease	55 (32)
Aortic valve disease	
Tricuspid calcific	84 (48)
Bicuspid	58 (33)
Rheumatic	9 (5)
Prosthetic valve dysfunction	6 (3)
Undetermined	17 (10)
Toronto SPV bioprosthesis	
Size 21 mm	3 (2)
Size 23 mm	14 (8)
Size 25 mm	43 (25)
Size 27 mm	62 (35)
Size 29 mm	52 (30)
Coronary artery bypass	55 (32)
Aortic clamping time (min)	85 $\pm$ 23
Cardiopulmonary bypass time (min)	106 $\pm$ 29

NYHA, New York Heart Association. Percentages are shown in parentheses.

size (AI $\cdot$ Year, $P < .001$). Analysis by a mixed model corroborated that there was a significant difference in the slope of the sinotubular junction/valve size relationships over time (AI $\cdot$ Year, $P = .001$) and a significant difference between the AI groups for the sinotubular junction/aortic anulus relationship ($P < .001$).

TABLE 2. Clinical profile of patients with AI of more than 1+ and random patients without AI

	AI >1+	No AI	Pvalue
No. of patients	19	23	
Mean age (y)	59 $\pm$ 11	64 $\pm$ 9	.1
Bicuspid aortic valve	4 (21)	8 (35)	.3
Aortic valve lesion			
Stenosis	14 (74)	20 (87)	
Insufficiency	3 (16)	1 (4)	
Mixed	2 (10)	2 (9)	.4
Hypertension	4 (21)	7 (30)	.4
Coronary artery disease	15 (79)	16 (70)	.6
Mean follow-up (y)	5.2 $\pm$ 2.5	7.5 $\pm$ 0.9	.001

TABLE 3. Doppler echocardiographic diameters by AI grade

Diameter (cm)	AI 0	AI 1+	AI 2+	AI 3+	Pvalue*
Aortic anulus	2.09 $\pm$ 0.2	2.09 $\pm$ 0.2	2.15 $\pm$ 0.1	2.28 $\pm$ 0.3	.09
Sinus	2.83 $\pm$ 0.3	2.91 $\pm$ 0.4	2.98 $\pm$ 0.2	3.30 $\pm$ 0.4	.003
STJ	2.61 $\pm$ 0.3	2.72 $\pm$ 0.3	2.83 $\pm$ 0.3	3.25 $\pm$ 0.4	<.001
Ascending aorta	3.17 $\pm$ 0.5	3.66 $\pm$ 1.3	3.48 $\pm$ 0.4	3.90 $\pm$ 0.6	<.001
STJ/AA	1.25 $\pm$ 0.1	1.30 $\pm$ 0.1	1.31 $\pm$ 0.1	1.43 $\pm$ 0.1	<.001
STJ/AV size	0.98 $\pm$ 0.1	0.99 $\pm$ 0.1	1.04 $\pm$ 0.1	1.16 $\pm$ 0.1	<.001

Values are mean $\pm$ SD. AI, Aortic insufficiency grade; STJ, sinotubular junction; STJ/AA, ratio of STJ/aortic anulus; STJ/AV size, ratio of STJ/size of aortic valve prosthesis. AI 4+ developed in only 2 patients; therefore, their estimates are not given.

*Pvalue for 1-way analysis of variance for differences between AI grades.

No predictors of dilation of the sinotubular junction could be identified.

Clinical Outcomes

No operative deaths occurred, but there were 21 deaths during the follow-up period: 2 valve-related (1 endocarditis and 1 stroke), 3 cardiac (1 sudden, 1 myocardial infarction, and 1 right-sided heart failure), and 16 due to other causes, mostly malignancy. The survival was $82\% \pm 4\%$ at 8 years, as illustrated in Figure 2. There were 13 thromboembolic events: 7 strokes and 6 transient ischemic attacks. The freedom from thromboembolic events was $92\% \pm 2\%$ at 8 years.

There were 2 cases of prosthetic valve endocarditis, 1 early and 1 late in a patient with AI of more than 1+. Both patients were reoperated on; the patient with early endocarditis died. The freedom from prosthetic valve endocarditis was $98\% \pm 1\%$ at 8 years.

Structural valve deterioration developed in 5 patients. Four patients with documented structural valve deterioration had dilation of the sinotubular junction. All 5 patients were successfully reoperated on and found to have tears in one or more aortic cusps. The freedom from structural valve deterioration at 8 years was $93\% \pm 4\%$ for all

Figure 1. Changes in the diameter of the sinotubular junction in patients with AI of more than 1+ (AI>1+) and in those without AI (No AI). Analysis of covariance, AI · Year, $P < .001$.

Figure 3. Freedom from structural valve deterioration (SVD) in patients with AI of more than 1+ (AI>1+) and in those without AI (No AI).

Figure 2. Survival after AVR with the Toronto SPV bioprosthesis.

Figure 4. Freedom from reoperation in patients with AI of more than 1+ (AI>1+) and in those without AI (No AI).

patients, $99\% \pm 1\%$ for those without AI and $82\% \pm 12\%$ for those with AI of more than 1+ ($P = .004$), as shown in Figure 3.

Eight patients have required repeat AVR: 2 for endocarditis, 5 for primary tissue failure, and 1 for AI due to dilation of the sinotubular junction without primary tissue failure. The freedom from repeat AVR at 8 years was $92\% \pm 4\%$ for all patients, $97\% \pm 12\%$ for those without AI, and $82\% \pm 12\%$ for those with AI of more than 1+ ($P = .02$), as shown in Figure 4.

At the last follow-up contact, 153 patients were alive: 81% were asymptomatic, 19% were in New York Heart Association functional class II, and no patient was in class III or IV. No patient was taking warfarin sodium, and none has had hemorrhagic complications.

Discussion

AVR with stentless porcine aortic valves has provided excellent clinical outcomes.⁷⁻⁹ The hemodynamic performance of these valves has also been excellent and appears to be better than that of stented porcine aortic valves and similar to that of aortic valve homografts.^{10,11} Indeed, most patients have normal or near normal left ventricular mass and function at 5 years postoperatively.⁷ These findings may explain the apparent advantage in late survival after AVR with stentless valves.^{9,12} We compared the survival after AVR with stented and stentless valves in a case-match study and found better survival in patients with stentless valves.⁹ The reduction in late deaths was largely due to fewer cardiac deaths in the stentless group.⁹ Coincidentally, in this present series there were 21 late deaths but only 5 were cardiac or

valve-related. These observations have been the main impetus for us to continue using stentless biologic valves, particularly in patients with impaired left ventricular function.

The Toronto SPV bioprosthesis has been used clinically only for a decade. This valve has exceptionally good hemodynamics.⁷ However, we found that AI of more than 1+ developed in 11% of our patients during the first 8 years of follow-up. Because dilation of the sinotubular junction causes AI in patients with normal aortic valve cusps,¹⁻⁴ we reviewed all echocardiograms on the 19 patients in whom AI of more than 1+ developed and measured the diameters of the aortic anulus, aortic sinuses, sinotubular junction, and ascending aorta. These diameters were compared with those of a random sample of 23 patients without AI, as shown in Table 3. The development of AI correlated with dilation of the sinotubular junction in most patients. We believe that dilation of the sinotubular junction preceded the development of AI, because the aortic cusps had no evidence of degeneration in any of the patients with AI graded as 2+ and in 1 of the patients with 3+ AI. We could not identify any factor predictive of dilation of the sinotubular junction, probably because of the relatively small number of patients. We expected to find that dilation of the sinotubular junction would have been more common in patients with bicuspid aortic valve disease, but this was not the case. Systemic hypertension was not a predictor either.

Dilation of the sinotubular junction pulls the commissures of the aortic valve apart, preventing the cusps from coapting, and causes central AI. The native aortic valve does not become incompetent with minor dilation of the sinotubular junction because the cusps are elastic and compensate for the increase in distance between the commissures. However, glutaraldehyde-fixed aortic cusps are relatively inelastic and cannot compensate for the dilation. The lack of coaptation of the cusps caused by the dilation of the sinotubular junction increases the mechanical stress with resulting premature valve failure. Indeed, among 5 patients reoperated on for structural valve deterioration, gross dilation of the sinotubular junction was present in 4. One additional patient was reoperated on because of moderate AI due to dilation of the sinotubular junction, and the aortic cusps were intact.

Dilation of the sinotubular junction is not specific to AVR with the Toronto SPV bioprosthesis. Jin and Westaby¹³ described an identical problem after AVR with the Medtronic Freestyle valve (Medtronic, Inc, Minneapolis, Minn) and noted progressively more severe AI as the sinotubular junction diameter increased postoperatively. Dilation of the sinotubular junction also occurs after aortic root replacement with pulmonary autografts.⁵ We believe we have resolved the problem of dilation of the sinotubular junction of the pulmonary autograft in the aortic position by banding the sinotubular junction with a strip of synthetic

fabric. Hence, banding of the sinotubular junction in patients who have AVR with the Toronto SPV bioprosthesis should prevent dilation of the sinotubular junction. A strip of polyester fabric 4 to 5 mm wide should be used to band the aorta at the level of the sinotubular junction and should be secured to the posts of the Toronto SPV valve with interrupted sutures to avoid its migration. The diameter of this band should be identical to that of the valve implanted. Banding of the sinotubular junction may enhance the durability of the Toronto SPV bioprosthesis by preventing dilation of the sinotubular junction.

AVR with stentless valves is a technically more demanding operation than AVR with stented valves. When a stentless valve is used, many surgeons believe that a full root replacement is preferable to reduce the risk of postoperative AI, regardless of the type of stentless biologic valve used.¹⁴⁻¹⁷ Although this may be so, we believe that aortic root replacement should be reserved for patients who have aortic root disease. The majority of older patients who need AVR have normal or near normal aortic root geometry and only the cusps are diseased. If a stentless porcine valve is used in these patients, the technique of subcoronary implantation is probably the safest one. Surgical removal of the Toronto SPV valve is not more complex than removal of any stented aortic valve. Seven of 8 patients who have required repeat AVR were operated by us without operative death or serious complication. To re-replace an aortic root is a far more complex operation.

Limitations of the Study

The number of patients in whom AI developed is relatively small in this study. Because moderate or severe AI developed in only 6 patients, we had to include those who had mild AI to demonstrate that dilation of the sinotubular junction is the principal cause of late postoperative AI. In addition, we could not prove that dilation of the sinotubular junction was the cause of AI, and it is conceivable, albeit unlikely, that AI caused dilation of the sinotubular junction.

In summary, 11% of patients who underwent AVR with the Toronto stentless porcine valve had AI of more than 1+. AI was strongly associated with dilation of the sinotubular junction, resulting in an increased risk of premature valve failure and reoperation. Banding the sinotubular junction may prevent dilation and enhance the durability of this valve.

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Discussion

Dr Neal Kon (Winston-Salem, NC). Dr David has been a pioneer in the use of stentless porcine aortic valves. He introduced them into his clinical practice in the early 1980s and demonstrated their huge hemodynamic advantage. He later showed that these hemodynamic benefits result in significant left ventricular mass reduction, which translated into improved quality of life and survivals for his patients. Dr David has also taught us through his work with aortic root remodeling that the aortic valve consists not only of aortic valve leaflets, but also the aortic anulus, the sinuses of Valsalva, and the sinotubular junction. It is the appropriate geometric relationship of all these components that results in a competent aortic valve.

When using a subcoronary implant technique, achieving these geometric relationships perfectly can be difficult. It is necessary to replace just the leaflets, so one must now place new porcine cusps appropriately in a human foreign root. Even if one does this perfectly, which can be difficult, the aortic root, and particularly the sinotubular junction, has a tendency to change with time. Dr David told me that every 10 years the sinotubular junction dilates about 2 mm.

Eleven percent of the patients in this study had mild or greater AI. The development of significant AI was associated with dilation of the sinotubular junction.

Dr David has shown us a potential problem with the subcoronary implantation technique, which may lead to premature valve failure. He also proposes a cure, the cure being placing a Dacron band around the sinotubular junction at the time of implantation to prevent dilatation. I agree with this problem but I am not sure I agree with the cure.

We have used a short aortic root replacement, which is probably better called a *total AVR*, as the technique of choice for stentless valve implantation. We use this technique regardless of the presence or absence of root disease.

In 7 years of follow-up, no patient has had more than trivial AI, and there has been no increase in gradients over time. Also, the sinotubular junction, the aortic anulus, and the sinus of Valsalva measurements do not change with time when a short root replacement is done.

Dr David, I have 2 questions for you. First, why not use a short root replacement technique instead of banding the sinotubular junction when the life expectancy of the patient is less than 15 years and when the native aortic root is not symmetrical? Do you predict that the more perfectly you implant the stentless valve, since this can be variable with stentless valves, the better the durability will be?

Second, when a surgeon slightly oversizes a stentless valve and uses a subcoronary technique, allowing a little extra leaflet coaptation on the chance that AI might develop, will that valve fail prematurely as well?

Dr David. Thank you, Dr Kon. I think your comments are very appropriate and I appreciate your questions. I do not know whether aortic root replacement or subcoronary implantation is better when a stentless porcine valve is used. Being a student, I will continue to look into this matter with interest. There are not enough data in the literature on operative mortality for aortic root re-replacement. My bet is that mortality is not 1% or 2%. If it is 20% or 30%, then implantation of a stentless valve in the subcoronary position would be safer as the first procedure because the operative risk of replacing these valves is low when performed electively. Even if a Dacron band were placed on the sinotubular junction, it would not be difficult to reoperate on these patients.

A porcine xenograft aortic root may function very well when used for aortic root replacement. However, this is not always the case when an aortic homograft or pulmonary autograft is used. We are seeing the same incidence of AI after aortic root replacement with aortic homografts or pulmonary autografts that we see with stentless porcine valves in the subcoronary position at 8 to 10 years of follow-up. My bet is that they dilate too. We have already documented this problem with the Ross procedure.

The second question concerns oversizing a stentless valve. Although I did do that some 5 to 10 years ago, we have now learned that this is probably a mistake. It is better to use a valve the size of the anulus and adjust the sinotubular junction to the size of the valve. Oversizing, particularly xenograft tissue, causes bends on the cusps, and those bends eventually tear or calcify prematurely. There is experimental work proving that already.