Observation of Cavitation in a Mechanical Heart Valve in a Total Artificial Heart

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Recently, cavitation on the surface of mechanical heart valves has been studied as a cause of fractures occurring in implanted mechanical heart valves. The cause of cavitation in mechanical heart valves was investigated using the 25 mm Medtronic Hall valve and the 23 mm Omnicarbon valve. Closing of these valves in the mitral position was simulated in an electrohydraulic totally artificial heart. Tests were conducted under physiologic pressures at heart rates from 60 to 100 beats per minute with cardiac outputs from 4.8 to 7.7 L/min. The disk closing motion was measured by a laser displacement sensor. A high-speed video camera was used to observe the cavitation bubbles in the mechanical heart valves. The maximum closing velocity of the Omnicarbon valve was faster than that of the Medtronic Hall valve. In both valves, the closing velocity of the leaflet, used as the cavitation threshold, was approximately 1.3–1.5 m/s. In the case of the Medtronic Hall valve, cavitation bubbles were generated by the squeeze flow and by the effects of the venturi and the water hammer. With the Omnicarbon valve, the cavitation bubbles were generated by the squeeze flow and the water hammer. The mechanism leading to the development of cavitation bubbles depended on the valve closing velocity and the valve stop geometry. Most of the cavitation bubbles were observed around the valve stop and were generated by the squeeze flow. *ASAIO Journal* 2004; 50:205–210.

In cases involving a single mechanical heart valve, the clinical findings reported to date have indicated that pitting and erosion occur on valve leaflets.[1] Before impact, fluid contained in the gap between the housing and the approaching leaflet is squeezed out, which results in a local pressure drop. If the pressure drop falls below the vapor pressure of the liquid, cavitation bubbles will occur.[2] When cavitation bubbles flow onward into a higher pressure region, the rapid collapse of these bubbles may generate a high speed microjet and shock waves. The collapsing cavitation bubbles generate high pressures. If the bubbles collapse near the material surface, they may damage the surface of mechanical heart valves.[3]

Proposed mechanisms of generating the low pressures necessary for cavitation to occur include water hammer, venturi, and squeeze flow effects. Water hammer and squeeze flow phenomena depend on the leaflet velocity just before closure.

Graf *et al.* measured the critical disk closing velocity that induces cavitation and focused primarily on the pressure drop caused by the deceleration of a disk.[4] Lee *et al.* and Shu *et al.* examined the cavitation threshold of dp/dt for different mechanical heart valves using stroboscopic photography.[5,6] He *et al.* investigated the mechanism of the formation of cavitation bubbles in cases involving Medtronic Hall valves; in that study, the average value of the increase in the rate of transvalvular pressure was used as an index loading rate.[7] However, the gradient of the ventricular pressure differed according to ventricular chamber compliance. In previous studies, the au-

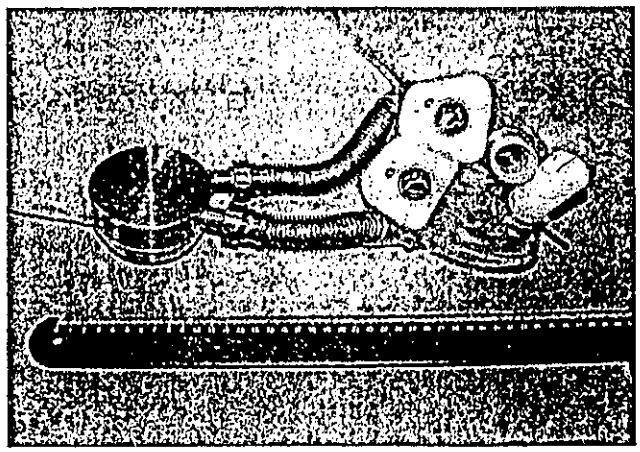

Figure 1. An electrohydraulic total artificial heart.

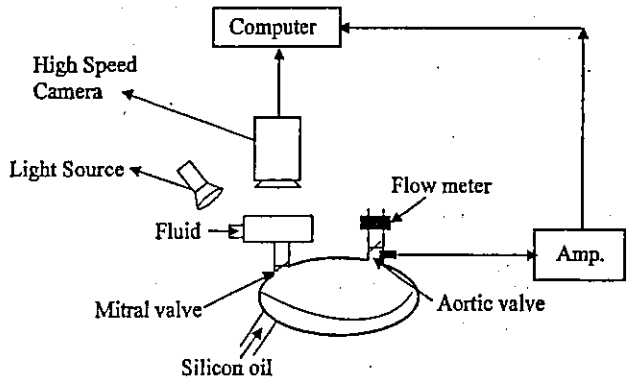

Figure 2. High speed camera system used for the observation of cavitation bubbles.

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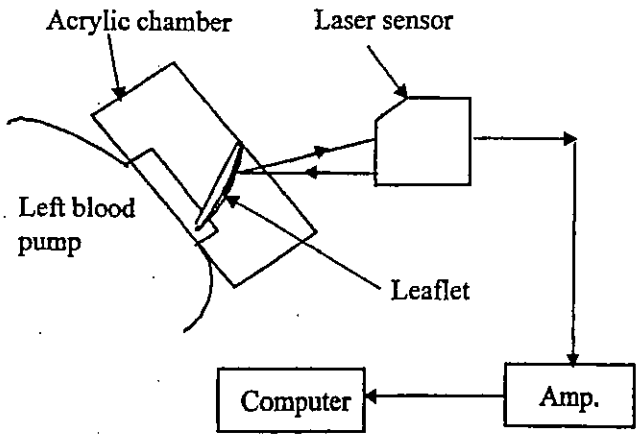

Figure 3. Laser sensor system used for the measurement of valve closing motion.

thors have shown that cavitation erosion on the valve surface increases with an increase in the closing velocity.[8] Moreover, erosion pit generation caused by cavitation was shown to be restricted to an area on the valve surface next to the edge of the valve stop where the squeeze flow had occurred. Because it is known that the maximum closing velocity of the leaflet contributes to the occurrence of squeeze flow, this velocity was used as an index of the cavitation threshold in the current study.

To investigate the mechanism of cavitation associated with the Medtronic Hall valve and the Omnicarbon valve in an electrohydraulic total artificial heart, two parameters were measured. First, an image was created of the cavitation bubbles using a high-speed camera. Next, the closing of the valve was observed using a laser displacement sensor.

Materials and Methods

The electrohydraulic total artificial heart (stroke volume 75 mL) used in this study was developed by the National Cardiovascular Center in Japan; this heart model consists of two diaphragm type blood pumps, an actuator, and a controller (**Figure 1**). The actuator is connected to both blood pumps by a flexible tube. The flexible tubes are filled with silicon oil. This electrohydraulic total artificial heart system functions as follows: silicon oil drives the blood pump via the inverse or reverse rotation of the impeller. The electrohydraulic total artificial heart used in this study was connected to an overflow mock circulatory loop tester.

A 25 mm Medtronic Hall valve and a 23 mm Omnicarbon valve were mounted in the mitral position. The leaflet diameter of the two valves was 20 and 18 mm, and the opening angle of those valves was 70° and 68°, respectively. Throughout the experiments, the 23 mm Medtronic Hall valve was mounted in the aortic position. The blood pumps were run at a heart rate from 60 to 100 beats per minute (bpm), and the cardiac outputs ranged from 4.8 to 7.7 L/min, respectively. Regarding the pressure conditions, the preload and afterload of the right blood pump was fixed at 10 and 30 mm Hg, respectively. However, the preload and afterload of the left blood pump were fixed at 10 and 100 mm Hg, respectively. Room temperature tap water was used as a test fluid.

To create an image of the cavitation bubbles, a high-speed camera (Memrecam fx 6000, nac, Tokyo, Japan) was used (**Figure 2**). The chamber was constructed from acrylic resin for optical access, and the high-speed camera was placed on top of the acrylic chamber; the cavitation bubbles were recorded at 10,000 frames per second.

A CCD laser displacement sensor (LK-080, Keyence, Corp., Tokyo, Japan) with a resonance frequency of 1 kHz was used

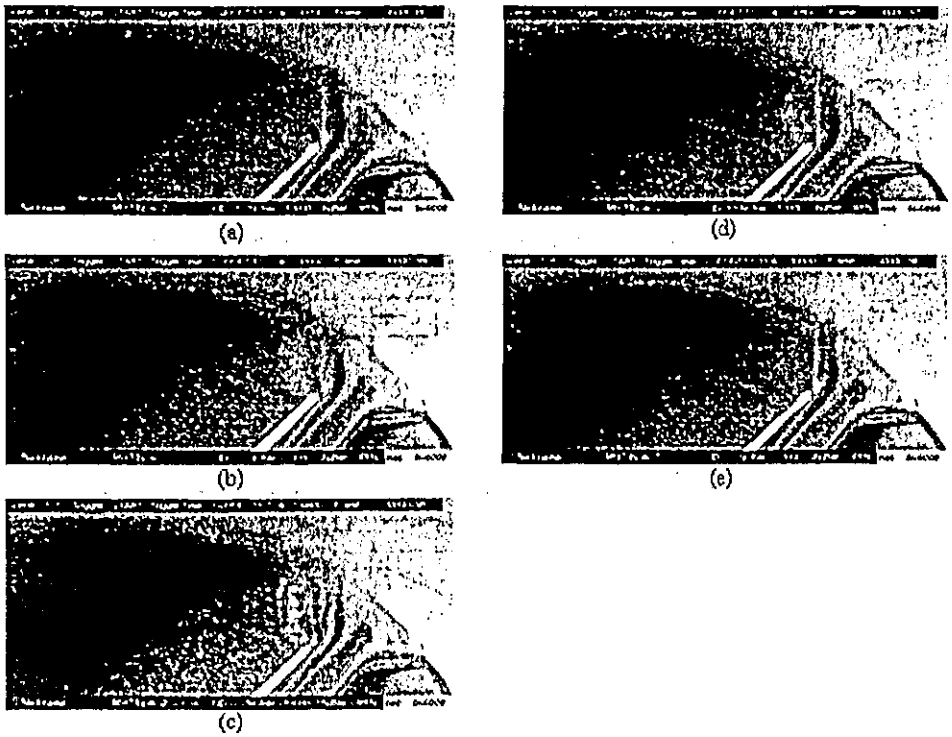

Figure 4. Cavitation bubbles in the Medtronic Hall valve (70 bpm, 10,000 frames per second): (a) contact, (b) 100 μs after contact, (c) 200 μs after contact, (d) 300 μs after contact, (e) 400 μs after contact.

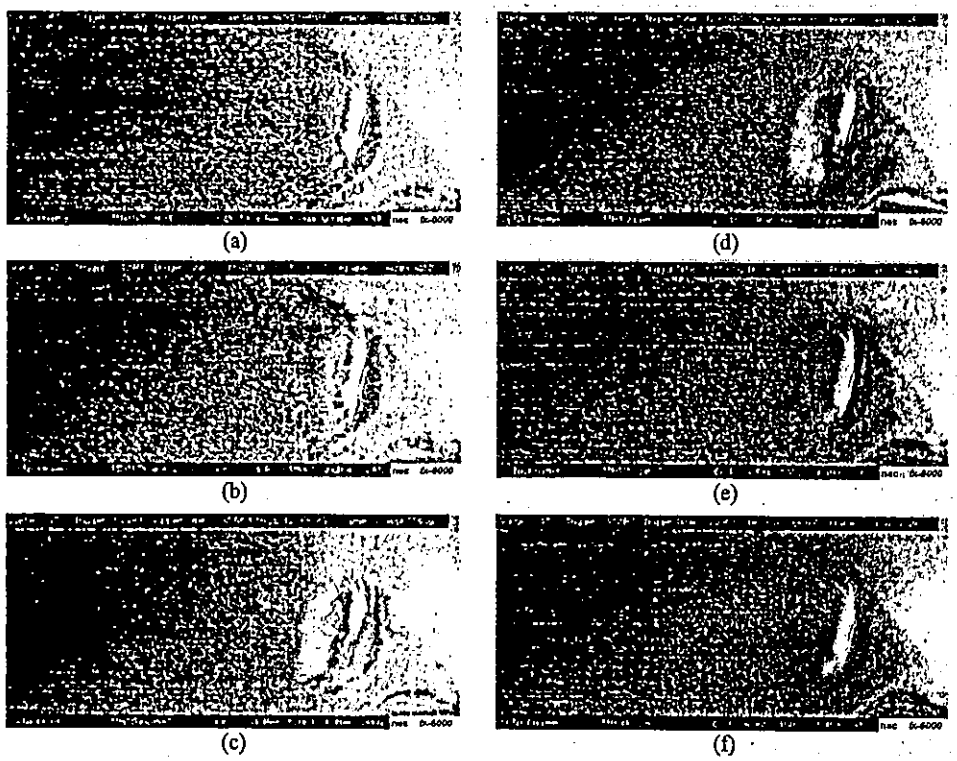

Figure 5. Cavitation bubbles in the Medtronic Hall valve (100 bpm, 10,000 frames per second): (a) contact, (b) 100 μs after contact, (c) 200 μs after contact, (d) 300 μs after contact, (e) 400 μs after contact, (f) 500 μs after contact.

to measure the opening and closing behavior of the leaflet (**Figure 3**). The laser sensor was placed on top of the acrylic chamber. To measure leaflet motion, a triangulating laser light method was used. To measure the flow rate, an electromagnetic flow meter (FR-130T, Nihon kouden, Tokyo, Japan) was positioned on the outflow side.

Results

The photographs of the cavitation bubbles associated with use of the Medtronic Hall valve are shown with the heart rate data in **Figures 4 and 5**. The duration of the interval between images was 100 μs, and the heart rate was 70 bpm. The cavitation bubbles were observed from the edge of the valve stop after the valve had closed. Cavitation bubbles were generated by the squeeze flow between the valve and the valve stop. When the heart rate reached 100 bpm, cavitation bubbles were observed in the following locations: at the edge of the valve stop, in the narrow gap between the valve and the valve housing, and on the inner side of the leaflet. The bubbles were generated by the squeeze flow, the venturi effect, and the water hammer effect, respectively. The cavitation bubbles were observed for duration of 400–500 μs.

Photographs of the cavitation bubbles associated with the Omnicarbon valve are shown in **Figures 6 and 7**. When the heart rate was 70 bpm, cavitation bubbles were observed near the edge of the valve stop. However, when the heart rate reached 100 bpm, cavitation bubbles were observed near the edge of the valve stop as well as on the inner side of the leaflet. The bubbles were generated by squeeze flow and water hammer effect. These cavitation bubbles were observed in intervals of 200–400 μs. This duration was shorter than the corresponding duration associated with the Medtronic Hall valve.

With both valves, the area in which cavitation bubbles appeared and the intensity and size of the bubbles generally increased with an increase in the valve closing velocity; in both cases, cavitation bubbles were observed at heart rates exceeding 70 bpm.

The closing of the Medtronic Hall and Omnicarbon valves is shown in **Figures 8 and 9**. The vertical axis represents the opening motion of the leaflet, and the radians reflect the closing motion of the leaflet. The vertical axis also represents the opening angle of the leaflet. For example, 1.2 on the vertical axis reflects an open state, and 0 represents a closed state. At the exact moment when the valve closed, the leaflet accelerated and reached a maximum velocity. In both valves, the closing duration decreased with a decrease in the heart rate.

The maximum closing velocity for each valve is shown in **Figure 10**. The maximum closing velocity of the Omnicarbon valve was faster than that of the Medtronic Hall valve. The maximum closing velocity of the two valves ranged from 0.78 to 2.2 m/s. Furthermore, it was found that this velocity increased with an increase in the heart rate. As indicated, the error bars were obtained from 20 measurements.

Discussion

The mechanism leading to the development of cavitation bubbles differed according to the geometry of the valve stop and the closing velocity. As shown in **Figure 11**, there is no narrow gap between the leaflet and the valve housing in the case of the Omnicarbon valve, and, therefore, no cavitation bubbles were created by the venturi effect. Cavitation bubbles were observed from the edge of the valve stop, on the inner side of the leaflet, and in the narrow gap between the leaflet and the valve housing. However, most of the cavitation bub-

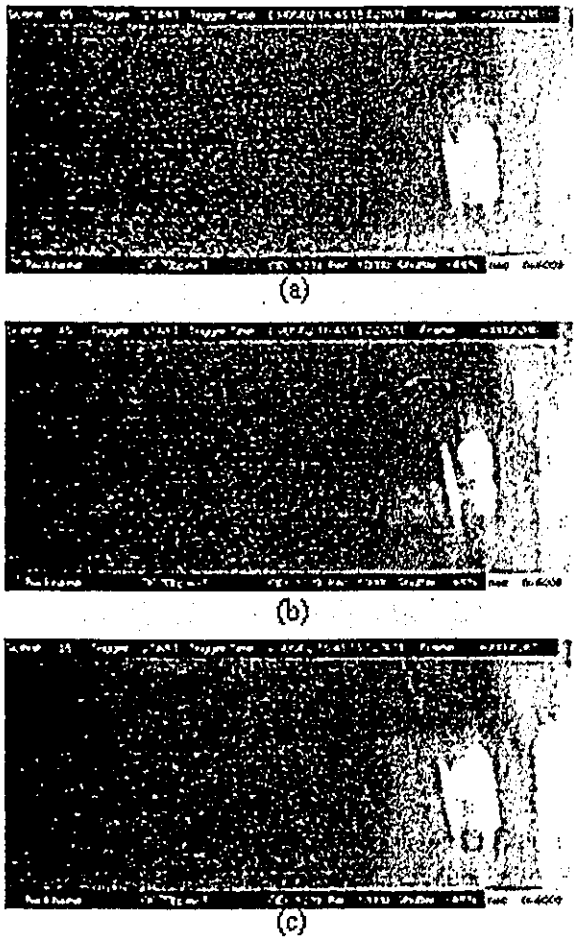

Figure 6. Cavitation bubbles in the Omnicarbon valve (70 bpm, 10,000 frames per second): (a) contact, (b) 100 μs after contact, (c) 200 μs after contact.

bles were generated in a region close to the inside edge of the valve stop. It also was observed that the area in which cavitation bubbles appeared increased with an increase in the closing velocity and the valve stop area. These results furthermore suggest that cavitation bubbles are induced by squeeze flow.

In previous studies, it was assumed that the squeeze flow, which occurs just before valve closure, leads to the pressure drop that supports cavitation formation.[8,9] A simple mechanism that accounts for the occurrence of the squeeze flow is shown in **Figure 12.** Assuming the valve closed with the maximum closing velocity and in a uniform flow from the gap, the following simple equation can be solved with respect to squeeze flow velocity U:

$$U(0) = \frac{VL}{b} = \frac{L\,db}{b\,dt}, \tag{1}$$

where b is the gap between the valve and the valve stop when a squeeze flow is generated, L is half of the length of the valve stop, and V is the valve closing velocity, and t is time. As shown in **Equation 1,** the squeeze flow velocity increases with an increase in the valve closing velocity and the valve stop area. As shown in **Figure 11,** the valve stop area (dot line of **Figure 11**)of the Medtronic Hall valve is greater than that of the Omnicarbon valve, such that there were more cavitation bubbles generated with the Medtronic Hall valve.

As shown in **Figure 11,** when the squeeze flow was emitted through gap size b, Rajaratnam reported the jet flow $U_{m(x)}$ as follows:[10]

$$\frac{U_m(x)}{U(0)} = \frac{3.5}{\sqrt{\frac{x}{b}}}, \tag{2}$$

where x is the distance from the valve stop. As can be seen in

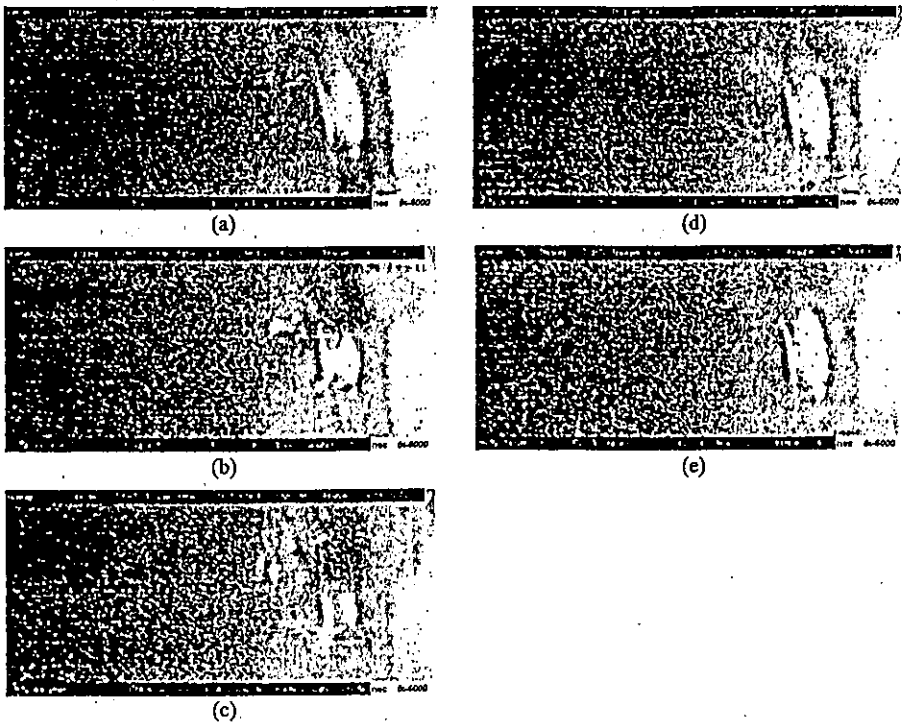

Figure 7. Cavitation bubbles in the Omnicarbon valve (100 bpm, 10,000 frames per second): (a) contact, (b) 100 μs after contact, (c) 200 μs after contact, (d) 300 μs after contact, (e) 400 μs after contact.

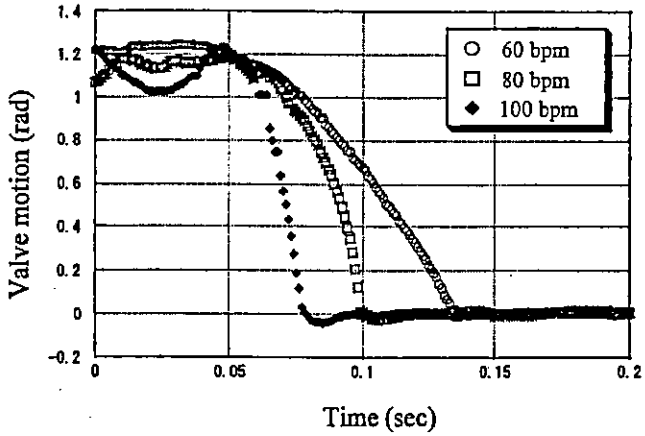

Figure 8. Closing motion of the Medtronic-Hall valve. bpm, beats per minute; rad, opening angle.

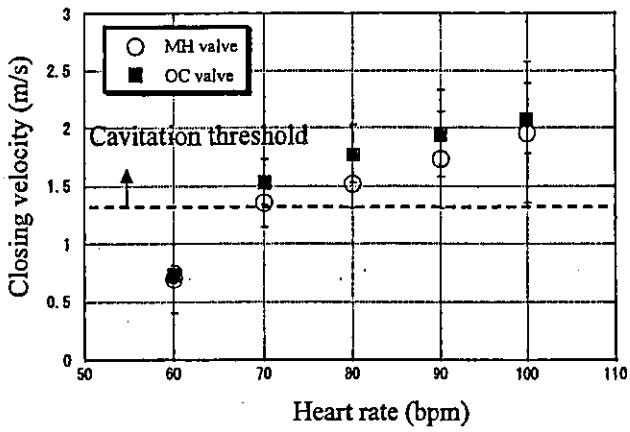

Figure 10. Maximum valve closing velocity. bpm, beats per minute.

Equation 2, the jet flow velocity $U_{m(x)}$ is directly proportional to , and, therefore, **Equation 2** may be altered to read as follows:

$$x = b\left\{\frac{3.5}{U_m(x)}\frac{VL}{b}\right\}^2, \quad (3)$$

When the flow velocity $U_{m(x)}$ exceeds 14 m/s, the pressure falls to a critical level. This flow velocity is widely used in the field of cavitation engineering as a critical fluid velocity at which cavitation bubbles may be generated. If it is assumed that a 14 m/s insert in $U_{m(x)}$ of **Equation 3**, x can be defined as the cavitation bubble generation area. As shown in **Figures 4–7**, the cavitation bubble generation area increased with an increase in the valve closing velocity and the valve stop area. For example, as shown in **Figures 4 and 5**, x of the cavitation bubbles generation area are 0.7 mm and 1.2 mm in the Medtronic Hall valve. If those values are inserted in **Equation 3**, and the gap sizes are 120 μm and 300 μm, however, its gap size is larger than that of other group results.[11] The authors think that the squeeze flow is generated just before leaflet closure. In the future, it will be necessary to investigate gap size induced by the squeeze flow and the squeeze flow velocity induced by the occurrence of cavitation bubbles in this experimental system.

These results also suggest that cavitation bubbles are induced by squeeze flow. As shown in **Figure 11**, the valve stop area of the Medtronic Hall valve was greater than that of the Omnicarbon valve, which led to an increase in the presence of cavitation bubbles observed in association with the Medtronic Hall valve. However, there was an unknown b of gap size in this study.

In particular, cavitation bubbles caused by the water hammer effect were observed only at high speed leaflet closing velocities. However, in the case of both valves, cavitation bubbles were observed at the edge of the valve stop at heart rates exceeding 70 bpm, as shown in **Figure 10**; thus the maximum closing velocity, approximately 1.3–1.5 m/s, could be considered as the cavitation threshold. With regard to the cavitation threshold, the squeeze flow is a very important parameter. In the future, it will be necessary to investigate the squeeze flow velocity induced by the occurrence of cavitation bubbles.

Cavitation phenomenon in mechanical heart valves differ depending on the working fluid. Because the purpose of this study was to select the best mechanical heart valves for the totally artificial heart, this comparison of the cavitation mech-

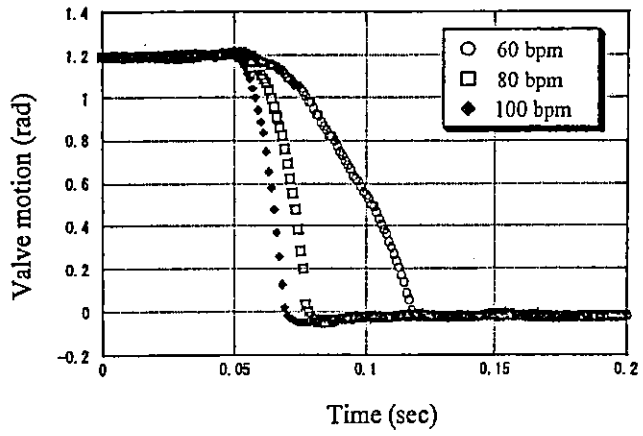

Figure 9. Closing motion of the Omnicarbon valve. bpm, beats per minute; rad, opening angle..

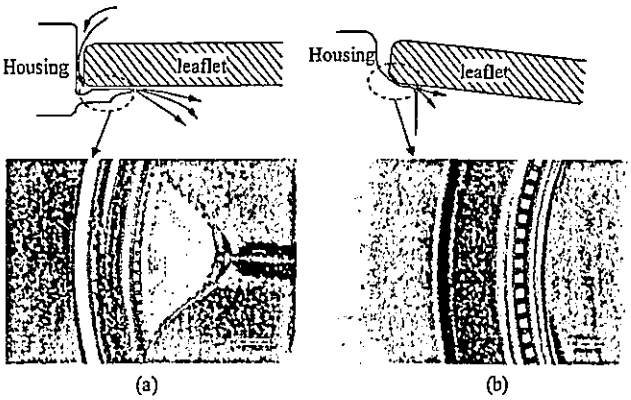

Figure 11. Configuration of the valve stop: (a) Medtronic Hall valve, (b) Omnicarbon valve.

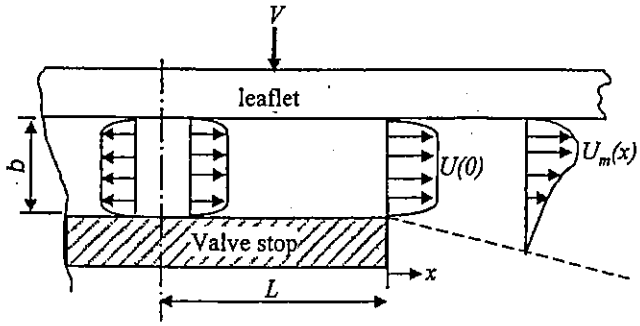

Figure 12. Mechanism for the occurrence of squeeze flow. *V*, valve closing velocity; *U*, squeeze flow velocity; $U_m(x)$, jet flow velocity.

anisms in the different mechanical heart valves appears to be valid. With regard to the cavitation threshold, the closing velocity of the leaflet is a very important parameter. From the viewpoint of squeeze flow and given the same closing velocity of the leaflet, a small size of valve stop could minimize cavitation. Even if the serious driving condition in the artificial heart, cavitation could occur when using monoleaflet valves in a clinical study.

Conclusions

Most cavitation bubbles were observed in the current study at the edge of the valve stop. The major cause of these cavitation bubbles was determined to be the squeeze flow. The formation of cavitation bubbles depended on both the valve closing velocity and the valve stop geometry. The maximum closing velocity of the Omnicarbon valve was faster than that of the Medtronic Hall valve. However, with both valves, a closing velocity of approximately 1.3–1.5 m/s could be considered as the cavitation threshold.

Acknowledgment

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Rationale for Off-Pump Coronary Revascularization to Small Branches—Angiographic Study of 1,283 Anastomoses in 408 Patients

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Background. Off-pump coronary artery bypass grafting (OPCAB) has gained wide acceptance in tandem with the development of the stabilizer and associated operative techniques. However bypass grafting to the small branches of a beating heart is technically demanding and remains controversial. In the present study we evaluated the graft patency and quality of anastomoses to small coronary arteries by early postoperative angiography.

Methods. Between March 2000 and December 2002 a total of 1,328 anastomosed sites to coronary branches were studied angiographically in 404 patients representing 88.6% of all cases who underwent OPCAB in this period. The coronary artery branches were categorized as large (>1.5 mm, group L: 1,028 anastomoses sites) or small (< 1.5 mm, group S: 300 sites) by intraoperative measurement. As in situ grafts the internal thoracic artery (ITA) and the gastroepiploic artery (GEA) were used at 504 and 28 distal anastomosis sites respectively. The radial artery (RA) was used as a composite graft for 739 distal anastomosis sites. Sequential bypass grafting was performed at 388 anastomosis sites in side-to-side fashion. Arterial grafts were used in 96.1% of total bypass grafting.

Results. The percentage of male gender was 78.3% in group S and 87.2% in group L ($p = 0.025$). The ITA was used in 43.7% of group L and 18.3% of group S ($p < 0.0001$). The RA was used in 49.4% of group L and 77% of group S ($p < 0.0001$). The overall patency and stenosis free rates (FitzGibbon Type A) were 97.2% and 96.2%. Graft patency and stenosis free rates in group S (96.7% and 93.3%) were as good as those in group L (97.5% and 97.1%). In group S, the patency and stenosis free rates of SV grafts were 71.4% and 57.1%. On the other hand, those of ITA grafts were 100% and 98.3% ($p = 0.53$ vs. saphenous vein graft [SVG]) and RA grafts were 95.8% and 92.1% ($p = 0.61$ vs. SVG) respectively. In group S, the graft patency and stenosis free rates of bypass to the obtuse marginal (OM) (93.7% and 87.5%) were slightly lower than those to other implantation sites left anterior descending (LAD: 100% and 97.3%; PL: 96.5% and 92.3%; DI: 98.0% and 96%; PDA: 97.0% and 97.0%; right coronary artery [RCA]: 100% and 100%) although there was no statistical significance. The graft patency and stenosis free rates were slightly better with side-to-side anastomosis than with end-to-side anastomosis (side-to-side: 98.1% and 95.8% vs. end-to-side 96.3% and 86.3%) in group S.

Conclusions. OPCAB to small coronary artery branches with arterial grafts provided satisfactory graft patency and stenosis free rates.

(Ann Thorac Surg 2004;77:1530–4)

Off-pump coronary artery bypass (OPCAB) has been widely applied as an attractive countermeasure to percutaneous catheter intervention in coronary artery disease (CAD). Recently, OPCAB has become a standard surgical option for CAD owing to the development of equipments such as the stabilizer and associated operative techniques [1–3]. Meanwhile patients with small coronary arteries represent a significant proportion of those undergoing coronary artery revascularization. In some previous reports percutaneous catheter intervention in small coronary arteries was associated with high success and low complication rates but had higher incidence of repeat revascularization even when stents were used [4–9]. Although bypass grafting to small branches on the beating heart is technically demanding and remains controversial, few reports have addressed the problems and outcomes of OPCAB for small coronary arteries [10]. In the present study we evaluated graft patency and quality of anastomoses to small coronary arteries in OPCAB by early postoperative angiography and compared these findings with those of the treatment of larger coronary arteries.

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Material and Methods

Between March 2000–December 2002, a total of 1,328 anastomosed sites on coronary branches were studied angiographically in 404 patients representing 88.6% of all cases who underwent isolated OPCAB during this period. Male patients accounted for 83% (n = 336) and female for 17% (n = 68). The coronary artery branches were categorized by size as large (≥ 1.5 mm; group L: 1,028 anastomosis sites) or small (< 1.5 mm; group S: 300 sites) by intraoperative measurement. To be precise a coronary artery was recorded as small, intraoperatively, when the maximum intraarterial shunt diameter that could be passed from the coronary arteriotomy in a distal direction was 1 mm or 1.25 mm, in conjunction with severe distal arteriosclerosis. As an intraoperative arterial shunt, we apply two different products; the Clear View (Medtronic, Fridley, MN) for the 1 mm and 1.25 mm sizes, and the AnastaFLO (Edwards Life Science, Irvine, CA) for the 1.5 mm and larger sizes. As all those anastomosis, that underwent coronary endarterectomy, could not accept even the 1 mm intraoperative arterial shunt, they were all included in Group S.

Our basic strategy for OPCAB has been previously described [1, 11]. We prefer multiple and complete coronary revascularization with composite and sequential grafting using all available arterial grafts—especially in-situ arterial grafts. Aortic no-touch is also the basic strategy [11]. Additionally in 17 cases using vein grafts we used aortic connecters that automatically anastomosed the saphenous vein graft (SVG) to the ascending aorta so as to avoid the complications related to the aortic clamp. Of these cases, nine were studied angiographically postoperatively. Six cases were in group L and six cases were in group S. In the early years we used the ITA-SVG composite grafts for 28 patients. Surgical technique and details of intraoperative and postoperative management were described previously [1, 11]. To prevent arterial spasm, continuous intravenous infusion of diltiazem (0.5–1.0 μg/kg) or nicardipine (0.1–0.2 μg) was used intraoperatively and during the first 16 hours after the operation. Diltiazem (100–200 mg/d) or amlodipine (2.5–5.0 mg/d) was then prescribed for oral administration in conjunction with aspirin (162 mg/d) beginning on the next morning [11].

The number of patients who underwent bypass grafting for small arteries was 179 (44.3%) whereas those who only had large arteries done numbered 225 (55.6%). The percentage of male gender was 78.2% (n = 140) in group S and 87.1% (n = 196) in group L ($p = 0.025$). In this series, bypass grafting with an arterial graft accounted for 96.1% of all anastomoses (n = 1277). The distribution of graft material is given in Figure 1. The internal thoracic artery (ITA) was used in 44% of group L and in 18% of group S ($p < 0.0001$) and the radial artery (RA) was used in 49% of group L and in 78% of group S ($p < 0.0001$). The distribution of anastomosis sites is given in Figure 2. Anastomosis of the left anterior descending branch (LAD) comprised 36% of group L and 11% of group S (p

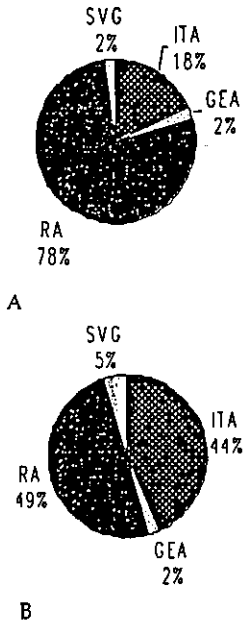


Fig 1. Distribution of graft material. (A) Group S. (B) Group L. (GEA = gastroepiploic artery; ITA = internal thoracic artery; RA = radial artery; SVG = saphenous vein graft.)

< 0.0001), that of the posterolateral branch (PL) was 24% in group L and 43% in group S ($p < 0.0001$), and that of the RCA was 2% in group L and 0.3% in group S ($p = 0.0171$). The distribution of the anastomosis method is given in Figure 3. Side-to-side anastomosis,—that is, sequential bypass—was performed in 27% of group L and 37% of group S ($p = 0.0019$). The proportion of patients using SVG who had undergone medical treatment for diabetes mellitus preoperatively was 31% (15/49) in group L and 29%(2/7) in group S. Side-to-side anastomosis was performed in 27% of group L (13/49) and 0% of group S (0/7).

Angiographic Study

Coronary and graft angiography was performed at 10–21 days (mean 14 days) after the OPCAB. Patients with renal dysfunction (serum creatinine > 2.0 mg/dL) were evaluated only by stress thallium myocardial scintigraphy and they were excluded from this study. Graft patency and stenosis were independently assessed by the single cardiologist team. Graft stenosis was regarded as significant if it was applied to FitzGibbon type A [12].

Statistical Methods

All data were reviewed retrospectively. All values are expressed as mean ± standard deviation (SD). A comparative analysis was performed between the different patient groups. Differences were analyzed using the univariate analysis (the Mantel-Haenszel χ^2 test, the two-tailed t test, Fischer exact test, or the Mann–Whitney U test, as appropriate). A value of p less than 0.05 was used to indicate significance.

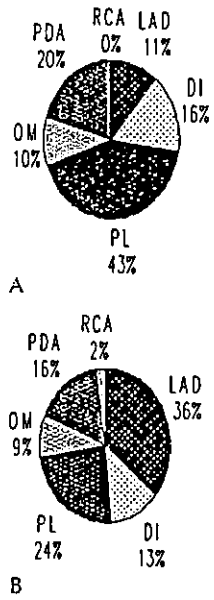


Fig 2. Distribution of anastomosis site. (A) Group S (%). (B) Group L (%). (LAD = left anterior descending branch; OM = obtuse marginal artery; PL = posterolateral branch; RCA = right coronary artery.)

Results

The overall patency was 97.2% (1292/1328), and the stenosis free rate was 96.1% (1,276/1,328). Patency was 97.5% (1,002/1,028) in group L and 96.7% (290/300) in group S ($p = 0.963$). The stenosis free rate was 96.7% (996/1,028) in group L and 93.3% (280/300) in group S ($p = 0.706$). The patency and stenosis free rates stratified by graft material are given in Figure 4. The patency rates of the ITA graft were 98.2% (441/448) in group L and 100% (55/55) in group S ($p > 0.999$). The stenosis free rates of it were 96.7% (434/449) in group L and 98.2% (54/55) in group S ($p > 0.999$). The patency rates of the SVG were 95.9% (47/49) in group L and 71.4% (5/7) in group S ($p = 0.763$). The patency rate of the SVG was 71.4% and that of the ITA was 100% ($p = 0.763$, vs. SVG), the gastroepiploic artery [GEA] was 100% ($p = 0.713$, vs. SVG), and the RA was 96.5% ($p = 0.772$, vs. SVG) in group S. The stenosis free rates of the SVG were 98% (48/49) in group L and 57.1% (4/7) in group S ($p = 0.530$). Among the SVG anastomosis with aortic connector devices, one bypass graft was occluded and this was in group L. The patency rates of the RA were 97.4% (495/508) in group L and 96.5% (223/231) in group S ($p = 0.954$) whereas the stenosis free rates of the RA were 97.4% (484/508) in group L and 93.5% (216/231) in group S ($p = 0.909$).

The patency and stenosis free rates stratified by the anastomosis site are given in Figure 5. The patency of the LAD site was 99.2% (365/368) in group L and 100% (34/34) in group S ($p > 0.999$). The stenosis free rate of the LAD was 97.8% (360/368) in group L and 97.1% (33/34) in group S ($p > 0.999$). In both groups S and L, there was no significant difference in patency rate between anastomosis sites. The stenosis free rate of the OM (86.2%) was lower than that of other sites in group S.

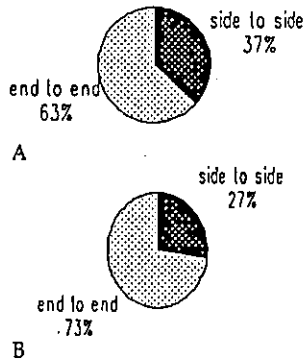


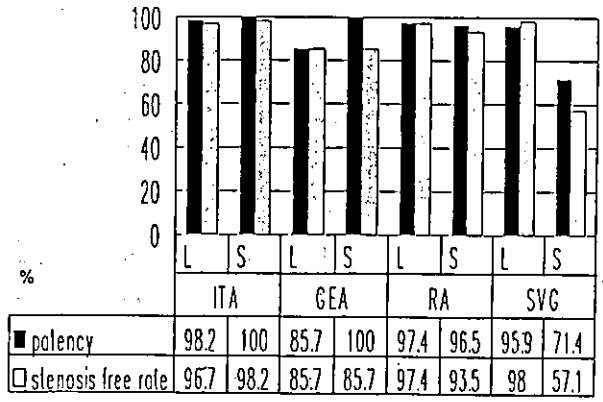
Fig 3. Distribution of anastomosis method. (A) Group S. (B) Group L.

The patency and stenosis free rates stratified by the anastomosis site are given in Figure 6. The rates of patency and freedom from stenosis in side-to-side anastomosis were better than those in end-to-side anastomosis in both groups L and S although the difference was not significant.

Coronary endarterectomy was carried out in 8 patients, all of whom could not accept the 1.5 mm or larger shunt and the patency rate was 87.5% (7/8).

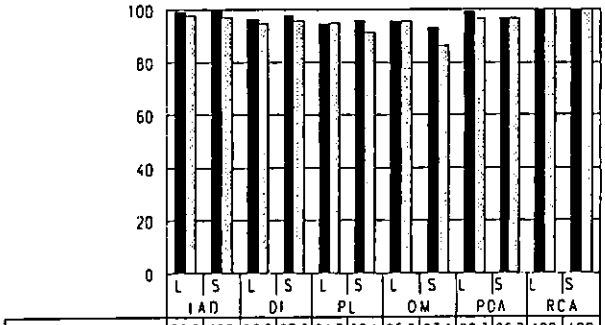
Comment

Recently OPCAB has been rapidly revived because of its cost effectiveness and the availability of commercial stabilizing devices and, of course, the further development of the operative technique [1–3, 13, 14]. However OPCAB is still a somewhat more or less technically demanding procedure than is conventional CABG despite local site stabilization and anesthetic management such as induced bradychardia. Natural motion and counteraction of the heart to derangement during heart elevation is still a serious concern in the pursuit of an accurate anasto-



	ITA		GEA		RA		SVG	
■ patency	98.2	100	85.7	100	97.4	96.5	95.9	71.4
□ stenosis free rate	96.7	98.2	85.7	85.7	97.4	93.5	98	57.1

Fig 4. Patency and stenosis free rate stratified by graft material. (GEA = gastroepiploic artery; ITA = internal thoracic artery; RA = radial artery; SVG = saphenous vein graft.)

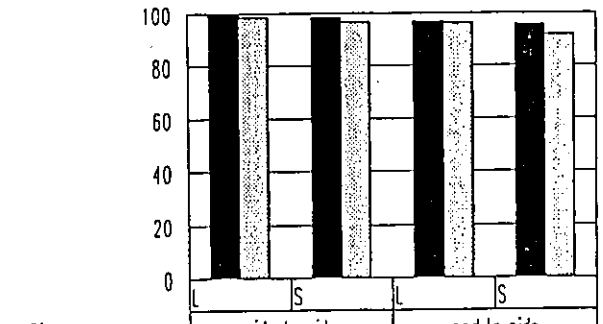


	LAD		DI		PL		OM		PCA		RCA	
	L	S	L	S	L	S	L	S	L	S	L	S
patency	99.2	100	96.2	97.9	94.7	96.1	95.6	93.1	99.3	96.7	100	100
stenosis free rate	97.8	97.1	94.6	95.7	95.1	91.4	95.6	86.2	96.9	96.7	100	100

Fig 5. Patency and stenosis free rate stratified by anastomosis site. (LAD = left anterior descending branch; OM = obtuse marginal artery; PL = posterolateral branch; RCA = right coronary artery.)

mosis. This uneasiness could cause distress to the surgeon especially when the target artery is small [10]. The results of this study show that the quality of OPCAB to small coronary arteries is as good as that to large arteries with respect to early graft patency and freedom from stenosis.

The definition of small coronary branch has not been established clearly before and several previous reports set up their criteria [4–10]. Ramstrom and associates reported the coronary artery bypass graft to small vessel disease which was recorded intraoperatively when the maximum diameter of the probe that could be passed from the coronary arteriotomy in a distal direction was 1 mm or 1.5 mm in conjunction with severe distal arteriosclerosis [10]. To refer this report, we used the intracoronary shunt for the measurement of coronary artery branches. The size of a coronary vessel we measured in this study may not the real diameter of the coronary branches, but the surgical diameter of the coronary branches, because the coronary vasospasm could reduce its size after the vessel is touched. However we adopted the surgical diameter for the definition of small coronary



%	side to side		end to side	
	L	S	L	S
patency	99.3	98.2	96.8	95.8
stenosis free rate	98.6	96.4	96.3	91.6

Fig 6. Patency and stenosis free rate stratified by anastomosis method.

artery because the surgical diameter is the very matter of great importance for surgeons.

Our basic strategy of OPCAB has been described previously [1, 11]. The essential philosophy of our institution comprises the aorta no-touch technique using an in-situ graft and composite and sequential grafting methods combined with total arterial revascularization using only internal thoracic artery and composite radial artery grafts. In fact, bypass grafting with an arterial graft accounts for 96.1% of all anastomoses (n = 1277). Furthermore when using vein grafts we have recently adopted the application of the aortic connecters that automatically anastomose the SVG with the ascending aorta so as to avoid the complications of the aortic clamp; this yields the benefit of reduced cerebrovascular accidents by avoiding aortic manipulation [15].

Previous reports have shown that the results of CABG are generally less favorable in women than in men [10, 16]. Although many factors are present the smaller diameter of female coronary arteries, which can cause technical difficulties, has often been proposed as a dominant factor. In this report the percentage of female gender was significantly larger in group S. This result shows that women have a risk that is associated with small arteries.

In a previous report the patency was inversely related to the size of the grafted vessel in the LAD and circumflex (CX) areas in OPCAB [17]. However, in our series, the patency and stenosis free rate of small arteries were proven to match those of larger arteries even with OPCAB. Moreover, as described in previous reports, women have a higher prevalence of varicose or stripped saphenous veins [10]. Assuming this to be true, and given our recent preference for arterial grafts over vein grafts, there is less likelihood of trouble with diseased vessels.

This study provides us with information about the distribution of the small coronary arteries. The small coronary arteries were more inclined to be located in the posterolateral (PL) than in the LAD. Although this represents not a clinical result but an anatomical one, it possesses clinical significance because we prefer to employ total arterial bypass grafting.

Patency and quality of the anastomoses were excellent although late follow-up will be necessary. Some pitfalls emerge with regard to the patency and stenosis free results. Poor long-term patency of vein grafts in CABG and the gross atheromatous changes seen on angiography have been reported. In this study early patency of the SVG in small arteries has no significant difference with that of the ITA or the RA. The patency and stenosis free rate of small SVG did not have something to do with the morbidity of diabetes mellitus, application of aortic connector, or the type of anastomosis method. Also the stenosis free rate of the OM in small arteries was slightly lower than at other anastomosis sites. This supports the technical difficulty of anastomosis at the OM including the lifting and stabilization of the heart. The patency and stenosis free rates of side-to-side anastomosis were comparable to but slightly better than those of end-to-side anastomosis. This demonstrates the benefit of the dia-

mond shape anastomosis which enlarges the anastomotic area.

There is the main limitation in this study that the angiographies were done early after the procedure. The angiographic study done at 6 or 12 months after surgery is the subject to be done in the future.

Conclusion

OPCAB to small coronary artery branches with arterial grafts provides satisfactory graft patency and freedom from stenosis.

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SHOULD THE TRANSVERSE AORTIC ARCH BE REPLACED SIMULTANEOUSLY WITH AORTIC ROOT REPLACEMENT FOR ANNULOARORTIC ECTASIA IN MARFAN SYNDROME?

by

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Should the transverse aortic arch be replaced simultaneously with aortic root replacement for annuloaortic ectasia in Marfan syndrome?

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Objective: The purpose of this study was to determine, on the basis of the late fate of the intact aortic arch with abnormal tissue after aortic root replacement, whether the intact aortic arch should be replaced prophylactically at the time of aortic root replacement for annuloaortic ectasia in Marfan syndrome.

Methods: A retrospective review was performed in 85 patients with Marfan syndrome who underwent aortic root replacement for annuloaortic ectasia with or without aortic dissection (mean age 37 years, range 19-61 years). These 85 patients were divided into four groups according to the postoperative condition of the residual aorta. In group I (n = 47), the patients underwent aortic root replacement for annuloaortic ectasia with or without localized dissection in the ascending aorta. In these patients the residual aorta, including the aortic arch, was therefore intact. In group II (n = 10), the aortic arch was intact, although the descending thoracic aorta was dissected because of the preoperative type B dissection. In groups III and IV, the patients had type A dissection involving the transverse arch associated with annuloaortic ectasia. In group III (n = 13), residual dissection existed in the descending thoracic aorta after concomitant total arch replacement. In group IV (n = 15), the aortic arch and the descending thoracic aorta were dissected.

Results: There were 5 early deaths (3 in group I, 1 in group II, and 1 in group III). Subsequent operations were required in 10, 5, 6, and 7 cases in groups I, II, III, and IV, respectively. Regarding the aortic arch, only 2 of 53 survivors of the initial hospitalization with an intact aortic arch (groups I and II) underwent subsequent total arch replacement for the onset of dissection in the aortic arch, and 4 of 14 survivors of the initial hospitalization with a residual dissecting arch (group III) needed subsequent total arch replacement. Actuarial freedom from arch repair among patients with an intact aortic arch (91% at 15 years) was significantly higher than that among patients with a residual dissecting arch (49% at 15 years, $P = .0078$).

Conclusions: The incidence of new dissection in the residual intact arch after aortic root replacement was extremely low. Therefore prophylactic replacement of the intact arch does not appear to be necessary at aortic root replacement for annuloaortic ectasia in Marfan syndrome.

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ACD

Since Dr Antonine Marfan first described a case of Marfan syndrome in 1896,[1] there have been reports of numerous investigations on this inheritable disorder of connective tissue, which often evolves to cause progressive dilatation of the aortic root and ascending aorta.[2] Recent advances in molecular biology and genetics have enabled us to elucidate the precise mechanism of this abnormal arterial elasticity,[3,4] and it is now known that Marfan syndrome is associated with mutations in the fibrillin 1 gene on chromosome 15q21.1. Fibrillin is an important component of connective tissue microfibrils, which form a meshwork of elastin in the aorta. Depletion of fibrillin may alter the pattern of elastin deposition so that the aorta is less able to withstand recurrent wall stress, resulting in progressive aortic dilatation, dissection, or rupture. Prophylactic surgery thus is crucial to improvement of the overall prognosis for patients with Marfan syndrome. The current recommendation is to operate when the size of the aortic root or ascending aorta is 5.0 to 5.5 cm. In patients with additional risk factors, such as progressive dilatation of an aneurysm or a family history of dissection, rupture, or aortic regurgitation, surgery for annuloaortic ectasia should be considered for a smaller size of the aortic root.[5-8] However, the patient with Marfan syndrome still has the potential risk of dissection or rupture in the residual intact aorta after aortic root replacement for isolated annuloaortic ectasia. Therefore emergency total arch replacement through median resternotomy will be required if acute dissection occurs in the residual aortic arch after aortic root replacement. It is generally accepted that elective concomitant total arch replacement for an intact arch has a lower risk than does emergency total arch replacement through median resternotomy.

The purpose of this study was to determine whether not the intact aortic arch should be replaced prophylactically and aggressively at the time of aortic root replacement for annuloaortic ectasia in Marfan syndrome. To accomplish this, we studied the late fate of the intact residual arch after aortic root replacement in a series of 85 patients.

Patients and Methods

Between October 1979 and May 2002, a total of 117 patients with Marfan syndrome underwent surgery for cardiovascular disease at the National Cardiovascular Center, Osaka, Japan. The diagnosis of Marfan syndrome was based on internationally established diagnostic criteria.[9] Among the 117 patients with Marfan syndrome, 85 underwent aortic root replacement for annuloaortic ectasia with or without aortic dissection at the initial operation. These patients were divided into four groups according to the postoperative condition of the residual aorta (Figure 1).

In group I (n = 47), the patients underwent aortic root replacement for annuloaortic ectasia with or without localized dissection in the ascending aorta. In these patients the remaining aorta including the aortic arch was therefore intact. In group II (n = 10), the aortic arch was intact, although the descending thoracic aorta was dissected because of preoperative Stanford type B dissection. In the groups III and IV, the patients had Stanford type A dissection involving the transverse arch associated with annuloaortic ectasia. In group III (n = 13), persistent dissection existed in the descending thoracic aorta after concomitant total arch replacement. In group IV (n = 15), the residual aortic arch and descending thoracic aorta were dissected. The distribution of aortic disease in the groups is shown in Table 1.

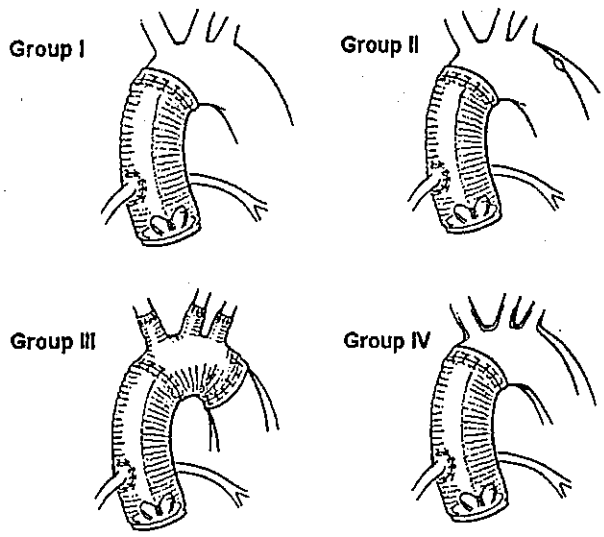

Figure 1. Eighty-five patients who underwent aortic root replacement for annuloaortic ectasia at initial operation were divided into four groups according to postoperative condition of residual aorta. Group I, all remaining aortas intact (n = 47); group II, aortic arch intact, although type B dissection remained (n = 10); group III, dissection remained in descending thoracic aorta after concomitant total arch replacement (n = 13); group IV, residual dissection including aortic arch persisted after aortic root replacement alone (n = 15).

This study was approved by the institutional review board of the National Cardiovascular Center, Osaka, Japan.

Follow-up

Data were prospectively collected from the database of the National Cardiovascular Center. Follow-up was performed in all cases up to May 31, 2002. The mean follow-up time was 110 ± 78 months (maximum 287 months).

Statistical Analysis

The continuous data in this study are expressed as mean value ± SD, and categoric variables are expressed as percentage. Comparisons of the characteristics were performed with the χ^2 test for nominal variables and the Student t test in the two groups for continuous and ordinal variables. Actuarial survival and freedom from reoperation and subsequent arch repair were estimated by the Kaplan-Meier product-limit method. Survival curves were compared between the groups with the log-rank test. Survivals presented in this report include in-hospital (death within 30 postoperative days or before hospital discharge). Reoperations that

TABLE 1. Distribution of aortic disease

	I (n = 47)	II (n = 10)	III (n = 13)	IV (n = 15)
Age (y, mean ± SD)	38.7 ± 12.7	37.8 ± 7.1	32.4 ± 8.9	34.4 ± 6.9
Male/female ratio	31:16	8:2	7:6	9:6
Sinus of Valsalva (mm, mean ± SD)	67.9 ± 13.6	63.2 ± 7.3	61.5 ± 3.6	64.8 ± 20.2
Aortic regurgitation (No.)	39	8	11	12
Type of dissection (No.)				
Acute Stanford type A	2*		4	4
Chronic Stanford type A	5*		9	11
Acute Stanford type B		1		
Chronic Stanford type B		9		
Site of initial tear (No.)				
Ascending aorta	7		12	15
Descending thoracic aorta		10	1	

*Localized dissection in ascending aorta.

appeared with a nonbiased time course were expressed as linearized rates.

Results

Surgical Procedure

Surgical procedures in each group are given in Table 2. Sixty-eight patients (80.0%) underwent composite graft repair. The prosthetic valves used in this study included 56 mechanical and 12 bioprosthetic valves. Seventeen patients (20.0%) underwent valve-sparing procedures (13 reimplantation, 4 remodeling).[5,10] From 1990 onward, in all the patients with a dissected arch, excluding 4 who were in a state of shock, concomitant total arch replacement was performed. At total arch replacement, cervical vessels were reconstructed with a branched graft to exclude as much diseased aortic tissue as possible (Figure 1). This technique averted the problem of bleeding from the suture line in dissected aortic tissue.

TABLE 2. Types of operations

	I (n = 47)	II (n = 10)	III (n = 13)	IV (n = 15)
Bentall	36	7	11	14
Reimplantation	7	3	2	1
Remodeling	4			
Concomitant procedure				
Coronary artery bypass grafting			2	
Mitral valve plasty	5	1		1
Tricuspid annuloplasty				1
Maze	1			
Others	1*			1†

*Ventricular septal defect closure.
†Cesarean delivery and hysterectomy.

Operative Morbidity and Mortality

There were 5 early deaths (5.9%). The operative mortalities were 6.4% (3/47) in group I, 10.0% (1/10) in group II, 0% in group III, and 6.7% (1/15) in group IV. Causes of death were low cardiac output (n = 1), ventricular tachycardia (n = 1), and sudden cardiac death (n = 1) in group I; low cardiac output (n = 1) in group II; and low cardiac output (n = 1) in group IV. Postoperative complications included bleeding (n = 3), intraoperative balloon pump insertion (n=2), percutaneous extracorporeal membrane oxygenation support (n = 2), stroke (n = 1), respiratory dysfunction (n = 3), liver dysfunction (n = 4), and renal failure (n = 1) in group I; bleeding (n = 1), intraoperative balloon pump insertion (n = 1), left ventricular assist device insertion (n = 1), mediastinitis (n = 1), and liver dysfunction (n = 1) in group II; bleeding (n = 1), respiratory failure (n = 1), intraoperative balloon pump insertion (n = 1), and percutaneous extracorporeal membrane oxygenation support (n = 1) in group III; and bleeding (n = 1), renal failure (n = 1), and respiratory failure (n = 1) in group IV.

Reoperation

Thirty-seven (46.3%) of 80 patients who survived initial hospitalization required 67 subsequent operations for the remaining part of the aorta, valve regurgitation, or late complications such as pseudoaneurysm formation or prosthetic valve dysfunction after the initial operation. Focusing on reoperations for the residual aorta beyond the location of earlier repairs, 28 patients required 47 subsequent operations (Figures 2 and 3). The actuarial freedoms from reoperation for all patients were calculated to be 80.3% ± 4.8% at 5 years, 59.1% ± 6.9% at 10 years, and 48.0% ± 8.1% at 15 years. The linearized rate of reoperation was 6.0 ± 11.4 events per 100 patient-years in 80 patients who survived initial hospitalization.

In group I, 10 of 44 patients who survived initial hospitalization underwent the second-stage operation. The actuarial freedoms from reoperation in the 44 initial survivors were 89.5% ± 5.7% at 5 years, 78.5% ± 7.4% at 10 years, and 64.3% ± 11.0% at 15 years. The linearized rate of

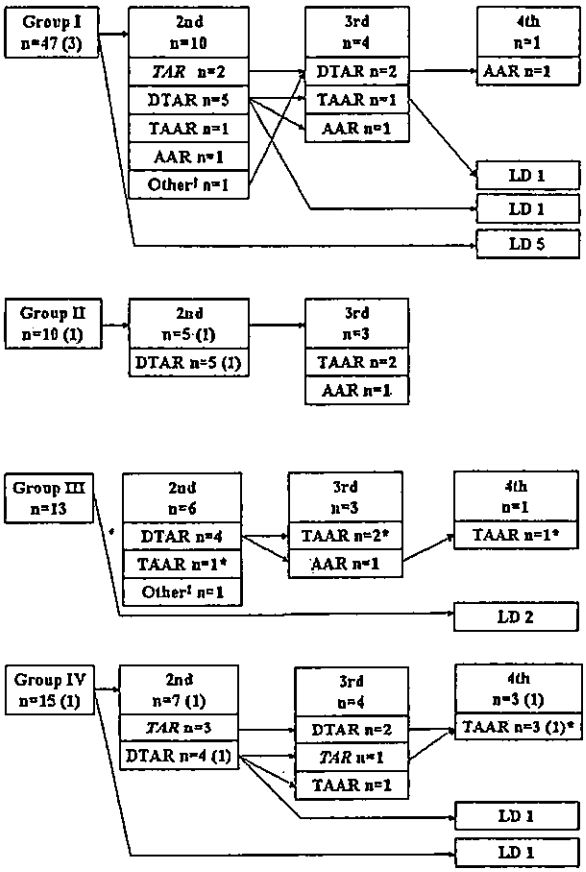

Figure 2. Outcomes of patients in each group. Numbers in parentheses represent in-hospital deaths. *TAR*, Total arch replacement; *DTAR*, descending thoracic aortic replacement; *AAR*, abdominal aortic replacement; *TAAR*, thoracoabdominal aortic replacement; *LD*, late death. *Asterisk* indicates replacement of entire aorta; *dagger* indicates femerofemeral bypass for malperfusion because of acute type B dissection; *double dagger* indicates external iliac artery–superior mesenteric artery bypass for malperfusion because of acute type B dissection.

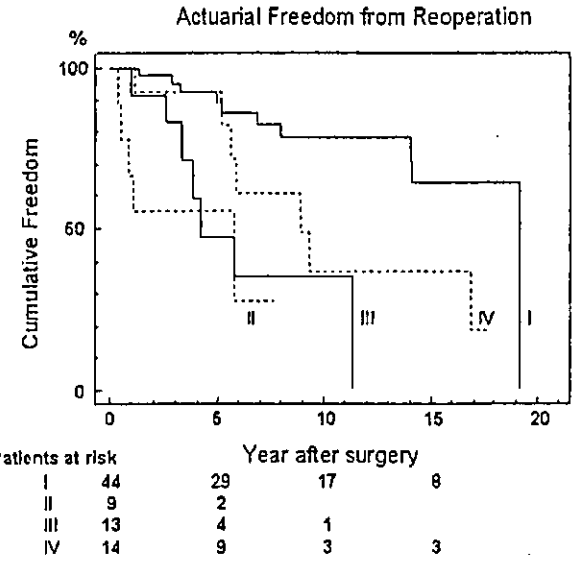

Figure 3. Actuarial freedom from reoperation in each group.

reoperation was 2.7 ± 5.9 events per 100 patient-years. The reasons for the second-stage operation were onset of type B dissection in 7 patients, onset of aortic arch dissection in 2 patients, and abdominal aortic aneurysm in 1 patient. The time until the onset of type B dissection ranged from 16 months to 14 years after aortic root replacement. The actuarial freedoms from operation for the onset of type B dissection were 89.4% ± 5.1% at 5 years, 81.2% ± 7.2% at 10 years, and 73.9% ± 9.6% at 15 years. In 2 patients who received total arch replacement, the times after aortic root replacement until the onset of aortic arch dissection were 5 and 14 years. The actuarial freedoms from aortic arch repair were 97.0% ± 3.0% at 5 years, 97.0% ± 3.0% at 10 years, 89.5% ± 7.7% at 15 years, and 89.5% ± 7.7% at 20 years.

In group II, 5 of 9 patients who survived initial hospitalization required 8 subsequent operations for residual type B dissection. However, there was no onset of arch dissection or retrograde dissection toward the residual intact aortic arch. The actuarial freedom from reoperation among the 9 patients who survived initial hospitalization was 55.6% ± 16.6% at 5 years. The linearized rate of reoperation was 10.4 ± 10.6 events per 100 patient-years.

In group III, 6 of 13 patients who survived initial hospitalization required 10 subsequent operations for residual dissection in the descending thoracic aorta. The actuarial freedoms from reoperation were 47.6% ± 16.8% at 5 years and 35.7% ± 16.3 % at 10 years. The linearized rate of reoperation was 35.7 ± 16.3 events per 100 patient-years.

In group IV, 7 of 14 patients who survived initial hospitalization required 14 subsequent operations for dissection in the residual aorta including the aortic arch. The actuarial freedoms from reoperation were 92.3% ± 7.4% at 5 years and 36.9% ± 16.3% at 10 years. The linearized rate of reoperation was 10.4 ± 19.5 events per 100 patient-years. Among the 7 patients who underwent subsequent operations, 4 underwent total arch replacement. The actuarial freedoms from subsequent arch repair were 88.9% ± 10.5% at 5 years, 64.8% ± 16.5% at 10 years, and 48.6% ± 18.7% at 15 years.

Reoperation in patients with an intact aorta versus patients with a residual dissecting aorta. As mentioned previously, the residual aorta was intact after the operation in group I. On the other hand, patients in groups II, III, and IV had residual aortic dissection after the operation. Eighteen of 36 patients who survived initial hospitalization with aortic dissection required 32 subsequent operations. The actuarial freedoms from reoperation among these patients were 68.8% ± 8.3% at 5 years, 32.7% ± 10.6% at 10 years,

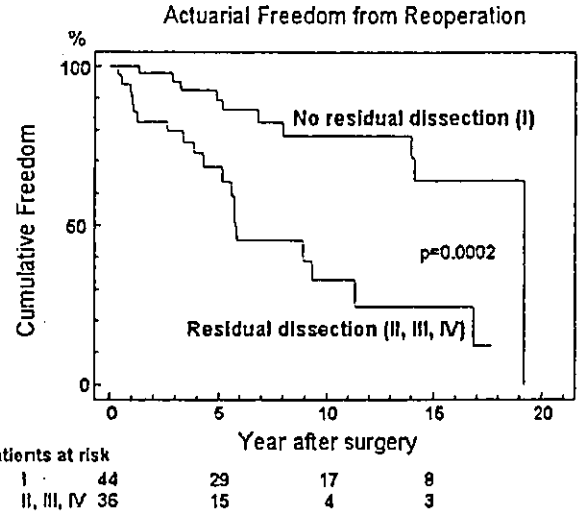

Figure 4. Actuarial freedom from reoperation in patients with and without residual dissection, showing significant difference in actuarial freedom from reoperation ($P = .0002$) between groups.

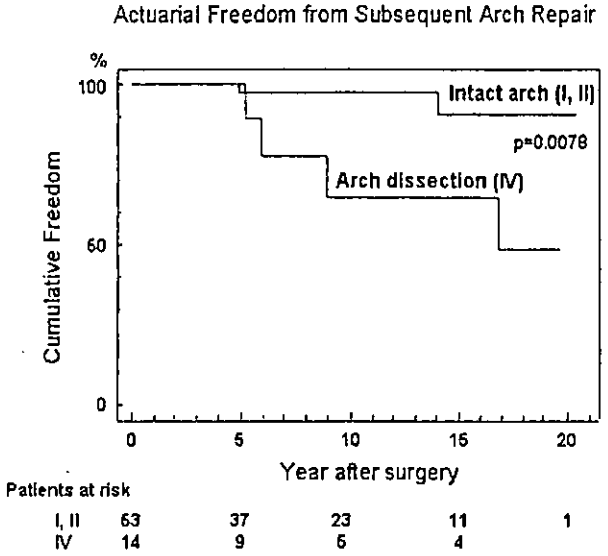

Figure 5. Actuarial freedom from reoperation for aortic arch in the patients with and without arch dissection, showing significant difference between groups ($P = .0078$).

and 24.6% ± 10.7% at 15 years (Figure 4). The linearized rate of reoperation was 10.2 ± 14.7 events per 100 patient-years among the 36 patients who survived initial hospitalization. There were significant differences in the actuarial freedom from reoperation ($P = .0002$) and linearized rate of reoperation ($P = .0028$) between the patients with and without aortic dissection.

Reoperation in patients with concomitant total arch replacement versus patients with aortic root replacement alone. Between patients with concomitant total arch replacement (group III) and patients with aortic root replacement alone (group IV), there were no significant differences in the actuarial freedom from reoperation ($P = .177$) and the linearized rate of reoperation ($P = .900$).

Reoperation for aortic arch in patients with arch dissection versus patients without arch dissection. As mentioned previously, the residual aortic arch was intact in groups I and II. Only 2 patients among the 53 group I and II patients who survived initial hospitalization underwent subsequent total arch replacement for the onset of aortic arch dissection. The actuarial freedoms from operation for the aortic arch were 97.4% ± 2.5% at 10 years and 90.5% ± 7.1% at 20 years (Figure 5). There was a significant difference ($P = .0078$) in the actuarial freedom from operation for the aortic arch between patients with a residual intact arch (groups I and II) and patients with arch dissection (group IV).

Late Survival and Late Outcome

There were 14 late deaths (Figure 2). In group I, 7 late deaths were due to intracerebral bleeding (n = 1), septicemia (n = 1), liver cancer (n = 1), renal failure (n = 1), tuberculosis (n = 1), and unknown causes (n = 2). In group II, 1 patient died at the second-stage operation of descending thoracic aorta replacement for type B dissection. In group III, 2 late deaths occurred as a result of bowel ischemia subsequent to malperfusion from residual dissection and an unknown cause. In group IV there were 4 late deaths. Two patients died of rupture of the residual dissection. One patient died at the second-stage operation for type B dissection. One patient died at the fourth-stage operation for graft replacement of the entire aorta. Overall actuarial survivals were 85.4% ± 3.9% at 5 years, 80.0% ± 4.7% at 10 years, 73.6% ± 6.2% at 15 years, and 60.6% ± 10.3% at 20 years. Actuarial survivals of individual groups are shown in Figure 6.

Late survival in patients with residual aortic dissection versus patients without residual aortic dissection. All the patients in groups II, III, and IV had residual aortic dissection after the initial operation. Among these patients, the actuarial survivals were 83.4% ± 6.3% at 5 years, 79.6% ± 7.0% at 10 years, 71.6% ± 9.9% at 15 years, and 57.3% ± 15.0% at 20 years (Figure 7). On the other hand, actuarial survivals in group I were 86.9% ± 5.0% at 5 years, 80.2% ± 6.5% at 10 years, 74.5% ± 8.2% at 15 years, and 67.0% ± 10.2% at 20 years (Figure 7). There was no significant difference between group I and the other groups combined ($P = .7657$).

Discussion

Aortic root dilatation, with subsequent aortic valve regurgitation, aortic dissection, or rupture, is a common and morbid cardiovascular abnormality in Marfan syndrome.

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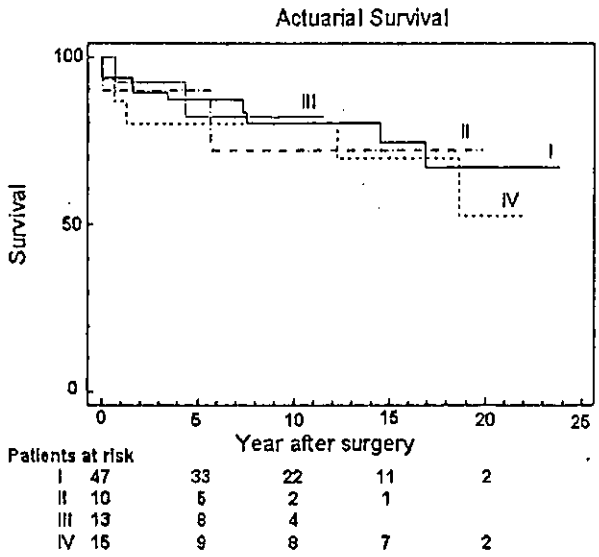

Figure 6. Actuarial survival in each group.

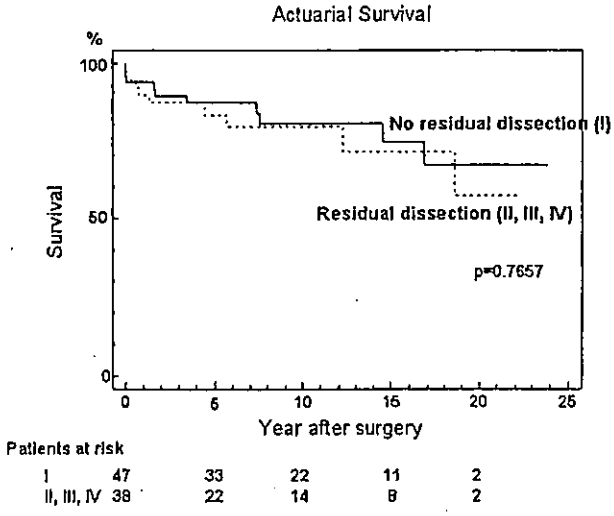

Figure 7. Actuarial survival in patients with and without residual dissection, showing no significant difference ($P = .7657$) between groups.

The introduction of aortic root replacement with a composite graft by Bentall and De Bono[11] in 1968 significantly improved the surgical results in such cases. In recent years, elective surgery has been performed with an operative risk below 5% to provide a full recovery and a normal lifestyle.[5-7,12] In our series of operations on patients with Marfan syndrome, the mortality among patients with aortic root replacement between 1990 and 2002 was 4.2% (2/48), which was almost half (8.1%) that in earlier experiences between 1977 and 1989. Moreover, the life expectancy of patients with Marfan syndrome has significantly improved (actuarial survivals were 80% at 10 years and 61% at 20 years).

However, the abnormal aortic tissue in Marfan syndrome requires multiple surgical reconstructions, and the quality of life of patients with Marfan syndrome is significantly restricted by repeated operations. Of all 85 patients in this study, 28 (32.9%) required a total number of 47 subsequent staged operations. This was more common among patients with residual aortic dissection after the initial operation. In this study, the actuarial freedom from reoperation among patients with residual dissection (group II, III, IV) was significantly lower than that among patients without residual dissection (group I). The linearized rate of reoperation was also significantly higher. Unfortunately, this trend of reoperation was immutable even in patients who received concomitant total arch replacement for associated type A dissection. Among patients with aortic dissection in the aortic arch, there was no significant difference in the actuarial freedom from reoperation ($P = .177$) and the linearized rate of reoperation ($P = .900$) between patients with concomitant total arch replacement (group III) and patients with aortic root replacement alone (group IV). Indeed, more than 60% of patients with Marfan syndrome require a reoperation within 10 years, irrespective of whether the total aortic arch is replaced or not. These results showed that concomitant total arch replacement was therapeutic but not curative, because multiple reentries in the fragile dissected septum in Marfan syndrome disturbed the thrombosed closure of the residual false channel.[13-15] On the basis of these results, controversy still continues regarding whether concomitant total arch replacement is necessary for a dissecting aortic arch.[16,17] Concerning the second-stage operation, however, it is generally accepted that replacement of the descending thoracic aorta through left thoracotomy is preferable to total arch replacement through median resternotomy. Additionally, regarding the staged operation for entire aorta replacement, concomitant total arch replacement is more advantageous than aortic root replacement alone. Among our patients, 4 patients in group III had a complete aortic reconstruction with a total of 12 operations, whereas 3 patients in group IV had complete aortic reconstruction with a total of 12 operations (Figure 2). We therefore recommend total arch replacement for type A dissection involving the aortic arch simultaneously with aortic root replacement for annuloaortic ectasia as long as the patient's condition permits this.

Another point highlighted by this study is the late fate of the residual intact arch after aortic root replacement with abnormal tissue, that is, the potential risk of dissection or rupture in the future. This is a key to answering the primary question of whether the intact aortic arch should be replaced prophylactically and aggressively at the time of aortic root replacement for annuloaortic ectasia.

In general, the cause of aortic dissection is the process of injury and repair of the aortic media by the turbulence of blood flow.[18] The initial intimal tear of a dissection is most frequently situated within the first few centimeters of the ascending aorta.[19] In Marfan syndrome, aortic dissection sometimes occurs in a normal-sized ascending aorta, although the aortic root and sinus of Valsalva are enlarged.[8] On the basis of our results, we propose the possible mechanism of a tear in ascending aorta among patients with Marfan syndrome as follows. Turbulence in the ascending aorta is exacerbated by both the velocity of ejected blood and the interface between the ejected blood and the relatively stagnant blood. However, in a normal aortic root, the position of the leaflets helps to reduce turbulence by masking the dilated sinuses and producing a uniform diameter above the ventriculoarterial junction when blood is ejected through the valve orifice.[20-24] In contrast, in a gourdlike aortic root, which is typical of annuloaortic ectasia in Marfan syndrome, this mechanism is not effective. The high-velocity flow of ejected blood reaches the ascending aortic wall with medial degeneration, and turbulence occurs at the junction between the ascending aorta and the dilated sinus of Valsalva because the leaflets cannot mask the dilated sinus of Valsalva (Figure 8).[25] Another factor in dissection is the different tensile strength of the aortic wall itself, which depends on the content of elastic fibers and collagen. The contents of elastic fibers and collagen differ between the ascending aorta and the sinus of Valsalva[26-30] and also between the inner and outer layers of the aortic wall itself. This leads to the "breaking point" of the internal layer in the ascending aorta during aortic dilatation.[21] Therefore these two major factors will be eliminated by composite graft replacement of the dilated sinus of Valsalva and the proximal ascending aorta. This hypothesis is supported by the long-term results of patients with an intact arch in our study. The incidence of new dissection in the residual intact arch after aortic root replacement was extremely low; only 2 of the 53 patients who survived initial hospitalization with an intact arch underwent subsequent total arch replacement for the onset of arch dissection, whereas 4 of the 14 patients who survived initial hospitalization with dissection in the aortic arch underwent subsequent total arch replacement. These results show that prophylactic replacement of the intact aortic arch is not necessary at the time of aortic root replacement for annuloaortic ectasia, because aortic root replacement itself plays a prophylactic role in aortic dissection.

The major limitation of our study was that the time scale of this study ranged through 22 years. During this period mortality and morbidity in aortic surgery were clearly improved by refinements in surgical technique and perioperative management. Additionally, a large proportion of the patients required ongoing treatment and follow-up. Another limitation of this study was that the numbers of patients in each group were too small to ascertain any statistically significant difference between groups for operative mortality or the necessity for reintervention.

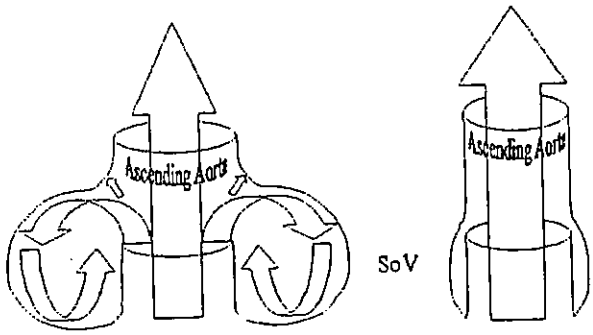

Figure 8. ***Right,*** **Normal aortic root. Position of leaflets helps to reduce turbulence by masking dilated sinus to produce uniform diameter above ventriculoarterial junction when blood is ejected through valve orifice.** ***Left,*** **Typical annuloaortic ectasia in Marfan syndrome. High-velocity flow of ejected blood reaches ascending aortic wall with medial degeneration, and turbulence occurs at junction between ascending aorta and dilated sinus of Valsalva** ***(SoV)*** **because leaflets cannot mask dilated sinus.** ***Arrows*** **indicate blood flow. Sinus of Valsalva has less elastic fiber than collagen; ascending aorta has more elastic fiber than collagen.**

In conclusion, the incidence of new dissection in the residual intact arch after aortic root replacement is extremely low, because aortic root replacement may remove the factors provoking dissection. According to our results, prophylactic replacement of the intact arch in Marfan syndrome is not necessary during aortic root replacement for annuloaortic ectasia.

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Discussion

Dr Nicholas Kouchoukos *(St Louis, Mo).* In their study, Tagusari and colleagues have focused on the question of whether the aortic arch should be replaced at the time of replacement of the dilated aortic root in patients with Marfan syndrome. Among the 57 patients with an intact arch at the time of aortic root replacement, only 2 required subsequent arch replacement for new-onset arch dissection. The actuarial freedom of reoperation in this group was 90% at 10 years. These observations are in agreement with the results of other large series of patients with Marfan syndrome and an intact arch who have undergone only aortic root replacement. The aggregate experience strongly suggests that replacement of the arch is not indicated in this setting.

What is less clear is whether the aortic arch should be replaced at the time of aortic root replacement in patients with acute or chronic type A dissection. Albeit the number of patients with dissection in this series was small, 29 patients, no difference in survival or in freedom from any operation was observed at 10 years between the 15 patients undergoing only aortic root replacement and the 13 patients who had aortic root and simultaneous arch replacement. However, the rate of reoperation on the aortic arch was significantly higher among the patients with dissected arch who underwent only aortic root replacement than among the patients with an intact arch. This suggests that replacement of the dissected arch may be advantageous.

Only 10 of the 46 patients with dissection in this series had acute dissection. Tagusari and colleagues did not examine outcomes in this subgroup, presumably because of small numbers. However, the decision to replace the aortic arch in the presence of acute dissection may be associated with higher risk than if the dissection is chronic.

I have several questions for Dr Tagusari. First, on the basis of your findings, what is your current strategy for management of patients undergoing aortic root replacement who have a type A dissection?

Dr Tagusari. We perform aortic root replacement with composite graft and total arch replacement.

Dr Kouchoukos. Do you manage the patients with acute dissection in this setting any differently than you manage the patients with chronic dissection?

Dr Tagusari. In general, the patient with Marfan syndrome who has an aortic dissection is young. Accordingly, we should perform total arch replacement simultaneously to save further operation.

Dr Kouchoukos. Do you recommend complete aortic arch replacement in the setting of acute dissection?

Dr Tagusari. Yes.

Dr Kouchoukos. A valve-sparing procedure was performed in 17 of the 86 patients in the series. How much of the ascending aorta was replaced in these patients? Do you believe, from your experience to date, that this is a durable procedure in patients with Marfan syndrome?

Dr Tagusari. For the patient with Marfan syndrome, I prefer a composite graft replacement to valve-sparing operation, especially a remodeling procedure, because in our histologic findings the aortic valve showed concentric layering of collagen fibers mixed with glycosaminoglycans. This means severe degeneration of the leaflet itself. Actually, 4 of the 17 patients who underwent valve-sparing operations (2 of 13 reimplantations and 2 of 4 remodelings) needed aortic valve replacement.