

Long-Term Survival After Open Repair of Chronic Distal Aortic Dissection

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Background. The optimal treatment of chronic distal aortic dissection remains controversial, with endovascular stent-graft techniques challenging traditional surgery.

Methods. From January 1994 to April 2007, 104 patients (82 male, median age 60.5 years) with chronic distal aortic dissection underwent surgical repair, 0 to 21 years after initial diagnosis of acute type A or B dissection (median 2.1 years). Twenty-three (22%) patients underwent urgent-emergent surgery. Mean aortic diameter was 6.9 ± 1.4 cm. Indications for surgery, other than aortic expansion, were pain in 6 (6%) patients, malperfusion in 6 (6%), and rupture in 11 (11%). Forty-nine (47%) had previous cardioaortic surgery (29% dissection-related), 21 (20%) had coronary artery disease, 12 (12%) had Marfan syndrome, and 4 (4%) were on chronic dialysis. Twenty-six (25%) had a thrombosed false lumen. Thirty (29%) patients required reimplantation of visceral arteries; 8.3 ± 2.7 segmental artery pairs were sacrificed.

Results. Hospital mortality was 9.6% (10 patients). Paraplegia occurred in 5 (4.8%). Twenty-seven patients

(26%) experienced adverse outcome (death within one year, paraplegia, stroke, or dialysis). Adverse outcome was associated with atheroma ($p = 0.04$, odds ratio = 4.3). Survival was 78% at 1, 68% at 5, and 59% at 10 years (average follow-up, 7.7 ± 4.1 years). Freedom from distal aortic reoperation was 99% at 1, 93% at 5, and 83% at 10 years. After one year, patients enjoyed longevity equivalent to a normal age-sex matched population (standardized mortality ratio = 1.38, $p = 0.23$). By multivariate analysis, atheroma ($p = 0.0005$, relative risk = 9.32) and age ($p = 0.0003$, relative risk = 1.15/year) were risk factors for long-term survival.

Conclusions. The efficacy of open repair for distal chronic dissection is highlighted by normal survival after the first year, and a low reoperation-reintervention rate.

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Medical management plays a major role in patients who have chronic distal dissection resulting either from conservatively treated type B dissection or after successful operations for type A dissection, but some of the affected aortas progress to aneurysmal dilatation [1–4]. Control of hypertension and beta-blocker therapy are considered cornerstones of treatment for patients surviving acute dissection [5], but nevertheless a high incidence of enlargement during the follow-up period has been reported, with a growth rate in the thoracic aorta as great as 4 mm/year [6]. Other sequelae, such as distal progression of the initial dissection with associated malperfusion and (or) the onset of intractable pain, can require urgent or emergent repair [7, 8]. Despite aggressive medical therapy and close surveillance with computed tomography, almost 20% of patients surviving the acute phase of type B dissection will develop fatal rupture [9]. Although many patients never require subse-

quent operation after successful repair of acute type A dissection [1], those with distal dissection into the thoracoabdominal aorta may require replacement of dilated portions of the distal aorta to avert rupture.

Surgical intervention has continuously evolved, and the results have improved since the publication of historic large series showing mortality rates up to 27% in elective procedures [10]. In some, but not all, reports on the surgical treatment of thoracoabdominal aneurysms, aortic dissection has been significantly associated with increased risk of the development of postoperative spinal cord injury [11–14]. The fear of neurologic complications as well as of severe morbidity and mortality after surgery has provided a major incentive for evaluation of other strategies as alternatives to the standard surgical approach.

The development of new stent grafts has expanded the spectrum of therapy for dissections. Some authors are now proposing thoracic endovascular stenting as a substitute for surgery in patients with distal chronic dissections who need intervention, asserting that minimizing access trauma, blood loss, and reducing intensive care unit and hospital stays will counterbalance the eventual need for late reintervention. Feasibility and safety have been reported by several centers [15–17], and initial

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enthusiasm has triggered a potentially unreflective preference for stent grafting. The recently published expert consensus on the treatment of descending thoracic aortic disease using endovascular stent-grafts [18] suggests that stent graft treatment of patients with chronic aortic dissection does not reduce the risk of aortic rupture or increase life expectancy, but enthusiasm for endovascular stenting remains unabated. The purpose of this study was to evaluate short-term and medium-term outcome, long-term survival, and the overall efficacy of surgical repair of chronic distal dissection in a contemporary series.

Material and Methods

Patient Demographics

A review of our institutional database disclosed 104 consecutive patients who underwent surgical repair of chronic distal dissection from January 1994 to April 2007. The Institutional Review Board approved this retrospective research and waived the need for individual patient consent.

Table 1 summarizes demographics and clinical characteristics of the entire cohort of patients. Mean age was 57.8 ± 13.4 years (median, 60.5; range, 26 to 83 years); 82 patients were male (79%); 86 were hypertensive (83%); and 39 were smokers (38%). Four patients (4%) with end-stage renal insufficiency were on hemodialysis and 12 patients had Marfan syndrome. Reliable information about the date of the acute dissection was gathered in 90 patients (87%). The average interval between the onset of symptoms and the operation was 3.6 ± 4.1 years (median, 2.1; range, 0 to 21 years); 29 patients (28%) required operation within one year of the initial diagnosis. In 26 patients (25%), preoperative evaluation showed a thrombosed false lumen. Nine patients had significant atheromatous deposition in the chronic distal dissected aorta.

The principal indication for surgery was the size of the descending or thoracoabdominal aorta in 76 patients (73%). Eleven patients (11%) required operation for rupture, 6 (6%) for visceral malperfusion, and 6 (6%) for intractable pain.

Forty-nine patients (47%) had undergone previous cardioaortic procedures (average 1.3 ± 0.5) prior to operation for chronic distal dissection. Most of the reoperated patients had undergone one previous operation, but 12 had had 2, and 1 had undergone 3 previous aortic procedures. Seven patients had undergone previous repair of type A dissection, and were left with a residual distal dissection; 11 had acute type B dissection repair followed by distal enlargement. There were 16 patients with chronic type B dissection in whom arch replacement with an elephant trunk was carried out prior to descending aorta replacement. Three of the previous operations were ascending aortic aneurysm repairs, 6 were Bentall procedures, 20 were complete arch repairs, and 8 were operations in the proximal descending thoracic aorta. Twelve previous procedures were abdominal aortic aneurysm repairs. The remaining patients had undergone

Table 1. Summary of Patient Demographics and Clinical Risk Profiles

Variables	No. (%) (n = 104)
Sex	
Male	82 (78.8%)
Female	22 (21.2%)
Age (years)	
Mean \pm SD	57.8 \pm 13.4
Median (range)	60.5 (26–83)
Indication for surgery	
Size	76 (73.1%)
Rupture	11 (10.6%)
Malperfusion	6 (5.8%)
Pain	6 (5.8%)
Other	4 (3.8%)
Marfan syndrome	12 (11.5%)
Thrombosed false lumen	26 (25%)
Atheroma	9 (8.7%)
Interval after initial diagnosis (years)	
Mean \pm SD	3.6 \pm 4.1
Median (range)	2.1 (0–21)
COPD	3 (2.9%)
Smoker	39 (37.5%)
CAD	21 (20.2%)
Requiring previous CABG	7
Requiring previous Stent	1
Hypertension	86 (82.7%)
IDDM	7 (6.7%)
Renal Insufficiency	
Chronic hemodialysis	4 (3.8%)
Previous neurologic dysfunction	6 (5.8%)
Preoperative paraplegia	1
Stroke	4

CABG = coronary artery bypass grafting; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; IDDM = insulin dependent diabetes mellitus.

aortic valve replacements (2), CABG (8), and various other procedures (4).

Fifty of the aneurysms were limited to the descending thoracic aorta. The extent of the remaining cases, based on the Crawford classification system, is as follows: 29 were type I; 9 were type II; 13 were type III; and 3 were type IV.

Surgical Management

Replacement of the aorta was limited to the descending thoracic aorta in 50 patients (48%), and extended to the thoracoabdominal aorta in 54 patients (52%). All patients underwent a left posterolateral thoracotomy, often with extension of the incision across the costal margin. The aneurysm was dissected free from mediastinal tissue, and, when necessary, the diaphragm was taken down circumferentially. The abdominal aorta was accessed through the retroperitoneum. The intercostal and lumbar arteries were dissected and temporarily occluded. If motor and somatosensory evoked potentials remained unchanged, the segmental vessels were sacrificed before

opening the aneurysm to avoid backbleeding and possible steal from the spinal cord circulation. In general, in view of their importance in supporting spinal cord perfusion, clamping of the subclavian artery was avoided, and the internal mammary artery and the superior epigastric axis were preserved. If repair of the distal arch was necessary (30 patients, 29%), an open proximal anastomosis was performed during a period of circulatory arrest. Concomitant coronary artery revascularization was performed in 9 patients: the left mammary artery was utilized in four cases, and a saphenous vein segment in five.

When the repair did not completely remove the entire length of dissected aorta, distal fenestration was carried out to maintain flow into both the true and the false lumens and ensure adequate perfusion of the distal branch arteries. Vascular grafts (median diameter, 24 mm; range, 14 to 32 mm) with up to 3 additional side arms were implanted in an end-to-end fashion. A total of 30 patients (29%) underwent reimplantation of at least one visceral vessel. In 13 patients, a long beveled anastomosis was performed, encompassing the orifices of the celiac trunk and superior mesenteric artery. In 17 patients an intervening graft segment was necessary to properly reattach the visceral arteries to the main graft. Intraoperative details for the entire series are summarized in Table 2.

Segmental Artery Sacrifice and Reimplantation

An average of 8.3 ± 2.7 segmental artery pairs (median 8; range, 3 to 15) were sacrificed overall. Reimplantation of the segmental arteries was performed in 10 patients, mainly in the early years of the present series. In two cases, the attempt to reattach an intercostal artery was prompted by intraoperative loss of somatosensory evoked potentials (SSEP).

Perfusion Strategies

A clamp-and-sew technique was used in only 2 patients (1.9%). Partial cardiopulmonary bypass was employed in 17 patients (16%), and partial left heart bypass was used in 30 (29%). Hypothermic circulatory arrest was carried out in 55 patients (53%). The decision to utilize hypothermic circulatory arrest was prompted by technical considerations, often involving the feasibility and safety of clamping the aorta proximal to the repair and the anticipated need for visceral artery reimplantation. The average cerebral ischemia time during hypothermic circulatory arrest was 35 ± 10 minutes at a mean esophageal temperature of $13.1 \pm 1.9^\circ\text{C}$. During this time, the arch was debrided if necessary, and the proximal anastomosis of a graft (with a side branch for subsequent perfusion) was performed. Perfusion of the arch vessels and coronary arteries was achieved during upper body perfusion, for an average of 32.3 ± 16 minutes, maintaining a radial artery pressure of 50 to 60 mm Hg.

Somatosensory Evoked Potential and Motor Evoked Potential Monitoring

Our surgical technique has previously been described in detail [19]. Briefly, SSEPs were elicited by stimulation of

Table 2. Summary of Intraoperative Details

Variables	No. (%) (n = 104)
Urgent/emergent operation	23 (22.1%)
Previous cardioaortic operation	49 (47.1%)
Number of previous operations	
Mean \pm SD	1.3 \pm 0.5
Range	0–3
Aortic size (mm)	
Mean \pm SD	69 \pm 14
Median (range)	65 (45–105)
Perfusion strategy	
None	2 (1.9%)
PCBP	17 (16.3%)
PLHB	30 (28.8%)
HCA	55 (52.9%)
HCA time (minutes)	35 \pm 10
Selective cerebral perfusion time	32.3 \pm 16 min
Mean esophageal temperature with HCA	13.1 \pm 1.9°C
Reimplanted visceral arteries (No. of patients)	30 (28.9%)
Beveled	13 (12.5%)
Via separate graft	17 (16.3%)
Reimplanted segmental arteries	10 (9.6%)
Concomitant CABG	9 (8.7%)
SSEP monitoring	96 (92.3%)
MEP monitoring	34 (32.7%)
CSF drainage	73 (70.2%)
Segmental arteries sacrificed (#)	
Mean \pm SD	8.3 \pm 2.7
Median (range)	8 (3–15)

CABG = coronary artery bypass grafting; CSF = cerebrospinal fluid; HCA = hypothermic circulatory arrest; MEP = motor evoked potential; PCBP = partial cardiopulmonary bypass; PLHB = partial distal aortic perfusion with left heart bypass; SSEP = somatosensory evoked potential.

the posterior tibial nerves and recordings were made from the scalp overlying the somatosensory cortex. For motor evoked potential (MEP) monitoring, the stimuli applied to the motor cortices were recorded from the skin over the tibialis anterior and abductor pollicis muscles. A decrease greater than 50% in amplitude with concomitant increased delay was considered to reflect a spinal cord ischemic event. The SSEP monitoring was performed in 96 patients (92%), and MEPs, beginning in 2002, were utilized in 34 patients.

Cerebrospinal Fluid Drainage

A catheter for cerebrospinal fluid (CSF) drainage was placed in 73 patients (70%). The CSF pressure was monitored during the operation and postoperatively: the CSF was drained at a maximum rate of 15 cc/hour as long as the pressure remained above 10 mm Hg.

Postoperative Management

The SSEPs were monitored until the patient awakened. Thereafter, hourly brief neurologic examinations were performed for 72 hours. The target for postoperative

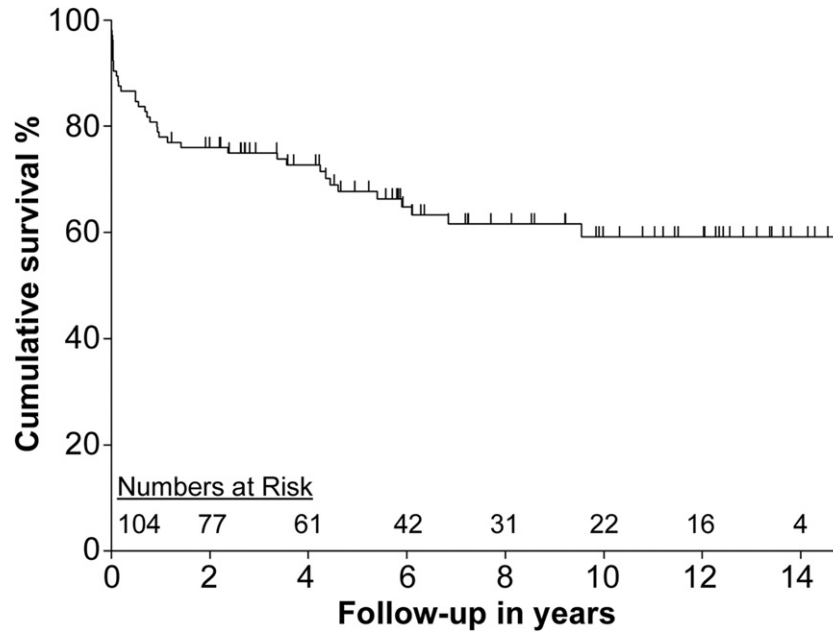


Fig 1. Kaplan-Meier survival curve for all patients (n = 104) after surgical repair of chronic distal dissection. Vertical marks represent censored patients.

mean aortic pressure was generally 90 mm Hg, although in a few patients with severe preoperative hypertension we aimed for even higher pressures. Constant monitoring of central venous pressure was also undertaken. The CSF drainage was continued for 48 to 72 hours, and methylprednisolone was used for 72 hours.

Follow-Up

Patients were followed by their referring cardiologist and contacted periodically by our research personnel. Annual computed tomographic scans were scheduled in all patients. Postoperative events were compiled and analyzed according to the Guidelines for Reporting Morbidity and Mortality after Cardiac Valvular Operations and our institutional checklist. For this study, the follow-up was closed on April 2, 2009 and was 100% complete. Follow-up ranged from 0 to 15 years (median 5 years), with an average of 5.7 ± 4.5 years. The average follow-up among survivors was 7.7 ± 4.1 years. Long-term survival was evaluated for the 81 patients still alive one year postoperatively (median follow-up 5 years; range, 0.1 to 14).

Statistical Methods

Data were entered in Excel (Microsoft, Redmond, WA) spreadsheets and transferred to a SAS file (SAS Institute Inc, Cary, NC) for data description and analysis. Patient and disease characteristics are described as percents, median (range), or means (standard deviation). The two major outcomes considered were adverse outcome (one-year mortality, stroke, paraplegia, or new permanent dialysis) among all patients in the sample, and long-term survival among patients who were alive one year after surgery. For the latter, follow-up time started one year after the procedure and terminated at death or last contact alive.

Table 3. Summary of Postoperative Details and Complications of the Entire Cohort (n = 104)

Variables	n (%)
Hospital mortality	10 (9.6%)
Paraplegia	5 (4.8%)
Stroke	6 (5.8%)
Bleeding requiring reoperation	4 (3.8%)
Renal insufficiency	5 (4.8%)
Creatinine ≥ 1.6 mg/dL	2 (1.9%)
Temporary dialysis ^a	2 (1.9%)
Permanent dialysis	1 (0.9%)
Respiratory complications	16 (15.4%)
Tracheotomy	15 (14.4%)
ECMO	1 (0.9%)
Cardiac complications	3 (2.9%)
Cardiac arrest	1 (0.9%)
Permanent pacemaker insertion	2 (1.9%)
ICU stay (days)	
Mean \pm SD	6.2 \pm 5.5
Median (range)	4.5 (0–33)
Hospital stay (days)	
Mean \pm SD	18.3 \pm 14.5
Median (range)	13 (0–75)
Adverse outcome ^b	27 (26%)

^a Patients did not require further dialysis after discharge. ^b Adverse outcome = one-year mortality, spinal cord injury, stroke or permanent dialysis.

ECMO = extracorporeal membrane oxygenation; ICU = intensive care unit.

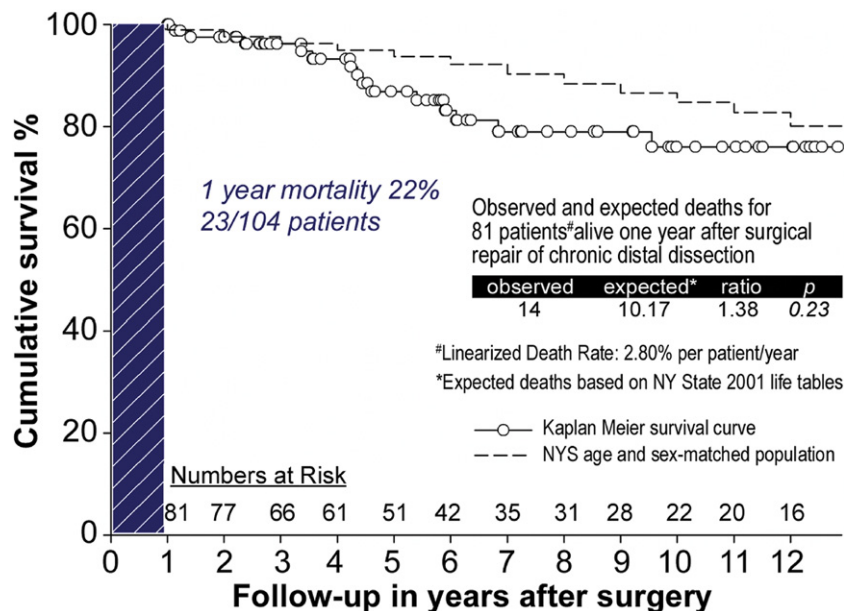


Fig 2. Longevity of one-year survivors.

Factors were tested for association with an adverse outcome by χ^2 tests or logistic regression, as appropriate. Long-term survival probabilities were estimated from a Kaplan-Meier life table. We also estimated the annual death rate for this group per person-year of follow-up, as well as the standardized mortality ratio. This gives the observed numbers of deaths relative to the number that would be expected based on New York State population death rates for comparable ages, genders, and follow-up times. Statistical significance of the standardized mortality ratio was tested under a Poisson model, implemented with SAS Proc Genmod (SAS Institute Inc) [20].

Results

Hospital mortality, defined as death in the hospital during recovery from surgery or within 30 days after operation, was 9.6% (10 patients). Overall survival was 78% at one year, 68% at five years, and 59% at ten years after surgery (Fig 1).

Postoperative Complications

Although an average of 8.3 ± 2.7 segmental artery (SA) pairs were sacrificed, and 38 patients (37%) required resection of an extensive (>10 SA) aneurysm, only 5 patients (4.8%) developed spinal cord injury (Table 3). All cases of paraplegia occurred between 1996 and 2000, when MEP monitoring was not yet routinely performed at our institution. An ischemic insult was detected by means of intraoperative SSEP monitoring in two cases, but hemodynamic and anesthetic manipulation, as well as prompt SA reattachment, did not restore adequate spinal cord blood flow. Three patients developed late-onset paraplegia despite intact intraoperative and postoperative SSEPs; in one case after a prolonged period of low mean arterial pressure after cardiac arrest. The

average number of SA pairs sacrificed in the five patients with spinal cord injury was 10.2 ± 2.7 , with a median of 10 (range, 7 to 14).

Six patients (5.6%) developed strokes, confirmed by either computed tomography or magnetic resonance imaging studies. Only one patient (0.9%) required postoperative permanent dialysis, while in two patients in-hospital dialysis was necessary to treat transient acute renal insufficiency.

There were no cardiac ischemic events, but one patient had cardiac arrest and two required insertion of a permanent pacemaker for bradycardia-tachycardia syndrome. Severe respiratory complications occurred in 16 patients (15.4%): 5 required tracheotomy and in one patient extracorporeal membrane oxygenation support was utilized; 9 patients (9%) had prolonged initial intubation or required reintubation. Median intensive care unit and hospital stay were 4.5 and 13 days, respectively (ranges, 0 to 33 and 0 to 75).

Adverse Outcome

To better evaluate the true overall impact of the surgical approach, a composite of one-year mortality (including hospital deaths) and three major complications (stroke, permanent dialysis, and paraplegia) were defined as adverse outcome (AO). An adverse outcome occurred in 27 patients (26%).

Multivariate analysis identified the presence of atheroma in the resected aorta as the only independent risk factor for AO ($p = 0.04$, odds ratio = 4.3). Adverse outcome occurred in 5 of 9 patients (56%) in whom severe atheroma was identified during postoperative pathologic evaluation. Only 22 patients (23%) without atheroma experienced AO. By logistic regression, the interval between acute dissection and date of operation showed a significant association ($p = 0.02$) with AO, with the odds

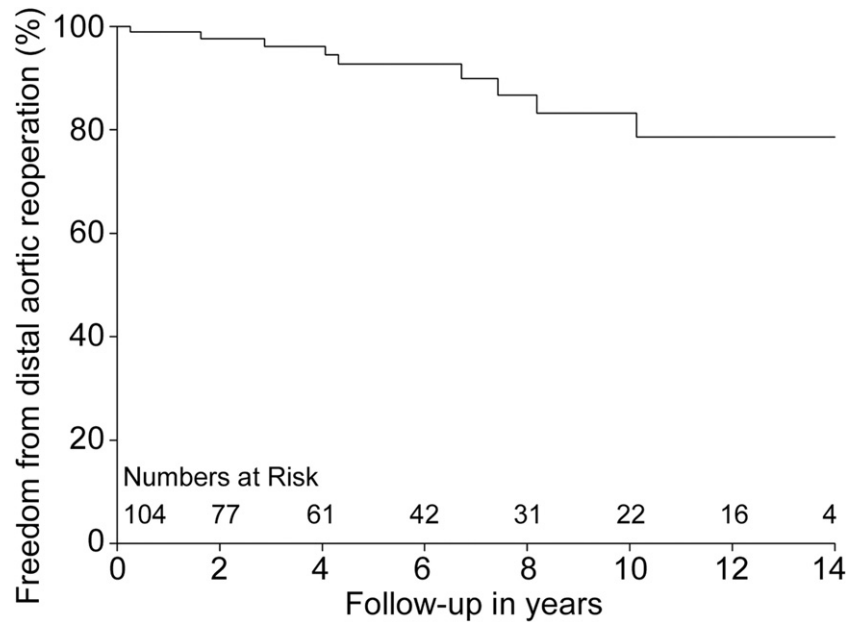


Fig 3. Freedom from distal aortic reoperation for the entire cohort of 104 patients.

increasing by a factor of 1.15 (95% CI 1.02 to 1.20) for each year of increase in interval time.

Long-Term Survival

Long-term survival was evaluated for the 81 patients still alive one year after surgery. Fourteen of them died, yielding a linearized death rate of 2.8% per patient-year. The low annual death rates of one-year survivors resulted in estimated long-term survival probabilities of 98% at one year, 83% at five years, and 76% after ten years (Fig 2). The New York State life tables for the year 2001 (median year of the surgical procedure among the 81 patients) were used to evaluate death rates for a comparable age and sex-matched population: 10.17 deaths were expected, giving a linearized death rate of 2.03% per patient-year. The standardized mortality ratio showed no significant difference between one-year survivors and a New York State comparable population (standardized mortality ratio = 1.38, $p = 0.23$). By multivariate analysis, atheroma ($p = 0.0005$, relative risk = 9.32) and age ($p = 0.0003$, relative risk = 1.15/year) were independent risk factors for long-term survival.

Freedom From Cardioaortic Reoperation

During the entire follow-up, 16 cardioaortic reoperations were performed on 15 patients after an average of 4.9 ± 3.2 years (median 4.7 years; range, 0.2 to 10 years). In 7 cases the reoperations were not dissection-related; 2 Bentall operations, 2 aortic valve replacements, 2 coronary artery bypass grafts, and one ascending aorta repair. Nine patients required surgery on the distal aorta for further dilation after initial surgical repair: 2 patients had their residual descending thoracic aorta replaced, 2 required abdominal aortic aneurysm repair, and in 5 patients a thoracoabdominal aneurysm repair was carried

out. The average interval between the first surgical repair and the subsequent distal aortic reoperation was 5.7 ± 2.9 years. No endovascular reintervention was required in any patient. Freedom from reoperation on the distal aorta was 99% at 1 year, 93% at 5 years, and 83% at 10 years (Fig 3). All patients survived the reoperation on the distal aorta, but one patient who underwent thoracoabdominal repair with visceral artery reimplantation developed acute renal failure and required permanent dialysis (adverse outcome rate, 11%).

Comment

Surgical repair of chronic distal dissection is usually followed by a non-negligible rate of short-term morbidity and mortality. Because an etiology of chronic dissection is widely accepted as significantly increasing the surgical challenge and the overall risk of intraoperative and postoperative complications after distal aortic resection, it is surprising that there is a lack of large studies focusing on this disease. A report by Estrera and colleagues [21] of 182 patients who underwent repair of thoracic aortic aneurysms showed that almost 50% had chronic dissection; their hospital mortality was 8.8%.

One of the major problems in evaluating any interventional therapy for chronic distal dissection has to do with the difficulty in being sure that one has included comparable patient populations when making comparisons. Because most acute type B dissections are currently treated medically and do quite well, surgical series tend to deal with those patients who have encountered threatening or symptomatic complications, which prompt consideration of an operation thought to carry a high risk. In this series we reported on all patients with chronic distal dissections, almost half of whom had undergone previ-

ous surgery. This included a small number with extensions of surgically treated type A dissections (7%); 16% with concomitant arch aneurysms who survived arch resection with an elephant trunk procedure and 11% with previous operation for type B dissection. The remaining patients were operated on for complications or progressive aortic dilation of medically treated acute type B dissections.

Critics of the selective surgical approach to chronic distal dissection claim that its mortality rates are unacceptable, and strongly demand safer strategies. The threshold for considering endovascular intervention is generally lower than for surgical repair, with some centers even advocating routine stenting of uncomplicated type B dissections. Endovascular series therefore include varying proportions of patients who would not be considered appropriate candidates for open surgery. Consequently, it seems logical that one should anticipate better results after endovascular interventions than after open surgery.

The rationale behind an aggressive endovascular approach is that sealing the proximal entry point between true and false lumina will decrease blood flow into the false lumen and thus promote thrombosis and stabilization of the downstream aorta. Unfortunately, endovascular stent grafting for type B dissection does not appear to deliver the expected results. In 2009, Guangqi and colleagues [22] published their experience with 121 consecutive patients undergoing endovascular repair for acute and chronic type B dissection; there were postoperative endoleaks in 22%, with a 30-day mortality of 8.2% in that group.

Our experience revealed a one-year attrition that can hardly be considered ideal. There was a hospital mortality of 9.6% and a 26% one-year incidence of an adverse outcome. In considering these figures, however, the high operative risk expected in such patients should be acknowledged: there were high proportions of urgent and complicated procedures (22% and 27%, respectively), and more than 50% of patients presented with thoracoabdominal aneurysms.

Paraplegia rate in our surgical cohort was below 5%, despite a high number of patients (37%) who underwent extensive aortic resection with sacrifice of more than ten segmental arteries. It is encouraging that no patients developed paraplegia after MEP monitoring was instituted. In two cases, despite intraoperative detection of spinal cord ischemia by SSEP and a prompt response including SA reattachment, spinal cord injury could not be avoided. We speculate that several of our strategies—craniocaudal stepwise SA sacrifice, careful hemodynamic manipulation, and routine preservation of the left subclavian artery (covered in up to 40% of endovascular procedures)—minimized the intraoperative and postoperative ischemic insult posed by sacrifice of segmental arteries and (or) helped the collateral network which supplies the spinal cord to respond.

We believe that crude hospital mortality does not accurately reflect the true cost to the patient of surgical repair. For this reason, we used adverse outcome, a composite of death, stroke, paraplegia, and new perma-

nent dialysis for the first postoperative year, to evaluate this cohort of patients. We were surprised that anticipated risk factors such as false lumen patency, urgent-emergent procedure, and indication for surgery were not associated with AO in our 104 patients by multivariate analysis. The only independent risk factor for AO was the presence of severe atheromatous disease of the dissected aorta, with an odds ratio of 4.3.

Our findings regarding the interval between the onset of symptoms and operation for chronic distal dissection may shed some light on the optimal timing of intervention in type B dissection as well as the possible importance of stenting the distal aorta when surgically treating acute type A dissection [23], a topic which is currently a matter of great interest. In both instances, timing is likely crucial for endovascular intervention, as remodeling of the aorta after exclusion of the entry tear is likely to occur only early in the dissection process, and stenting once the false and true lumens have become stabilized may even be counterproductive [24]. We found a statistically significant increase (odds ratio 1.15 per year, $p = 0.02$) in risk for AO with lengthening interval between acute dissection and surgery. The most conservative interpretation supports avoiding long delays before opting for surgery for complications after onset of distal dissection. But the relative safety of both surgery and stenting in the subacute phase of type B dissection, the theoretical appeal of early stenting even in uncomplicated acute type B dissection, and the improving results of surgery should perhaps prompt reconsideration of optimal management for acute type B dissection.

The raw overall survival of our patients (78% at one year, 68% at five years, and 59% at ten years after surgery) compares favorably to large contemporary surgical series focusing on late survival [25]. Recently published papers on short and midterm outcomes of stent grafting report results ranging from acceptable to disappointing: 82% and 65% survival at 3 years, respectively, for the Talent Thoracic Retrospective Registry [26] and Guangqi and colleagues [22], and 66% at five years for Böckler and colleagues [27]. Looking at these reports suggests that the widely touted safety of the endovascular approach is based more on wishful thinking than on real data. Even when midterm results of stents are favorable [15], concern about late success is always present. Freedom from reoperation-reintervention of 72% at three years, with a late failure of 33% for reintervention, can hardly be considered an optimal answer to the problem of aortic dissection.

Although endovascular stent graft treatment may slow the progression of aortic disease, we believe that only by means of surgical resection of the weakened segments of the aorta can the natural course of dissection be reversed. Some patients will require reoperation, but our experience of freedom from distal aortic reoperation in 93% at 5 years and 83% at ten years, and of restoration to normal of long-term survival in patients still alive one year after surgery, reaffirms the idea that resection of the diseased distal aorta provides durable relief from the sequelae of chronic distal dissection.

In conclusion, surgical management of chronic distal dissection in experienced hands offers acceptable rates of short-term morbidity and mortality, and promises excellent longevity and freedom from reoperation and reintervention in survivors. We anticipate that this study may prove useful in providing a benchmark for evaluating alternative strategies for the treatment of both chronic type B dissection and distal sequelae of surgically treated type A dissection.

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DISCUSSION

DR HAZIM J. SAFI (Houston, TX): I want to thank the Society for asking me to discuss this very important and timely paper, and I also want to thank Stefano Zoli for sending me the paper in a timely fashion. It addresses an important issue regarding long-term results following acute or chronic dissections. The mortality rate is excellent, the freedom from reoperation is superb. The unrestrained use of TEVAR [thoracic endovascular aortic repair] in the descending thoracic aorta (which seems to be ahead of the popular curve) is not indicated based on current evidence. For a chronic dissection, or for acute uncomplicated type B aortic dissection the mortality rate is less than 2% with

open repair, so there is no rationale for use of TEVAR. So this addresses this very important issue.

I have three questions. First, you talked about mortality which was correlated with atheromatous plaque and age. Did you evaluate the mortality rate in relation to renal dysfunction, in other words, glomerular filtration rate? In our experience, GFR was better than any other marker. Secondly, did you look at the cost of the surgical graft compared to TEVAR? Finally, did the incidence of stroke correlate with the atheromatous plaque in the proximal descending thoracic aorta or the liberal use of profound hypothermia?

Again, I want to thank the Society for asking me to discuss this paper.

DR ZOLI: Thank you very much, Dr Safi, for your kind comments and your very interesting questions. As for your first question, we did include renal failure and renal insufficiency as possible risk factors in our analysis both of adverse outcome, which includes one year mortality, and of long-term survival. We used the level of serum creatinine and the need for dialysis as indicators of renal insufficiency, and neither was an independent risk factor for poor immediate or long-term outcome. Perhaps a larger series would have identified renal dysfunction as a risk factor.

The short answer to your second question is that we did not make a cost comparison between open and endovascular repair.

As for your third question, the presence of severe atherosclerotic disease was the main risk factor for adverse outcome, which includes stroke, in our cohort of patients. Hypothermic circulatory arrest was not a risk factor for adverse outcome in this group of patients.

DR MARC R. MOON (St. Louis, MO): I think it is important to note, like Dr Safi said, with good medical therapy a 2% mortality rate can be expected with an acute type B dissection. So, patients with chronic type B dissections are going to become more and more frequent. One issue that we cannot forget, though, is aggressive medical therapy to prevent aneurysmal dilation long term. We found that the need for reoperation after a residual descending dissection following successful type A repair is directly related to blood pressure control and beta blocker use postoperatively. Is this a policy at your institution, are these patients referred in, and how do you follow them afterwards and treat them after you have done their primary repair?

DR ZOLI: Anti-impulse therapy with beta blockers is a cornerstone of postoperative care at our institution, and we are extremely careful to keep the mean arterial pressure as low as possible. As for the follow-up after the first operation, we try to include every single patient in a surveillance program which includes an annual CT [computed tomographic] scan.

DR LARS G. SVENSSON (Cleveland, OH): This is an interesting study because we are seeing more and more that you have to be very careful about your choice of patients for endovascular versus open. We have done a similar study to what you did. We looked at our chronic dissections, either type As with elephant trunks or type Bs with elephant trunks, and we found that in our patients who had open procedures, mostly descendings with dissections, the mortality rate was about 4.4% and with endovascular it ran about 6.8%, with no significant differences in other outcomes. The only advantage for endovascular was blood loss and hospital stay. When we followed the patients, and propensity risk adjusted survival it was the same, but the reintervention rate was much higher with the endovascular group. So once again just to emphasize, as you pointed out, we have to become more careful in who we choose for the endovascular stenting procedures. We need to be more careful about matching the procedure to the patient's needs and not the patient to one procedure. Obviously with degenerative aneurysms we tend to stent most of them, but for chronic dissections I think this is an area where we have a fair amount of equipoise about studying it further.

DR ZOLI: Thank you very much, Professor Svensson, for your comments on this topic. I absolutely agree with you.

DR THORALF SUNDT (Rochester, MN): I have a technical question. For the patients in whom you used profound hypothermia and circulatory arrest, how did you cannulate for arterial inflow?

DR ZOLI: We usually use the femoral artery for arterial inflow.

DR SUNDT: There is a problem with that and atheroma and it might be the cause of your high stroke rate. You might consider some other options, be it a chimney graft on the axillary artery, the ascending aorta, or lately we have been just cannulating the apex and pass one of these nice new femoral percutaneous cannulae right across the aortic valve. That works pretty well. That will give you antegrade perfusion and you won't blow all of that debris up north.

DR ZOLI: Dr Griep prefers to cannulate the femoral artery, but certainly those are absolutely good alternative strategies.

DR ANTHONY L. ESTRERA (Houston, TX): Nice presentation. My question is going to focus really on the thrombosed false lumen. Can you elaborate a little more? I noticed that 25% or a quarter of your cases were a partially thrombosed false lumen. My understanding in aortic remodeling is that you stent it so you can thrombose the false channel, yet 25% of the patients had a thrombosed false lumen but still developed an aneurysm. It just underscores the importance of the work being performed by people in this room, John (Ikonomidis), looking for biomedical markers for disease expansion and disease progression. Was the remaining 75% partially thrombosed? Please elaborate.

DR ZOLI: The question of thrombosis and its relationship to progression of the dissection is a very interesting one. In 25% of our cases, the false lumen was completely thrombosed. Despite that fact, these patients still developed an aneurysm. Complete thrombosis of the false lumen may slow down the growth rate, but it doesn't necessarily stop it. In this analysis, we were somewhat surprised that the patency of the false lumen was not a risk factor. In a previous analysis, a patent false lumen was considered a risk factor at least for more rapid growth. But this is what the current data show. If we think about TEVAR procedures, the idea of covering the proximal intimal tear and trying to provide some sort of scaffolding for the distal aorta makes sense, but it appears that despite that, some patients may still develop a distal aneurysm.

DR MOON: I think one last thing that is important to focus on is your first conclusion; hospital mortality and paraplegia rates were acceptable in "expert hands."

DR ZOLI: Yes, that is correct.

DR MOON: I think we need to remind ourselves that these can be very challenging operations and are not very common, such that surgeons without an aortic focused practice probably should not "dabble" in this procedure.

DR ZOLI: It is definitely a challenging procedure, but we have to keep in mind that this series included patients with thoracoabdominal aneurysms; almost 30% of patients required visceral artery reimplantation. Not every procedure for a chronic type B dissection is as challenging as the ones that I presented today.