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Predictors of Aneurysmal Formation After Surgical Correction of Aortic Coarctation

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OBJECTIVES We sought to identify the predictors of aneurysmal formation after surgical correction of aortic coarctation.

BACKGROUND In 9% of patients, aneurysms develop late after corrective surgery of coarctation of the aorta, with a 36% mortality rate if left untreated. However, the predictors of postsurgical aneurysmal formation are unknown.

METHODS Of 25 aortic aneurysms requiring corrective surgery 152 ± 78 months after the initial coarctation repair, 8 were located in the ascending aorta (type A) and 17 at the site of previous repair (local type). Seventy-four patients without progression of the aortic diameter within 189 ± 71 months after coarctation repair were used for categorical data analysis in an attempt to identify the predictors of postsurgical aneurysmal formation.

RESULTS Advanced age at coarctation repair ($p = 0.004$) and patch graft technique ($p < 0.0005$) independently predicted local aneurysmal formation. Type A aneurysm was univariately associated with the presence of a bicuspid aortic valve ($p = 0.02$), advanced age at coarctation repair ($p = 0.044$) and a high preoperative peak systolic pressure gradient of 74 ± 21 mm Hg ($p = 0.041$). Conversely, multivariate analysis confirmed only the presence of a bicuspid aortic valve ($p = 0.015$) as an independent predictor of type A aneurysm. Receiver operating characteristic curve analysis revealed that 72% of patients with a postsurgical aneurysm had an operation at age 13.5 years or more, whereas 69% with no postsurgical aneurysm had an operation at a younger age.

CONCLUSIONS Use of the patch graft technique and late correction of coarctation can predict aneurysmal formation at the site of coarctation repair, whereas patients with a bicuspid aortic valve may be at risk for an aneurysm developing in the ascending aorta, particularly after late repair of aortic coarctation with high preoperative pressure gradients. (J Am Coll Cardiol 2002;39: 617–24) © 2002 by the American College of Cardiology

Severe coarctation of the thoracic aorta is usually corrected by surgical repair. Despite primary success, however, 9% of patients develop aortic aneurysms late after the operation, with aortic rupture and a lethal outcome reported in some (1–15). To avoid late aneurysmal formation, patients at risk require early identification. A postsurgical aneurysm was observed with subclavian flap angioplasty (17%), patch angioplasty (14%), tube graft repair (6%) or end-to-end anastomosis (3%) (1–15), often associated with persistent systemic hypertension, concomitant cardiovascular malformations or use of Dacron and/or silk sutures (6–9,11), and presented as false, true or dissecting at various sites of the thoracic aorta (3,8,9). With this wide scope of potential risk factors, the prediction of aneurysmal formation after coarctation repair is not feasible to date; thus, this controlled, retrospective analysis of data banks at three cardiovascular centers was conducted to design models for predicting aneurysms both in the ascending and descending aorta.

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METHODS

Patients. Between January 1989 and December 1999, 1,665 patients with surgical repair of a dissecting or true aneurysm of the ascending aorta (1,200 patients), the descending aorta (383 patients) or the aortic arch (82 patients) were registered at the Departments of Cardiovascular Surgery at the University Hospital Hamburg-Eppendorf, the Hannover Medical School and the Christian-Albrechts-University, all in Germany. Twenty-five patients (1.5%) with previous surgical repair of coarctation of the aorta and late aneurysmal formation were extracted from this database. The location, extent and diameter of the postsurgical aneurysm were assessed from tomographic imaging or angiography, or both, performed before the operation (16,17) and from surgical records on aneurysm repair. An urgent operation was performed for acute aortic dissection (Patient nos. 1 and 2) (Table 1), rapid progression (Patient nos. 4 and 15), rupture of an aneurysm (Patient nos. 16, 17 and 20) or marked aortic root enlarge-

Abbreviations and Acronyms
BP = blood pressure
CBAV = congenitally bicuspid aortic valve
ROC = receiver operating characteristic

ment with aortic regurgitation (Patient nos. 3 and 5 to 8); elective repair was performed in the remaining patients. Aneurysm in the ascending aorta (termed "type A") was repaired using the composite graft technique with re-implantation of coronary arteries (Patient nos. 2 and 4 to 7), a supracoronary tube graft (Patient nos. 1, 3 and 8), a biologic aortic valve prosthesis (Patient no. 4) or a bi-leaflet mechanical valve (17). Aneurysm in the descending thoracic aorta (termed "local" type) was repaired by resection of the dilated aortic segment and insertion of a tube graft (Patient nos. 9 to 25).

In the attempt to identify the stratifiers of risk for postsurgical aneurysmal formation, we studied a control group of 74 consecutive patients (40 men and 34 women; 11 ± 14 years of age at index operation) who had successful repair of aortic coarctation within the same time interval as the study patients; 38 of these patients had end-to-end anastomosis, 21 received patch grafts and 15 had a tube graft for coarctation repair (Table 2). This group had an uneventful follow-up of 189 ± 71 months, with exclusion of postsurgical complications, aortic wall abnormalities, aortic diameters exceeding 3.5 mm and progression of diameters, as assessed by serial tomographic imaging (e.g., transesophageal echocardiography, computed tomography, magnetic resonance imaging) during repeat follow-up visits (16,17). Patients with inflammatory cardiovascular disease and complex, concomitant cardiovascular malformations were not included (e.g., Turner's syndrome, Marfan syndrome, transposition of the great arteries, dextrocardia, ventricular septal defect, double-outlet right ventricle). Complete clinical and morphometric documentation and follow-up data were available for all patients (Table 1). Any cardiovascular event requiring hospital admission during follow-up after a repeat intervention for postsurgical aneurysmal repair was considered a "cardiovascular event."

Study variables. Sixteen variables were assessed, including: 1) age at the time of surgical correction of coarctation and 2) gender (Table 2). 3) Coarctation of the aorta was considered as ductal, preductal or postductal, with abrupt narrowing of the aorta at, proximal to or distal to the site of the ductus arteriosus, respectively (18). 4) The surgical technique of coarctation repair and 5) the presence of previous surgical repair of coarctation were assessed in each patient. Rest right arm blood pressure (BP) was assessed by sphygmoma-

Table 1. Patients With Aneurysm After Surgical Coarctation Repair

Pt. No.*	Age at Repair (yrs/gender)	Coarctation Repair to Aneurysm (months)	Type of Coarctation Repair	SBP (mm Hg)		PSG (mm Hg)		Aneurysm		Alive	FU (months)
				Preop.	Postop.	Preop.	Postop.	Type	Diameter (mm)		
1	24/M	122	End	130	160	90	0	Diss.	112	Y	152
2	31/M	107	Patch g.	180	140	55	0	Diss.	85	Y	89
3	16/M	229	Tube g.	210	180	82	0	True	80	Y	101
4	19/M	40	Tube g.	190	120	110	0	True	110	Y	145
5	36/F	216	End	195	140	85	0	True	90	Y	59
6	12/M	120	End	175	150	64	0	True	70	Y	132
7	12/M	246	End	176	135	60	0	True	50	Y	93
8	19/M	141	Tube g.	230	140	50	0	True	70	Y	108
9	24/M	254	End	180	150	60	38	False, rest	35	Y	17
10	16/M	288	Patch g.	180	121	45	45	False, rest	40	Y	16
11	15/M	119	Patch g.	210	170	45	55	False, rest	70	Y	120
12	11/F	112	Patch g.	150	140	40	0	False, sut.	50	Y	72
13	12/F	230	Tube g.	170	160	90	12	False, sut.	30	Y	46
14	13/M	169	Patch g.	125	150	60	0	False, sut.	80	Y	12
15	24/M	189	Patch g.	170	125	40	0	False, sut.	60	Y	75
16	28/M	179	Patch g.	160	120	80	0	False, sut.	50	N	OP
17	15/F	48	Patch g.	130	150	50	0	False, sut.	65	N	120†
18	24/M	97	Patch g.	210	140	65	0	True, opp.	64	Y	64
19	6/M	120	Patch g.	140	138	50	0	True, opp.	55	Y	23
20	16/M	187	Patch g.	180	120	100	0	True, opp.	100	Y	32
21	11/M	218	Patch g.	170	140	70	0	True, opp.	65	Y	56
22	12/F	88	Patch g.	170	110	90	0	True, arch	44	Y	396
23	35/M	211	Tube g.	200	200	60	5	True, arch	—	Y	154
24	30/M	60	Patch g.	200	120	40	0	True, arch	55	Y	50
25	14/F	11	Tube g.	110	100	40	0	True, arch	30	Y	108

*Patient nos. 1 to 8 had type A aneurysms and Patient nos. 8 to 25 had local type aneurysms. †This patient died during an operation for a recurrent anastomotic aneurysm.
Diss. = dissection; end = end-to-end anastomosis; F/U = time from coarctation repair to last follow-up; g. = graft; N = no; OP = intraoperative death; opp. = opposite a patch graft; preop. = preoperative; postop. = postoperative; PSPG = peak systolic pressure gradient; SBP = systolic blood pressure; sut. = aneurysm from suture line; Y = yes.

Table 2. Patients With and Without Aneurysm After Coarctation Repair

Variable	Postsurgical Aneurysm		Control Subjects (n = 74)	p Value		
	Type A (n = 8)	Local (n = 17)		Global Test*	Type A vs. C†	Local vs. C†
1) Age at coarctation repair (years)	21 ± 9	18 ± 8	11 ± 14	0.012	0.044	0.049
2) Male gender	7	12	40	0.124		
3) Type of coarctation				0.432		
Ductal	2	8	36			
Postductal	6	7	27			
Preductal	0	2	11			
4) Type of coarctation repair				0.0004	0.427	0.0003
End-to-end anastomosis	4	1	38			
Patch graft (Dacron)	1	13 (10)	21 (1)			
Tube graft (Dacron)	3 (2)	3	15 (11)			
5) Previous coarctation repair	0	0	7	0.494		
Preoperative BP (mm Hg)						
6) Systolic—N > 90%	8 (186 ± 29)	16 (168 ± 29)	65 (144 ± 46)	0.611		
7) Diastolic—N > 90%	7 (99 ± 10)	16 (97 ± 12)	55 (80 ± 31)	0.183		
8) PSPG	74 ± 21	60 ± 20	52 ± 27	0.048	0.041	0.418
Postoperative BP (mm Hg)						
9) Systolic—N > 90%	6 (146 ± 18)	9 (138 ± 24)	31 (137 ± 22)	0.172		
10) Diastolic—N > 90%	2 (82 ± 12)	6 (79 ± 14)	18 (80 ± 13)	0.629		
11) PSG	0	9 ± 18	17 ± 19	0.026	0.03	0.233
12) Presence of CBAV	7	3	30	0.003	0.02	0.097
13) Presence of PDA	0	1	21	0.045	0.106	0.062
14) Family history	1	0	3	0.402		
15) Postoperative beta-blockers	2	6	10	0.092		
16 Postoperative LVH	4	4	17	0.296		

*Freeman-Halton test for categorical variables and analysis of variance for continuous variables. †$p < 0.05$ in the global test; the Dunnett test was performed for continuous variables, the Freeman-Halton test for categorical variables. Data are presented as the mean value ± SD or number of patients.

A vs. C indicates comparison of type A group versus control group; CBAV = congenitally bicuspid aortic valve; local vs. C = comparison of local type group versus control group; BP = blood pressure; LVH = left ventricular hypertrophy; N > 90% = number of patients in >90% percentile; PDA = patent ductus arteriosus; PSPG =peak systolic pressure gradients.

nometry on three different days before coarctation repair and during follow-up visits. Both systolic and diastolic BPs were given as the average of three preoperative and three postoperative measurements. Systolic BP (variables 6 and 9) and diastolic BP (variables 7 and 10) were elevated, with measurements above the 90th percentile with age below 18 years and systolic BP >140 mm Hg and with age above 18 years and diastolic BP >90 mm Hg (19,20). Peak systolic pressure gradients were recorded from percutaneous retrograde catheterization as the intra-aortic pullback pressure across either the coarctation or the site of coarctation repair. In 10 control subjects and 3 patients (Patient nos. 21, 24 and 25), estimates of preoperative pressure gradients were derived from pulsed Doppler flow studies (variables 8 and 11) (21). 12) The presence of a congenitally bicuspid aortic valve (CBAV) was assessed by echocardiography and from surgical records (Patient nos. 1 to 8). 13) The presence of patent ductus arteriosus was documented on preoperative cardiac catheterization or color flow Doppler mapping. 14) Each patient had a clinical and echocardiographic examination of his/her kindred for the presence of coarctation (21). 15) Patients were considered to be under postoperative beta-receptor blockade with oral administration of atenolol or metoprolol over at least six months after coarctation repair; 16) left ventricular hypertrophy was assessed at least six months after the operation (22).

Review of published data. Using MEDLINE (key words: aneurysm, aorta, coarctation and surgery), the English and German published data were screened for records on both the prevalence and outcome of aneurysmal formation after surgical correction of coarctation. Reports from series including at least 20 patients with surgical correction of coarctation were included in this analysis, as shown in Table 3.

Statistical analysis. The relationship between developing aneurysms after coarctation repair and each of the 16 variables was evaluated for aneurysms in the ascending aorta (type A) and descending aorta (local) and for the control group, using analysis of variance for continuous variables and the Freeman-Halton test for nominal and categorical variables. With the global test significant at $p < 0.05$ (two-sided), the Dunnett test was performed for post hoc multiple comparisons of continuous variables between both type A and local aneurysm groups and the control group; categorical variables were compared using the Freeman-Halton test with the Bonferroni correction. Only variables with statistical significance set at $p < 0.05$ (two-sided) for these univariate tests were included in a multivariate logistic regression model. Estimates of risk (odds ratio) were calculated based on coefficients from the logistic models. Receiver operating characteristic (ROC) curve analysis was performed to assess the patient's age at coarctation repair as

Table 3. Reported Prevalence of Aortic Aneurysm After Coarctation Repair

Reference	Patients With Aortic Aneurysm After Coarctation Repair/Patients With Coarctation Repair					Death in Patients With Aortic Aneurysm After Aneurysmal Repair	
		Type of Coarctation Repair					
	All Procedures	Subclavian Flap	Patch Graft	Tube Graft	End-to-End A.	Not Performed	Attempted/ Performed
Pennington et al. (1)	4/164	—	—	2/59	2/92	1/4	—
Sørland et al. (2)	1/138	—	—	—	1/138	1/1	—
Clarkson et al. (3)	8/73	—	5/52	3/21	—	1/5	—
Hehrlein et al. (4)	18/303	—	18/285	0/8	0/10	2/3	2/15
Del Nido et al. (5)	3/63	—	3/63	—	—	—	0/3
Rheuban et al. (6)	8/79	0/5	8/45	0/3	0/26	—	—
Koller et al. (7)	5/343	—	—	5/47	0/296	—	1/5
Ala-Kulju and Heikkinen (8)	22/67	—	22/67	—	—	—	0/22
Bromberg et al. (9)	7/29	—	7/29	—	—	—	—
Kron et al. (10)	10/197	0/58	9/56	1/7	0/76	—	0/5
Pinzon et al. (11)	64/215	29/92	9/26	—	26/97	—	—
Bogaert et al. (12)	33/73	—	33/73	—	—	1/33	—
Parks et al. (13)	20/39	—	20/39	—	—	6/20	—
Knyshov et al. (14)	48/891	0/10	43/494	1/32	43/333	18/18	4/30
Heger et al. (15)	0/37	—	0/9	0/7	0/21	—	—
Total (%)	251/2,711 (9)	29/165 (17)	177/1,238 (14)	12/184 (6)	33/1,089 (3)	30/84 (36)	7/80 (9)

A. = anastomosis; — = no information available.

a discriminator of increased (from low) risk for postsurgical aneurysm. Any cardiovascular event requiring hospital admission was analyzed by the Kaplan-Meier method separately for aneurysms in the ascending and descending aorta, with the log-rank test for comparison of both curves; Patient no. 17 developed an aneurysm in the ascending aorta after repair of a postsurgical aneurysm in the descending aorta and, thus, could not be assigned to either group. Data are presented as the mean value ± SD or frequencies. Statistical analysis was performed using SPSS software (SPSS for Windows, Release 10.0.7, SPSS Inc. 1989 to 1999, Chicago, Illinois).

RESULTS

Types of postsurgical aneurysm. Reoperation for aortic aneurysmal formation was performed at 153 ± 71 months (type A, located in the ascending aorta) and 152 ± 78 months (local type, located in the descending aorta) after the index coarctation repair ($p = 0.98$). Type A aneurysms developed a dissection in two patients (Patient nos. 1 and 2), and six patients had moderate (Patient nos. 5 and 8) or severe aortic valve regurgitation (Patient nos. 1 to 4). With one exception, the diameters of the ascending aorta were at least 70 mm (Patient no. 7) (Fig. 1), and marked (Patient nos. 1, 2, 4 and 5) or moderate (Patient nos. 7 and 8) media degeneration was present in all but one aortic specimen obtained during aneurysm repair (Patient no. 3). Local aneurysms developed proximal to recurrent isthmic stenosis (Patient nos. 9 to 11), at suture lines (Patient nos. 12 to 17), with rupture in two patients (Patient nos. 16 and 17), or at or adjacent to the aortic patch (Patient nos. 18 to 21) or with tube grafts extending into the aortic arch (Patient nos. 22–25). In type A aneurysms, aortic diameters were larger (83 ± 21 mm vs. 56 ± 18 mm in the local type; $p = 0.003$), CBAVs were more prevalent (Patient nos. 1, 2, 4 to 8, 12, 21 and 24; $p = 0.002$) (Table 2) and emergency repair was slightly more common, as compared with local aneurysms (37% vs. 23%; $p = 0.64$).

Postsurgical follow-up of aneurysmal repair was 110 ± 31 months for type A aneurysms and 80 ± 92 months for local aneurysms. The five-year actuarial freedom from cardiovascular events (±SE) was slightly lower in type A than in local aneurysms (75 ± 15% vs. 100%; $p = 0.08$) (Fig. 2). Cardiovascular events in surgically corrected type A aneurysms included cerebral bleeding after oral anticoagulation (Patient no. 5), acute coronary syndrome (Patient no. 6), recurrent aortic aneurysm (Patient no. 24) and aortic valve incompetence (Patient nos. 2, 4 and 8). Perioperative mortality (30-day), however, was similar in both types of aneurysms (0 in type A aneurysms vs. 1 intraoperative death in local aneurysms; $p = 0.48$).

Predictors of postsurgical aneurysm. Although preoperative and postoperative BP, anatomic type of coarctation, use of Dacron material and concomitant cardiovascular abnormalities were not associated with postsurgical aneurysms, univariate analysis identified advanced age at the index coarctation repair ($p = 0.044$), a bicuspid aortic valve ($p = 0.02$) and high preoperative peak systolic pressure gradients (74 ± 21 mm Hg in patients with type A aneurysms vs. 52 ± 27 mm Hg in control subjects; $p = 0.041$) as risk factors of type A aneurysms (Table 2). Multivariate analysis, however, corroborated only a bicuspid aortic valve ($p = 0.015$) as an independent predictor of type A aneurysmal formation (Table 4). Similarly, the use of patch grafts ($p <$

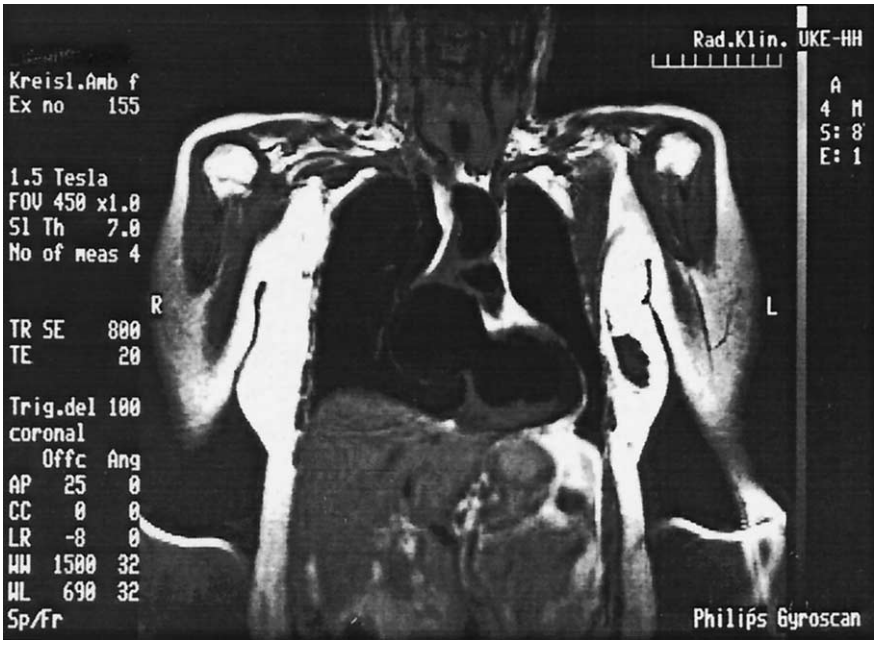

Figure 1. Spin-echo magnetic resonance scan of marked type A aneurysmal formation after surgical correction of aortic coarctation in a patient with a bicuspid aortic valve (Patient no. 5; Table 1).

0.0005) and advanced age at the index coarctation repair ($p = 0.004$) emerged as independent predictors of a local aneurysm (Table 4).

Receiver operating characteristic curve analysis revealed 13.5 years of age at coarctation repair as the threshold for increased risk of postsurgical aneurysmal formation ($p < 0.0005$) (Fig. 3). Seventy-two percent of patients with a postsurgical aneurysm were surgically repaired beyond the critical age of 13.5 years, whereas 69% of patients with uneventful cases were repaired under the age of 13.5 years.

DISCUSSION

Formation of an aortic aneurysm after corrective surgery for coarctation was previously reported in 9% of patients, irrespective of the technique of surgical correction (1–15) (Table 3). Our retrospective analysis identified 17 cases of

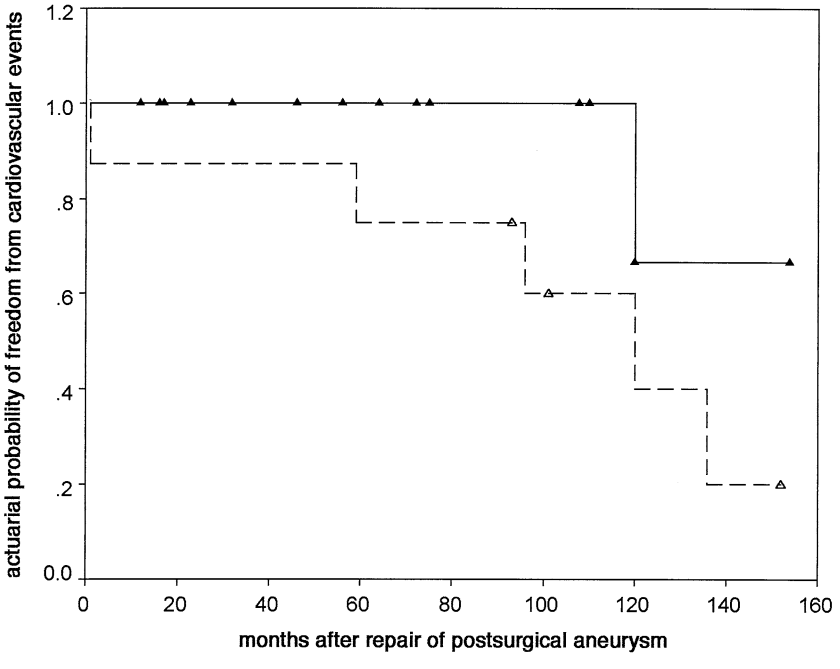

Figure 2. Actuarial probability of event-free survival of patients with postsurgical aneurysms after repair of aortic coarctation. There is a trend toward a higher probability of cardiovascular events in type A aneurysms (**dashed line, open triangles** = censored cases) than in local aneurysms (**solid line, solid triangles** = censored cases) ($p = 0.08$). The mean event-free survival time was 104 months (95% confidence interval 70 to 137).

Table 4. Multivariate Analysis

Variable	OR	95% CI	p Value
Type A aneurysm			
Presence of CBAV	21.82	1.84–258.8	0.015
Preoperative PSPG	1.04	0.99–1.08	0.058
Local aneurysm			
Patch graft repair	18.49	3.62–94.38	< 0.0005
Age at coarctation repair	1.08	1.02–1.13	0.004

*Age at coarctation repair (p = 0.316) and postoperative PSG (p = 0.888) were not significant; CI = confidence interval; OR = odds ratio; see Table 2 for other abbreviations.

postsurgical aneurysm located in the descending aorta at the site of previous coarctation repair among a total of 383 surgically corrected descending aortic aneurysms (4.4%) and 8 aneurysms in the ascending aorta after coarctation repair among 1,200 surgically corrected aneurysms in the ascending aorta (0.7%). These aneurysms were subjected to corrective surgery 152 ± 78 months after the initial coarctation repair. The outcome of postsurgical aneurysms was adverse: death occurred in 36% of patients if left untreated and in 9% even after surgical resection, whereas only 4% of patients had an early death after surgical repair of a postoperative aneurysm. The results from this analysis are in agreement with previous reports (1–15) (Table 3).

Type A aneurysms. Among a total of 25 aneurysms after coarctation repair, a significant proportion (n = 8) developed in the ascending aorta (32%; type A), with extensive aortic widening of 88 ± 31 mm; 6 of them revealed moderate or severe aortic valve regurgitation (75%), 2 were associated with dissection (25%) and 3 required emergent repair (37%). Although there were no early or late fatalities 110 ± 31 months after aneurysmal resection, the rate of

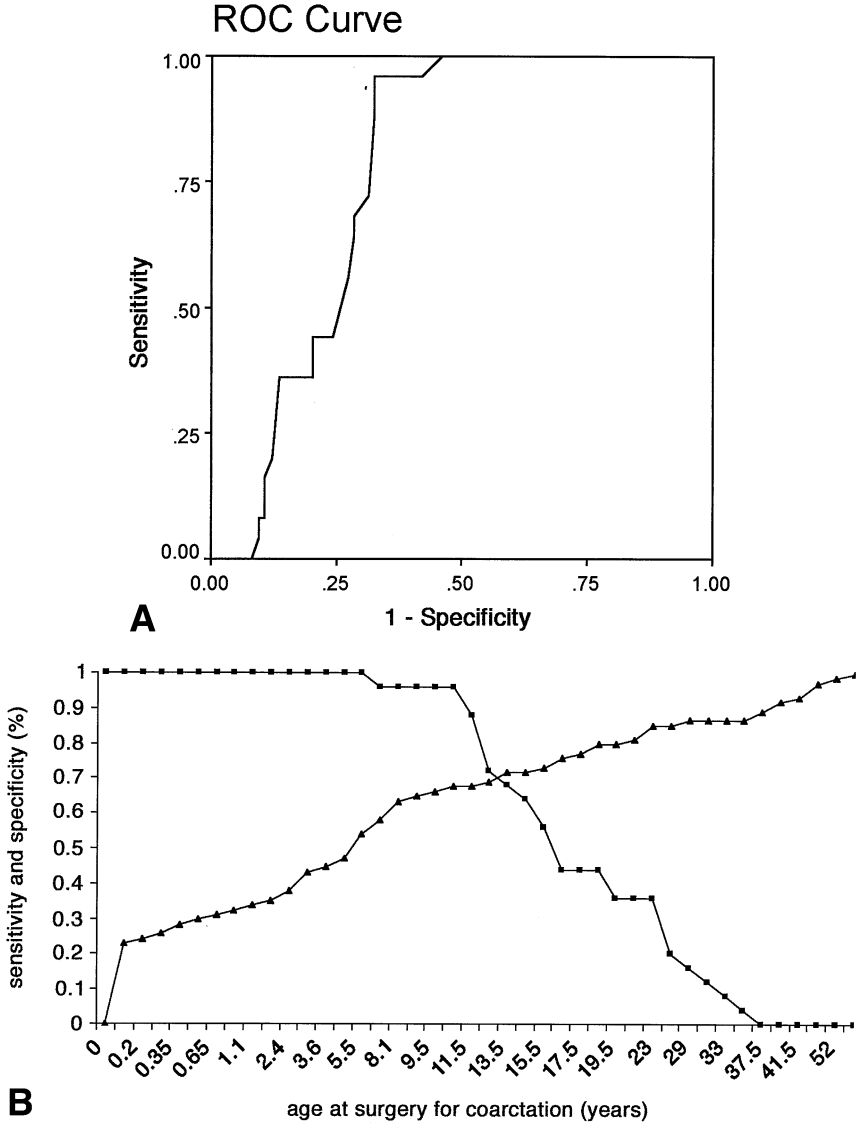

Figure 3. **(A)** Receiver operating characteristic (ROC) curve analysis revealed that 13.5 years of age at the index surgical repair of coarctation was a threshold for increased risk of postsurgical aneurysmal formation. The area under the ROC curve was 0.76 (95% confidence interval 0.67 to 0.85; p < 0.0005). **(B)** For better identification of the cut-off age separating high and low risk, data are displayed separately for sensitivity **(squares)** and specificity **(triangles)**.

postsurgical five-year actuarial freedom from cardiovascular events was only 75 ± 15%.

Type A aneurysms were independently associated with the presence of a CBAV on multivariate analysis (Table 4). The prevalence of a bicuspid aortic valve in our control group was 40%, similar to other studies, although seven bicuspid aortic valves in eight type A aneurysms clearly exceeded a frequency of 21% to 52% reported in coarctation unassociated with Marfan syndrome or Turner's syndrome (7,11,23). Bicuspid aortic valves were often associated with aneurysms, aortic valve leakage or stenosis after coarctation repair (7,11,24). A stenotic aortic valve, however, was not observed in our study. Marked media degeneration was observed in six of seven patients with a type A aneurysm (86%), exceeding a frequency of 50% reported in local aneurysms after coarctation repair (4). Because formation of postsurgical aneurysms was related to advanced age at coarctation repair, chronic systemic hypertension preoperatively may have propelled media degeneration long before widening of the aortic diameter. This notion was supported by a high preoperative peak systolic pressure gradient of 74 ± 21 mm Hg associated with a risk of type A aneurysmal formation. Thus, long-standing, uncorrected severe coarctation was likely to increase the inherent risk of a bicuspid aortic valve to develop aneurysm and dissection of the aortic root.

Local aneurysms. Seventeen of 25 postsurgical aneurysms developed at the site of previous coarctation repair (68%). The present analysis provided statistical confirmation that use of a patch graft was independently associated with late local aneurysmal formation (1–15). Moreover, the current analysis is the first to reveal that advanced age at coarctation surgery (above 13.5 years according to ROC curve analysis) was an independent predictor of late aneurysmal formation. However, different anatomic features of aneurysms indicated various mechanisms of development. Although false local aneurysms arose from suture lines (6 of 9 patients) or at isthmic restenosis (3 of 9 patients), true local aneurysms usually formed opposite to a patch graft (Table 1). In contrast to previous series, however, Dacron was rarely employed (3,9), and silk sutures (1,4,7) or transverse aortic arch hypoplasia (11,12) was not encountered in our study.

Conclusions. Aneurysmal formation is common after coarctation repair and is not exclusively related to patch graft repair. Most notably, postsurgical aneurysms in the ascending aorta (type A) are completely unrelated to surgical techniques. Proximal aneurysms have escaped attention in previous reports, but they represent one-third of aortic aneurysms after coarctation repair. With the high prevalence of severe aortic regurgitation, dissection and rupture, this condition can be life-threatening and continues to carry a high morbidity, even after successful repair. Patients with a bicuspid aortic valve, advanced age at coarctation repair and high preoperative systolic peak pressure gradients are most likely to develop proximal aneurysms. Therefore, regular follow-up imaging should be offered for timing of elective aortic root surgery. Local aneurysms develop at the site of previous coarctation repair and represent two-thirds of postsurgical aortic aneurysms. According to our prediction model, aneurysmal formation at the site of coarctation repair may be prevented by avoiding the patch graft technique and performing repair of coarctation before the age of 13.5 years.

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