SURGERY FOR ADULT CARDIOVASCULAR DISEASE

DISSECTION OF THE ASCENDING AORTA AFTER PREVIOUS CARDIAC SURGERY: DIFFERENCES IN PRESENTATION AND MANAGEMENT

A. Marc Gillinov, MD
Bruce W. Lytle, MD
Richard J. Kaplon, MD
Filip P. Casselman, MD
Eugene H. Blackstone, MD
Delos M. Cosgrove, MD

Objective: This study was undertaken to determine the impact of previous cardiac surgery on the presentation, management, and outcome of late dissection of the ascending aorta. **Patients and methods:** From 1976 to 1998, type A dissection developed in 56 patients with a history of previous cardiac surgery. Interval from first operation to type A dissection was 49 ± 47 months (0.3-180 months). Previous operations were coronary artery bypass grafting (n = 40), aortic valve replacement (n = 8), and other (n = 8). **Results:** Type A dissection was acute in 34 patients and chronic in 22. In acute dissection, aortic insufficiency occurred in 50%, malperfusion in 12%, and rupture in 18%; 2 patients (6%) were in hemodynamically unstable condition because of rupture. Of patients with previous coronary bypass grafting, 98% had preoperative coronary angiography. Type A dissection was treated by supracoronary tube graft (84%), Bentall procedure (14%), or local repair (2%). Strategies for managing previous coronary bypass grafting included reimplantation of proximal anastomoses with a button of native aorta (29 patients), interposition graft to pre-existing saphenous vein grafts (9 patients), and new saphenous vein grafts (20 patients). Eight hospital deaths occurred (14%). **Conclusions:** We conclude that (1) patients having type A dissection late after cardiac surgery infrequently have cardiac tamponade and hemodynamic collapse; (2) patients with previous coronary bypass grafting require coronary angiography, because operative management must account for pre-existing coronary artery disease; and (3) operative mortality is low, and this may be attributable to preoperative hemodynamic stability, delineation of coronary anatomy in those with previous coronary bypass grafting, and operative treatment of coronary artery disease. (*J Thorac Cardiovasc Surg* 1999;117:252-60)

The presentation, management, and outcome of primary dissection of the ascending aorta (Stanford type A or DeBakey type I or II) are well described.¹⁻⁵ The objectives in management of primary type A dissection are rapid diagnosis and prompt surgical treat-

ment, because mortality approaches 50% at 48 hours.^{1,6} In contrast, type A dissection occurring late after previous cardiac surgery presents different and complex issues in diagnosis and treatment. The purpose of this study was to determine the impact of previous cardiac

From the Department of Thoracic and Cardiovascular Surgery, The Cleveland Clinic Foundation, Cleveland, Ohio.

Read at the Twenty-fourth Annual Meeting of The Western Thoracic Surgical Association, Whistler, British Columbia, June 24-27, 1998.

Received for publication June 23, 1998; revisions requested Oct 7, 1998; revisions received Oct 23, 1998; accepted for publication Oct 30, 1998.

Address for reprints: A. Marc Gillinov, MD, Department of Thoracic and Cardiovascular Surgery/F25, The Cleveland Clinic Foundation, 9500 Euclid Ave, Cleveland, OH 44195.

Copyright © 1999 by Mosby, Inc.

0022-5223/99 \$8.00 + 0 12/6/95592

surgery on the presentation, management, and outcome of late type A dissection.

Patients and methods

Patients

Study group. From 1976 to January 1, 1998, 56 patients with previous cardiac surgery underwent surgical repair of type A dissection at The Cleveland Clinic Foundation. They were identified as follows. Initially, the prospective Cardiovascular Information Registry (CVIR) was used to identify all patients having surgery for type A dissection. The patients' medical records were then reviewed in detail, both to select those with previous cardiac surgery who required subsequent treatment of type A dissection and to verify the prospectively acquired CVIR clinical data. The study group was further refined by excluding patients who had previous aortic root surgery or who had recognized intraoperative type A dissection that was treated at the time of recognition. Patients with dissection of the descending aorta (Stanford type B, DeBakey class III) were also excluded.

Definition. Ascending aortic dissection was considered to be present when the dissection involved any portion of the aorta between the aortic anulus and the take-off of the innominate artery. Type A dissection was deemed acute when symptoms developed within 2 weeks of presentation. All other patients, including those who were free of symptoms, were classified as having chronic type A dissection.

Patient characteristics. Mean age at the time of operation for type A dissection was 62 ± 11 years; 15 patients (27%) were 70 years of age or older. Forty-nine (88%) were male. Chronic hypertension was present in 85% of patients. Two patients with acute type A dissection had Marfan's syndrome and both of these patients had previous mitral valve surgery. The previous cardiac operations are listed in Table I. These were performed at outside hospitals in 64%. Five patients were noted to have a dilated ascending aorta at initial operation.

Follow-up. Systematic CVIR follow-up every 2 years was supplemented by telephone interview with the patient or referring cardiologist, or both. Complete follow-up information was available in 43 of 48 hospital survivors (89.6%). Patients were followed up a total of 393 patient-years, with a mean follow-up among survivors of 9 ± 4.6 years, ranging from 2.6 to 22 years. Thirty-three percent were followed up for 10 or more years and 12% for 15 or more years.

Data analysis

Presentation of ascending aortic dissection. Univariable comparisons were made between patients having acute versus chronic dissections by the χ^2 or Fisher's exact test for dichotomous variables and *t* tests (with adjustment for unequal variance) for continuous variables. Multivariable logistic analysis was used to identify the constellation of factors characterizing those with acute versus chronic dissection. *P* for retention of factors was .1. The risk factors examined are the patient variables listed in the appendix.

Hospital outcome. Univariable analyses were performed for hospital death similarly to that for presentation (described

Table I. Previous cardiac operations

Operation	No.	%
CABG	40	71
AVR	8	14
AVR + CABG	4	7
AVR + mitral valve repair	1	2
CABG + mitral valve repair	1	2
Mitral valve repair	1	2
MVR	1	2
Total	56	100

CABG, Coronary artery bypass grafting; AVR, aortic valve replacement; MVR, mitral valve replacement.

earlier). A multivariable logistic regression analysis was performed to identify incremental risk factors for hospital death. The variables examined were those listed in the appendix. The *P* value for retention of variables in the model was .1. A separate analysis was performed for acute dissections.

Freedom from events. Nonparametric, non-risk-adjusted estimates of freedom from events were obtained by the method of Kaplan and Meier.⁷ For each event, a parametric method was used to resolve the number of hazard phases, identify the shape of the hazard function, and estimate its parameters.⁸

Presentation. Confidence limits are those equivalent to ± 1 standard error. Unless otherwise stated, continuous variables are expressed as mean $\pm$ standard deviation.

Results

Presentation of type A dissection. Type A dissection was acute in 34 patients and chronic in 22. The mean interval from the initial cardiac operation to hospital admission with type A dissection was 48.6 ± 46.8 months (range 9 days–180 months). Fifty-one of 56 patients (91%) were admitted more than 1 month after the initial cardiac operation. Patients with acute type A dissection presented earlier than those with chronic type A dissection (*P* = .05, Fig 1).

Acute and chronic type A dissections produced different symptoms (Table II). Patients with acute type A dissection were more likely to have chest or back pain than those with chronic type A dissection. Aortic regurgitation occurred in approximately 50% of patients in both groups. Rupture was more common in those with acute type A dissection. In 7 patients aortic rupture was identified at surgery. In 2 patients with acute type A dissection, rupture caused hemodynamic instability; in 1 patient the aorta ruptured into the right side of the chest and in the other it ruptured into the pericardium, producing cardiac tamponade. Both of these patients died. All other patients were in hemodynamically stable condition at the time of arrival in the operating

Fig 1. Cumulative distribution of interval to presentation, stratified according to acuteness of presentation.

Table II. Comparison of patients with acute and chronic ascending aortic dissection

Variable	Acute dissection (n = 34)	Chronic dissection (n = 22)	P value
Patient characteristics			
Age (y)	60.2 ± 12.3	64.2 ± 7.4	.13
Male	85	91	.5
Hypertension	85	86	.9
Marfan syndrome	6	0	.4
Previous CABG	79	82	.8
Presentation			
Chest or back pain*	88	25	<.0001
Aortic insufficiency	50	59	.5
Malperfusion	12	4	.4
Hemodynamic instability	6	0	.4
Rupture	18	4	.009
Type I dissection	85	64	.4
Type II dissection	15	36	.4

Values are expressed as percent unless otherwise indicated. CABG, Coronary artery bypass grafting.

*Missing data in 2 patients.

room. Three patients had unsuspected rupture that was contained by adhesions, and none of them had hemodynamic compromise. The aorta ruptured into cardiac chambers in 2 patients, into the left atrium in 1, and into the right atrium in 1. The patient with rupture into the left atrium had congestive heart failure, whereas the patient with rupture into the right atrium was free of symptoms.

Management of type A dissection. The initial diagnosis of type A dissection was made by means of a variety of modalities, depending on availability at referring institutions and era of surgery. Initial diagnostic tests included computed tomographic scan (50%), transesophageal echocardiography (27%), aortography (21%), and magnetic resonance imaging (2%).

Once the diagnosis was suspected or confirmed, all patients were managed with intravenous β -blockers and vasodilators in an intensive care unit setting. Preoperative coronary angiography was performed in 44 of 45 patients with a history of coronary artery bypass grafting (CABG). No complications were attributable to coronary angiography. The single patient who did not have coronary angiography was the patient with aortic rupture into the right side of the chest; he became hemodynamically unstable shortly after transfer from an outside hospital and was taken directly to the operating room.

The site of ascending aortic dissection was identified with certainty in only 4 patients; in 2 of these, the dissection began at the aortic cannulation site, and in the other 2 it originated at a proximal saphenous vein graft anastomosis.

The operation was performed electively in all patients with chronic type A dissection. In contrast, 68% of patients with acute type A dissection had surgery within 24 hours of presentation for medical care (range 6 hours–8 days). Median time interval from hospital admission to operation in those with acute type A dissection was 12 hours.

At operation, femoral arterial cannulation was used in 79% of patients. The remaining patients had arterial return via the axillary artery. Hypothermic circulatory arrest was used in 91% of patients. Techniques for repair of the type A dissection included supracoronary tube graft (84%), Bentall procedure (14%), and local repair (2%). In patients with previous CABG, the Bentall procedure was used when there was important intrinsic aortic valve disease, extensive destruction of the aortic valve, or extensive destruction of the proximal aortic root. Three of 13 patients with previous aortic valve replacement had Bentall procedures to facilitate replacement of an aging bioprosthesis; in the other 10 patients with previous aortic valve replacement, the prosthetic aortic valve was preserved. Six patients required aortic arch repair.

All patients with previous CABG required operative management of coronary artery disease. Strategies differed depending on local anatomy and the status of bypass grafts and native coronary arteries. Techniques for managing previous CABG included reimplantation of proximal anastomoses with a button of native aorta (29 patients), interposition grafts to pre-existing saphenous vein grafts (9 patients), and new saphenous vein grafts (20 patients). In 13 patients, more than one technique was used.

Outcome. Eight hospital deaths occurred (14%; confidence limits 9%-20%), including 5 (15%) deaths in patients with acute type A dissection and 3 (14%) in patients with chronic type A dissection. Mode of death was bleeding in 4 patients and stroke in 4 patients. The presence of dissection-related complications, the acuity of dissection, the interval from presentation to operation, and the extent of dissection did not influence hospital mortality either overall or for acute dissection.

Important morbidity occurred in 14 patients (Table III). Only 2 patients had a perioperative myocardial infarction documented by new Q waves on the electrocardiogram. One of these patients had previous CABG, and the other had previous mitral valve repair. Both patients survived.

There were 10 late deaths, and the 10-year survival for the entire group of patients was 68% (Fig 2). There was no difference in survival between those with acute and chronic type A dissection. The most common cause of late death was congestive heart failure, occurring in 4 patients; 2 patients had sudden death. Reoperation for aortic disease was necessary in 6 patients, and 10-year freedom from reoperation on the aorta was 88% (Fig 3). Aortic reoperations included repair of thoracic aortic aneurysm in 3 patients and repair of abdominal aortic aneurysm in 3 patients. No patient died at reopera-

Table III. Postoperative hospital morbidity

Morbidity	No.	%	CL (%)
Respiratory insufficiency	6	11	6-17
Bleeding	3	5	2-10
Myocardial infarction	2	4	1-8
Mediastinitis	1	2	0.2-6
Leg wound infection	1	2	0.2-6
Pacemaker	1	2	0.2-6
Abdominal aortic rupture	1	2	0.2-6
Femoral artery embolism	1	2	0.2-6

CL, 70% confidence limits.

tion. At most recent follow-up, 91% of survivors are in New York Heart Association functional class I or II.

Discussion

Presentation. The presentation and natural history of acute type A dissection are well described in the literature. The onset of acute dissection is accompanied by chest pain in more than 95% of patients.¹ Dissection-related complications occur in the majority of patients. Without surgery, 50% of patients with acute type A dissection have intrapericardial rupture and die within 48 hours.^{1,6} This is the most common cause of death in patients with type A dissection.¹ In surgical series, up to 30% of patients have hemodynamic instability as a result of rupture before arrival in the operating room.⁹

Patients having acute type A dissection within 2 to 3 weeks of cardiac surgery also have a high risk of rupture and death.¹⁰⁻¹³ Nicholson and associates¹⁰ noted that cardiac tamponade and death occurred in 2 of 3 patients in whom type A dissection developed within 10 days of surgery. Similarly, Murphy and colleagues¹¹ observed cardiac tamponade and death in 3 of 9 patients in whom type A dissection developed within 3 weeks of cardiac surgery.

In this series, only 5 of 34 patients with acute type A dissection returned for treatment within 1 month of cardiac surgery, and the mean interval between primary operation and the development of acute type A dissection was 40 ± 44 months. Thus these patients differ from those in whom type A dissection develops as an intraoperative or early postoperative complication of cardiac surgery. Most patients had chest or back pain. As in primary acute type A dissection, 50% of patients had aortic insufficiency. Distal malperfusion occurred in 12%, which is somewhat lower than that reported in primary type A dissection. The most striking finding is that only 2 of 34 patients (6%) with acute type A dissection had hemodynamic compromise as a result of rupture. Although rupture occurred in 6 patients with acute type A dissection, in 4 the rupture was contained

Fig 2. Survival. **A**, Freedom from death for patients with acute and chronic type A dissection. **B**, Hazard function for death for patients with acute and chronic type A dissection.

by adhesions, preventing cardiac tamponade. In the other 28 patients with acute type A dissection, it is likely that thickening of the aortic wall and scar tissue from previous surgery prevented rupture. Thus, with fewer instances of malperfusion and infrequent cardiac tamponade, nearly all of these patients with acute type A dissection were in hemodynamically stable condition at arrival in the operating room.

Management. In recent years, great progress has been made in the application of noninvasive modalities

for the diagnosis of acute type A dissection.¹⁴⁻¹⁷ Several studies demonstrate that transesophageal echocardiography, computed tomography, and magnetic resonance imaging all have high sensitivity and specificity for the diagnosis of type A dissection.¹⁴⁻¹⁷ The role of coronary angiography in the preoperative management of acute type A dissection remains controversial. Most groups believe that coronary angiography is associated with an unnecessary and potentially harmful delay in surgical treatment.^{2,17} However,

Fig 3. Aortic reoperation. **A**, Freedom from aortic reoperation for patients with acute and chronic type A dissection. **B**, Hazard function for reoperation for patients with acute and chronic type A dissection.

recent data from Creswell and associates¹⁸ demonstrate the safety and benefit of coronary angiography in selected patients with acute type A dissection.

Preoperative coronary angiography is *required* in patients having late type A dissection after previous CABG. Accurate delineation of native coronary anatomy and the status of existing bypass grafts are essential in planning operative therapy. Because rupture and hemodynamic compromise are rare, the extra time

required for coronary angiography adds little risk. Coronary angiography was associated with no complications or morbidity. Once the diagnosis is secure and the coronary anatomy delineated, expeditious operation is advised. In general, operation should be undertaken within 24 hours of arrival in the hospital.

Operative management of type A dissection after previous cardiac surgery presents unique technical challenges. Aortic cannulation should be avoided. In this

series, most patients had femoral artery cannulation, and the remainder had cannulation of the axillary artery. In more than 90% of patients, deep hypothermic circulatory arrest was used to facilitate an open distal anastomosis, and we currently recommend this approach for all patients with type A dissection. Despite aortic insufficiency in 50% of patients, the aortic valve can be preserved in most. This agrees with the experience reported for primary type A dissection.¹⁹ In patients with previous aortic valve replacement, the prosthesis should be left in situ unless it is a bioprosthesis that has signs of degeneration or technical factors mandate aortic root replacement. Aortic arch repair is necessary in a minority of patients.

All patients with previous CABG require operative management of their coronary artery disease. Patent saphenous vein grafts may be managed by reimplanting the proximal anastomosis with a button of native aorta into the aortic graft. One or more saphenous vein grafts may be implanted as a single button. Alternatively, patent grafts may be attached to the aortic prosthesis with a saphenous vein interposition between the aortic prosthesis and the cut end of the original saphenous vein graft. This technique is particularly useful when there is extensive scar tissue, precluding safe mobilization of existing saphenous vein grafts. Saphenous vein graft disease or native coronary disease distal to existing grafts should be managed with new bypass grafts. Often, individual patients require application of more than one of these techniques. With the use of these techniques, only 1 patient with previous CABG had a perioperative myocardial infarction, and this was hemodynamically insignificant.

Outcome. Contemporary operative mortality for acute or chronic type A dissection is 10% to 15%.^{1,2} Numerous risk factors for operative mortality have been documented, including older age, malperfusion, cardiac tamponade, aortic arch repair, diagnostic delay, and concomitant CABG.^{2,4,17} In the current series, overall operative mortality was 14% despite the increased complexity of treating type A dissection in a reoperative setting. This acceptably low operative mortality is likely attributable to preoperative hemodynamic stability and appropriate intraoperative management of the ascending aorta and coronary artery disease.

Although a large proportion of deaths in surgical series of patients with primary acute type A dissection is attributable to cardiac failure,⁴ no patient in this series died of cardiac failure. The 8 operative deaths were evenly split between bleeding and stroke. In contrast to studies examining primary acute type A dissection, neither interval between presentation and opera-

tion nor concomitant CABG was a risk factor for operative mortality. Logistic regression analysis did not reveal any factors associated with operative mortality in patients with acute or chronic type A dissection.

Limitations. Patients included in this study are those who were admitted for surgical treatment of type A dissection after previous cardiac surgery. Those patients who died of type A dissection before being admitted for medical care were not identified and are not included in this series. Therefore the incidence of type A dissection after previous cardiac surgery could not be calculated. Because most patients had initial surgery at outside institutions, information concerning the size or quality of the aorta at primary surgery was largely unavailable. Therefore the data do not allow analysis of risk factors for late type A dissection after cardiac surgery. Finally, review of operative notes revealed the probable site of origin of the dissection in only 4 cases. It is therefore not possible to determine whether these dissections originated at previous surgical sites (aortic crossclamp, aortic cannulation, proximal anastomosis, aortotomy, vent site) or arose de novo.

Conclusions

Westaby² has written that type A dissection occurs in 2 groups of patients—those with aortic disease and those with chronic hypertension. Diagnostic work-up and management of these patients is standardized. We propose a third category of patients with type A dissection—those who have had previous cardiac surgery. These patients have distinctly different presentation and management from those in the first 2 categories. They are usually in hemodynamically stable condition, with cardiac tamponade and free rupture occurring rarely. Patients with previous CABG require coronary angiography and intraoperative management of coronary artery disease. Favorable preoperative hemodynamic status coupled with adherence to these management principles results in low operative mortality, similar to that seen in patients with primary type A dissection.

We thank Debbie Gladish, Luci Mitchin, and other members of the thoracic and cardiovascular research team for their efforts in assembling and verifying the clinical data.

REFERENCES

1. Crawford ES. The diagnosis and management of aortic dissection. *JAMA* 1990;264:2537-41.
2. Westaby S. Management of aortic dissection. *Curr Opin Cardiol* 1995;10:505-10.
3. Chirillo F, Marchiori C, Andriolo L, Razzolini R, Mazzucco A, Gallucci V, et al. Outcome of 290 patients with aortic dissection: a 12-year multicentre experience. *Eur Heart J* 1990;11:311-9.

4. Fann JJ, Smith JA, Miller DC, Mitchell RS, Moore KA, Grunkemeier G, et al. Surgical management of aortic dissection during a 30-year period. *Circulation* 1995;92(Suppl):III13-21.
5. Crawford ES, Svensson LG, Coselli JS, Safi HJ, Hess KR. Aortic dissection and dissecting aortic aneurysms. *Ann Surg* 1988; 208:254-73.
6. Isselbacher EM, Cigarroa JE, Eagle KA. Cardiac tamponade complicating proximal aortic dissection. Is pericardiocentesis harmful? *Circulation* 1994;90:2375-8.
7. Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. *J Am Stat Assoc* 1958;53:457-81.
8. Blackstone EH, Naftel DC, Turner ME Jr. The decomposition of time-varying hazard into phases, each incorporating a separate stream of concomitant information. *J Am Stat Assoc* 1986;81: 615-24.
9. Ehrlich M, Grabenwoger M, Simon P, Laufer G, Wolner E, Havel M. Surgical treatment of type A aortic dissections. *Tex Heart Inst J* 1995;22:250-4.
10. Nicholson WJ, Crawley IS, Logue RB, Dorney ER, Cobbs BW, Hatcher CR Jr. Aortic root dissection complicating coronary bypass surgery. *Am J Cardiol* 1978;41:103-7.
11. Murphy DA, Craver JM, Jones EL, Bone DK, Guyton RA, Hatcher CR Jr. Recognition and management of ascending aortic dissection complicating cardiac surgical operations. *J Thorac Cardiovasc Surg* 1983;85:247-56.
12. Subramaniam P, Skillington P. Acute aortic dissection as a complication of coronary artery surgery. *Aust N Z J Surg* 1996;66: 498-500.
13. Still RJ, Hilgenberg AD, Akins CW, Daggett WM, Buckley MJ. Intraoperative aortic dissection. *Ann Thorac Surg* 1992;53:374-80.
14. Svensson LG, Labib SB. Aortic dissection and aortic aneurysm surgery. *Curr Opin Cardiol* 1994;9:191-9.
15. Simon P, Owen AN, Havel M, Moidl R, Hiesmayr M, Wolner E, et al. Transesophageal echocardiography in the emergency surgical management of patients with aortic dissection. *J Thorac Cardiovasc Surg* 1992;103:1113-8.
16. Nienaber CA, von Kodolitsch Y, Nicolas V, Siglow V, Piepho A, Brockhoff C, et al. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med* 1993;328:1-9.
17. Rizzo RJ, Aranki SF, Aklog L, Couper GS, Adams DH, Collins JJ Jr, et al. Rapid noninvasive diagnosis and surgical repair of acute ascending aortic dissection. *J Thorac Cardiovasc Surg* 1994;108:567-75.
18. Creswell LL, Kouchoukos NT, Cox JL, Rosenbloom M. Coronary artery disease in patients with type A aortic dissection. *Ann Thorac Surg* 1995;59:585-90.
19. Fann JJ, Glower DD, Miller DC, Yun KL, Rankin JS, White WD, et al. Preservation of aortic valve in type A aortic dissection complicated by aortic regurgitation. *J Thorac Cardiovasc Surg* 1991; 102:62-75.

Appendix A: Variables examined in logistic regression analysis of hospital death

Patient variables

Demography: Age, sex

Previous operation: CABG, aortic valve replacement, mitral valve repair or replacement, date of operation

Character of dissection: Acute versus chronic, DeBakey I versus II, rupture, aortic regurgitation, interval from operation to dissection

Symptoms and signs: Chest pain at admission, back pain at admission, tamponade, arterial complications, coronary ischemia

Diagnostic modalities: Use of transesophageal echocardiography, computed tomographic scan, magnetic resonance imaging, catheterization

Aorta: Marfan's syndrome, bicuspid aortic valve

Procedure variables

Procedural: Interval from dissection to operation (acute dissection only), use of interposition graft, use of composite graft, coronary reimplantation, saphenous vein graft extension, arch repair

Support technique: Arterial cannulation site (axillary, femoral), venous cannulation (atrial, femoral), use of hypothermic circulatory arrest

Experience: Date of dissection repair

Discussion

Dr R. Scott Mitchell (*Stanford, Calif*). Dr Gillinov and his colleagues are to be congratulated on a thoughtful analysis of acute type A dissections occurring in patients with previous cardiac surgery. During a 22-year period, they treated 56 such patients, 34 of whom had acute dissection. Ascending aortic replacement was performed during a short interval of deep hypothermic circulatory arrest, with reimplantation of previous coronary grafts on an aortic button and preservation of the aortic valve, when possible.

Differences in presentation between these patients and the typical patients with an acute type A dissection were noted: Rupture, if it occurred, appeared to remain contained without the onset of hemodynamic instability. Second, because of the absence of hemodynamic instability, all patients save one underwent diagnostic angiography, which the authors state was mandatory for optimal management of coronary artery disease at the time of the operation. Although about two thirds of patients had operations within 24 hours of admission, time to operation ranged from 12 to 96 hours. Operative mortality was a respectable 14%.

Although I fundamentally agree with their thesis, I wonder whether it is really supported by their data. Patients were identified from the database of the cardiovascular surgery service as patients who had repair of aortic dissections after previous cardiac surgery. Is it not possible that patients could have been admitted to other services and died before cardiovascular surgery consultation? Second, and perhaps more important, a significant possibility for a selection bias exists, because these are all patients who survived to transfer, but excluded patients who may have died at an outlying hospital before transfer.

Dr Gillinov, inasmuch as instability and, in fact, death did occur in 2 patients with unrecognized ruptures, which you state was difficult to diagnose, are you advocating a semi-elective approach to these patients?

As regards technical aspects of these operations, were new saphenous vein grafts anastomosed directly to the Dacron graft?

Aortic regurgitation in these patients can cause left ventric-

ular distention during cooling. How did you prevent this complication in these redo procedures with adhesions?

Dr Gillinov. It is certainly one of the limitations of this study that we identified only those patients who had surgical consultation or a surgical procedure. We do not know the incidence of ascending aortic dissection after previous cardiac surgery, nor do we know the true overall mortality of this complication. It is standard practice at The Cleveland Clinic that any patient with an aortic dissection type A or B is admitted by both a cardiologist and a cardiac surgeon. We probably did not miss many who had the diagnosis established, but it is likely that there are patients who died in the hospital or outside before a diagnosis was established.

Dr Mitchell. Second, since The Cleveland Clinic, like Stanford, is a tertiary referral center, is it not possible that you have a selection bias and that many patients could have died before transfer? In fact, the ability to transfer a patient may actually identify a stable patient group.

Dr Gillinov. Yes, that speaks again to the same limitation of this study—that we included only those patients who had surgical treatment of the dissection. Sixty-four percent of the patients were referred from outside institutions and a similar proportion had their initial operation at outside institutions.

Dr Mitchell. Although we know the timing of operation from the time of admission, it would be helpful to know the time interval from the onset of symptoms until the time of arrival at The Cleveland Clinic. Do you have that information?

Dr Gillinov. The mean interval from initial cardiac surgery to presentation with type A dissection was 48.6 ± 46.8 months. This was approximately the same as the time to development of symptoms in those patients who were symptomatic.

Dr Mitchell. Since instability and death occurred in 2 patients with rupture and you describe rupture as being difficult to diagnose, are you really advocating this rather semi-elective approach to these patients?

Dr Gillinov. We prefer to classify the management of these patients as urgent rather than emergency. The median time from onset of symptoms to arrival in the operating room was 12 hours. We believe that when these patients arrive and are in stable condition, we do have sufficient time to perform coronary angiography safely and rapidly and then go from

the angiography suite to the operating room. The 2 patients who were in hemodynamically unstable condition arrived at our institution unstable, and these patients were taken directly to the operating room.

Dr Mitchell. As regards some surgical techniques, although you used aortic buttons for reanastomosing your old patent vein grafts, were new saphenous vein grafts attached directly to your aortic conduit? These anastomoses seem to have a pretty poor longevity record.

Dr Gillinov. In most cases, yes, they were.

Dr Mitchell. Aortic regurgitation in these patients can be difficult to manage with previously dissected and adhered ventricles. How did you avoid left ventricular distention during cooling for hypothermic arrest?

Dr Gillinov. With aortic regurgitation that is only 1+ or 2+ or sometimes 3+, a left ventricular vent can usually keep up. However, if the aortic regurgitation is severe and the ventricle distends, we will clamp the aorta and then continue to cool.

Dr Mitchell. These are very difficult and complex patients, and I think you are to be commended, not only for your excellent results, but especially for your contribution to our understanding of a different natural history.

Dr D. Craig Miller (Stanford, Calif). I know all these patients were not originally operated on at the clinic, but did you have a chance to look at the old operative notes? Were there any clues about something that could have been done better at the first operation that might have prevented this, namely, leaving behind a relatively dilated aorta?

Dr Gillinov. As you noted, most of that information was unavailable to me because two thirds of the patients had the initial operation elsewhere. Of the 36% who were treated at The Cleveland Clinic, a few had either a somewhat dilated or thin aorta in the first operation. None of our patients had an aorta approaching 5 cm in diameter though. It is hard to identify the origin of dissection in these patients—primary aortic disease, hypertension, or technical problems at the first operation.

Dr Neal W. Salomon (Baltimore, Md). Do you know what percentage of these patients were treated with hypothermic arrest?

Dr Gillinov. In our series, 91% were treated with hypothermic circulatory arrest, and in current practice we always use circulatory arrest and do an open distal anastomosis.