SURGERY FOR ADULT CARDIOVASCULAR DISEASE

PERCUTANEOUS BALLOON FENESTRATION AND STENTING FOR LIFE-THREATENING ISCHEMIC COMPLICATIONS IN PATIENTS WITH ACUTE AORTIC DISSECTION

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Objectives: Acute aortic dissection frequently causes life-threatening ischemia of end-organs, historically associated with mortality exceeding 60%. Reperfusion with the use of interventional radiologic methods has evolved as a promising treatment. We report results of our initial 6 years of experience with percutaneous balloon fenestration of the intimal flap and endovascular stenting. **Methods:** Forty patients (32 male and 8 female) with a median age of 53 years (range 16-86 years) underwent percutaneous treatment for peripheral ischemic complications of 10 type A and 30 type B acute aortic dissections since 1991. Twenty patients had ischemia of multiple organ systems. Thirty patients had renal, 22 had leg, 18 had mesenteric, and 1 had arm ischemia. **Results:** Fourteen patients were treated with stenting of either the true or false lumen combined with balloon fenestration of the intimal flap, 24 with stenting alone, and 2 with fenestration alone. Successful revascularization was achieved in $93\% \pm 4\%$ ($\pm 70\%$ confidence levels) of patients (37/40). Nine patients had procedure-related complications. The 30-day mortality rate was $25\% \pm 7\%$ (10/40), often related to irreversible ischemia of intra-abdominal organs that was present before the procedure. Of the remaining 30 patients, 5 have died and the remaining 25 continue to have relief of ischemic symptoms at a mean follow-up of 29 months. **Conclusion:** Percutaneous balloon fenestration of the intimal flap and endovascular stenting is an effective treatment for life-threatening ischemic complications of acute aortic dissection. (J Thorac Cardiovasc Surg 1999;117:1118-27)

Acute aortic dissection frequently causes life-threatening ischemia of distal end-organs. Historically, the mortality rate in patients with ischemic complica-

tions of an aortic dissection can exceed 60%, depending on the type and acuity of the dissection, as well as the specific region that is ischemic.^{1,2} Reperfusion by means of interventional radiologic methods has evolved into a promising approach,³⁻⁶ but no large series have been published. We therefore report the results of our initial 6 years' experience with percutaneous balloon fenestration of the intimal flap and endovascular stenting.

Patients and methods

Definitions and statistical analysis. Between September 1992 and February 1998, 40 patients underwent percutaneous treatment for peripheral ischemic complications of an acute aortic dissection (complications appearing within 14 days of the onset of symptoms). Patients underwent percutaneous treatment for extremity, renal, or mesenteric ischemia associ-

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

Received for publication July 15, 1998; revisions requested Sept 8, 1998; revisions received Nov 30, 1998; accepted for publication Jan 28, 1999.

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12/6/97599

Fig 1. Treatments of patients with acute aortic dissection from September 1992 through February 1998.

ated with an aortic dissection. Ischemia was determined by clinical suspicion based on signs, symptoms, and laboratory data in conjunction with imaging of the arterial supply to the affected region. If any anatomic arterial abnormality related to the aortic dissection involved the arterial supply to the region suspected of being ischemic, the anatomic abnormality was considered to be responsible for the ischemia, and the lesion was treated. Anatomic abnormalities discovered in the arterial supply to an asymptomatic region were not treated. For data analysis, involvement of either or both kidneys was counted as one vascular bed, as was involvement of either or both lower extremities.

Relevant data were collected from chart review for patients treated before 1995. Beginning in 1995, data were collected prospectively. Follow-up data were collected by telephone interview with the patients or their families and were available in all patients. Cause of death was determined by review of medical records or autopsy reports, if available.

Variability of clinically important fractions or rates is expressed as $\pm 70\%$ confidence levels (70% CL). Statistical analysis was performed with the use of SPSS for Windows (version 6.1, SPSS Inc, Chicago, Ill). Fifteen specific variables were evaluated as possible predictors of early mortality and overall mortality. Ordinal variables included type of dissection (A versus B), renal ischemia, mesenteric ischemia, extremity ischemia, ischemia of 1, 2, or 3 vascular beds, true

lumen collapse, sex, paraplegia/paraparesis, and stroke. Continuous variables included date of procedure, age, number of stented vessels, and number of fenestrations. Early mortality was defined as death within 30 days of the percutaneous procedure. Overall mortality was defined as all deaths due to any cause. Multivariable regression was performed with the use of the Cox proportional hazards model to analyze variables using a forward stepwise method. Variables with a P value of .1 or less (to avoid a type II or β error given the relatively small numbers of patients) in the univariable screening were entered into the multivariable equation. The magnitude of the clinical impact of predictive covariables on early and overall mortality is reported as an indicator of relative risk, e^{β} , but this can be strictly interpreted only for dichotomous covariables.

Patients. The 40 patients (32 male and 8 female patients) ranged in age from 16 to 86 years, with a mean of 53 years. Ten had type A and 30 had type B dissections. Four of the patients with an acute type B dissection had undergone surgical repair of a type A dissection 20 days to 8 years (median 2 years) before the percutaneous intervention; this represented a new dissection superimposed on the chronic type A dissection. Two of the patients had Marfan syndrome, and 1 had Ehlers-Danlos syndrome. Case histories of 18 of these patients have been previously reported.^{4,7} Two patients had initial resolution or improvement of ischemic symptoms fol-

Table I. *Distribution of ischemic complications in 40 patients with acute aortic dissection*

<i>Ischemic region</i>	<i>No.</i>
Renal/mesenteric/lower extremity	11
Renal/lower extremity	6
Renal/mesenteric	3
Renal	10
Lower extremity	5
Mesenteric	4
Upper extremity	1
Total	40

lowed by symptom recurrence, and 2 patients had persistent symptoms after the initial procedure. Each of these 4 patients underwent a second percutaneous intervention 2, 7, 7, and 39 days (mean 14 days) after the initial one, for a total of 44 procedures.

Nine of the 10 patients with a type A dissection had undergone surgical graft replacement of the ascending aorta up to 14 days (median 1 day) before the percutaneous procedure. The sole patient who did not have an operation had severe heparin-induced thrombocytopenia with positive antibodies. One of the 30 patients with a type B dissection underwent graft replacement of the descending thoracic aorta immediately before the percutaneous procedure. During this same period, 140 acute type A dissections and 24 acute type B dissections were surgically repaired. The proportion of patients with each type of dissection treated with surgery, percutaneous techniques, or both is shown in Fig 1.

Twenty patients had peripheral ischemic complications of multiple organ systems. Thirty patients had renal, 22 had lower extremity, 18 had mesenteric, and 1 had upper extremity ischemia. The distribution of ischemic symptoms is shown in Table I. No patient was referred specifically for treatment of stroke or spinal cord ischemia, although 4 patients who had a stroke and 3 patients who had paraparesis or paraplegia were treated for other ischemic complications.

Procedures. Only selected patients in whom there was clinical suspicion of extremity, renal, or visceral ischemia underwent arteriography; the diagnosis had been established earlier, usually by means of transesophageal echocardiography or computed tomography. When renal failure was present, portions of the diagnostic arteriogram were performed by the hand injection of carbon dioxide as the contrast agent during digital subtraction arteriography. Intravascular ultrasound (IVUS) was used to define anatomic details of the dissection in many cases. In some patients hemodynamic pressure measurements in both lumina were obtained to further evaluate the significance of branch vessel involvement. Stent placement and balloon fenestration of the intimal flap were performed on a compassionate-use basis after informed consent was obtained from the patient or his or her family.

Stent placement. Stents were placed into an aortic branch artery if the vascular bed supplied by this vessel was compromised by extension of the dissection flap into the vessel,

narrowing or occluding its true lumen. In most instances, attempts were made to restore flow to a branch vessel from the true lumen of the aorta. However, if the vessel was adequately perfused by the false lumen, it was not treated. In some instances, stents were placed from the false lumen of the aorta to restore flow to the true lumen of a branch vessel. Aortic stents were placed to prop open the severely collapsed true lumen and allow flow to reach distal branches supplied exclusively by the aortic true lumen (Fig 2). Balloon-expandable Palmaz stents (Johnson & Johnson Interventional Systems, Warren, NJ) and self-expanding Wallstents (Schneider, Plymouth, Minn) were placed into renal, iliac, superior mesenteric, and brachiocephalic arteries, the celiac axis, and into the aorta. For stent placement procedures, angiographic sheaths ranging from 7F to 14F were used for arterial access.

Balloon fenestration. The technique of balloon fenestration has been previously described.^{4,7,8} Earlier in our experience, balloon fenestration was carried out in the descending thoracic aorta, where IVUS guidance was helpful. Subsequently, balloon fenestration was performed only at the aortic bifurcation to treat lower extremity ischemia, because more proximal fenestration in the thoracic aorta failed to consistently provide adequate redirection of distal blood flow. Because of the oblique angulation of the iliac arteries at the aortic bifurcation, adjunctive use of IVUS was less useful. Therefore all balloon fenestration procedures are currently performed with only fluoroscopic guidance.

To perform balloon fenestration (Fig 3), we percutaneously catheterized both the true and false lumina. After creation of the fenestration, flow through the fenestration was assessed arteriographically. If flow to the ischemic lower extremity was considered inadequate, a stent was placed in the iliac artery, bridging the fenestration in the distal aortic flap. No stents were placed across any fenestration that was performed above the level of the abdominal aortic bifurcation.

In most cases, at the conclusion of both stent placement and balloon fenestration procedures, the sheaths were removed in the angiography suite and hemostasis at the puncture site was obtained with manual compression.

Results

Of the 40 patients with a peripheral ischemic complication, 14 patients were treated with stenting of either the true or false lumen combined with balloon fenestration of the intimal flap, 24 with stenting alone, and 2 with fenestration alone. Many patients had stents placed in multiple vessels. Twenty-two patients had stents placed in a renal artery, 15 in an iliac artery, 6 in the superior mesenteric artery, 1 in the celiac axis, 1 in the brachiocephalic artery, and 12 in the aorta. In aortic branch vessels, stents ranged from 5 mm in diameter in a renal artery to 14 mm in diameter in an iliac artery. In the aorta, stents ranged from 10 to 25 mm in diameter. Balloon fenestration of the intimal flap was performed in the descending thoracic aorta in 8

Fig 2. Endovascular stent placement used to treat a 51-year-old man with mesenteric and renal ischemia related to a type B dissection. Before percutaneous intervention the patient had resection of an ischemic right colon. **A**, Digital subtraction arteriogram with the catheter in the aortic true lumen demonstrates a severely collapsed true lumen that supplies the celiac axis. The origin of the superior mesenteric artery is occluded by an intimal flap. It fills through collateral vessels from the gastroduodenal artery (*large arrows*) and then occludes a few centimeters beyond the origin of a replaced right hepatic artery (*small arrow*). **B**, Arteriogram with injection of the false lumen demonstrates supply to the right renal artery and both lower extremities. The left renal artery does not fill from either the true or false lumen. **C**, Spot image in a lateral view demonstrates stents in the aorta, the proximal superior mesenteric artery, and the left renal artery (*arrow*). **D**, Arteriogram in a lateral view with the catheter in the aortic true lumen demonstrates filling of the superior mesenteric and left renal arteries.

Fig 3. Balloon fenestration of the intimal flap used to treat an 80-year-old woman with left lower extremity ischemia immediately after surgical repair of a type A dissection. **A**, Arteriogram with injection in the aortic false lumen demonstrates flow to the right lower extremity but no flow to the left lower extremity. The true lumen was severely collapsed, with no flow below the renal vessels. **B**, A large angioplasty balloon catheter is inflated in the false lumen to use as a target. A curved metallic cannula is placed in the true lumen and pointed toward the inflated balloon in the opposite lumen. **C**, After a small needle is passed through the cannula into the opposite lumen, a 5F angiographic catheter is advanced over the needle, and the needle is exchanged for a guide wire. An angioplasty balloon is advanced over the guide wire and inflated to dilate the fenestration in the intimal flap. **D**, Arteriogram after creation of the fenestration demonstrates good flow from the false lumen through the fenestration to the left lower extremity.

patients, in the upper abdominal aorta in 3 patients, and just above the level of the aortic bifurcation in 7 patients. Fenestration was performed at 2 separate lev-

els in 2 patients. Fenestration diameter ranged from 14 to 20 mm in the thoracic aorta, from 12 to 16 mm in the upper abdominal aorta, and from 8 to 15 mm at the

Table II. Outcomes of percutaneous treatment by distribution of ischemic complications in 40 patients with acute aortic dissection

Ischemic region	N	Type		Bowel resection		Early dialysis	Early amputation	Complication		Outcomes			Mean follow-up (mo)
		A	B	Early	Delayed			Major	Minor	Death		Alive	
										Early	Late		
Renal/mesenteric/ lower extremity	11	1	10	2	1	4	1	1	2	6	1	4	15.5
Renal/lower extremity	6	2	4						1	1	1	4	29.6
Renal/mesenteric	3		3	1		1			1	1		2	21.5
Renal	10	3	7			2		1	1	1	2	7	19.8
Lower extremity	5	2	3									5	40.2
Mesenteric	4	1	3		1			1			1	3	52.0
Upper extremity	1	1							1	1			
Total	40	10	30	3	2	7	1	3	6	10	5	25	

level of the aortic bifurcation. Of the 7 patients who had a fenestration performed at the level of the aortic bifurcation, 3 had a stent placed through the fenestration into the iliac artery.

Flow was successfully restored to the ischemic regions in 37 of the 40 patients ($93\% \pm 4\%$). In 3 patients revascularization of an ischemic vascular bed could not be successfully achieved. One patient had occlusion of the superior mesenteric artery related to a type B dissection, and this vessel could not be catheterized for stent placement. The patient was treated with a renal artery stent and surgical revascularization of the superior mesenteric artery and left leg. Another patient with an acute type B dissection was treated with placement of stents in the aorta and bilateral iliac arteries for renal and leg ischemia; however, the outflow from the right iliac artery was obstructed by thrombus. While arrangements were being made for the patient to undergo surgical thrombectomy, the aortic false lumen ruptured and the patient died 35 minutes after the procedure. The final patient in whom percutaneous revascularization of an ischemic region could not be accomplished had a type B dissection with severe collapse of the true lumen and mesenteric, renal, and lower extremity ischemia. She was treated with an aortic stent graft and bilateral renal artery stents, followed by resection of most of the infarcted small bowel and surgical removal of the large groin sheaths. Postoperatively, the right leg was cold, with no popliteal pulse. No further heroic measures were deemed appropriate after discussion with the family. She underwent right leg amputation but died of multiorgan failure 6 days after the percutaneous procedure.

An additional 2 patients required early resection of infarcted bowel but died of multiorgan failure 21 and 24 days after the percutaneous procedure. Two patients required delayed bowel resection (resection of ischemic

Table III. Complications in patients treated percutaneously for peripheral ischemic complications of acute aortic dissection

Complication	N	%
Stent misplacement	3	7.5
Recaptured and deployed	1	
Removed percutaneously	1	
Removed surgically from femoral artery	1	
Femoral artery pseudoaneurysm	2	5.0
Ultrasound-guided compression	1	
Surgical repair	1	
Transient ischemic attack	1	2.5
Thrombosis of renal artery stent with renal atrophy	1	2.5
Posterior tibial artery embolus with transmetatarsal amputation	1	2.5
Infection	1	2.5
Total	9	22.5

sigmoid colon in 1 and resection of an ischemic small bowel stricture in the other, both 1 month later), and these patients have survived for 39 and 68 months, respectively. Seven patients (3 of whom had required early bowel resection and 1, delayed bowel resection) required early dialysis; five died within 30 days, and 2 were no longer dialysis dependent at 39 and 59 months. No additional patients required early amputation or surgical revascularization for limb ischemia.

Ten of the 40 patients ($25\% \pm 7\%$) died within 30 days of the percutaneous procedure, all while still in the hospital. Causes of death included multiorgan failure in 6, rupture of the false lumen of the descending thoracic aorta in 2, right heart failure from extension of the dissection into the right coronary artery in the 1 patient with a type A dissection who was not operated on, and

complications related to surgical repair of a type A dissection in 1. The only statistically significant predictors of early mortality were ischemia of 3 vascular beds ($e^{\beta} = 3.70$) and later procedure date ($e^{\beta} = 1.68$) ($P = .008$). Five patients have died beyond the first 30 days after the percutaneous procedure, 1 of rupture of the false lumen of the descending thoracic aorta at 76 days, 1 of squamous cell cancer at 20 months, and 1 of congestive heart failure after 21 months. Two patients died sudden, unexplained deaths at 13 and 24 months and did not have an autopsy. The remaining 25 patients were alive at a mean follow-up time of 29 months. The variables found to be significant, independent predictors of overall death according to multivariable analysis were ischemia of 3 vascular beds ($e^{\beta} = 4.23$) and older age ($e^{\beta} = 1.04$) ($P = .006$). The clinical outcomes and follow-up times in those still alive are shown in Table II, categorized by distribution of ischemic complications.

Complications. Nine patients had procedure-related complications, which are detailed in Table III. Of these, only 3 are considered major because they were associated with important clinical consequences: One patient had thrombosis of a stented renal artery that eventually caused atrophy of a kidney, another required a transmetatarsal amputation because of an embolus to the posterior tibial artery, and the third had sepsis with blood cultures positive for *Staphylococcus aureus* after placement of a renal artery stent (no other source of infection could be identified). This last patient was treated with antibiotics until his death from aortic rupture 2.5 months later.

Discussion

Aortic dissection is complicated by ischemia of peripheral vascular beds in approximately 30% of patients.^{1,2,9,10} Historically, patients with ischemic complications have had markedly increased mortality rates, regardless of medical or surgical treatment.^{1,2,10,11} Surgical repair of the aortic dissection often resolves the peripheral ischemia.^{2,12} However, if laparotomy for suspected bowel ischemia or infarction is subsequently required after repair of the dissection, the mortality rate rises to 80%.² Until recently, good alternatives to direct surgical revascularization have not existed for patients with acute type A dissections in whom peripheral ischemia persists after graft replacement of the ascending thoracic aortic. Similarly, patients who would otherwise be treated medically for a type B dissection have required surgical revascularization if peripheral ischemic complications arise. The evolution of percutaneous techniques provides the potential opportunity to reperfuse ischemic regions without the morbidity associated with a major vascular surgical procedure.

In the overall Stanford surgical experience of patients with aortic dissections, renal or visceral ischemia was an independent determinant of higher operative mortality risk.¹⁰ Similarly, focusing on just those patients undergoing thoracic aortic replacement who had peripheral vascular complications of their dissection, impaired renal perfusion was a significant, independent predictor of operative death, whereas stroke, paraplegia, and mesenteric or extremity ischemia were not.² In this current (smaller) study, ischemia of any one specific vascular bed was not linked with a higher likelihood of death; rather, in these 40 patients treated with percutaneous methods, ischemia of 3 vascular beds was the only significant patient-related risk factor for early mortality (and was associated with a nearly 4-fold increase in risk [$e^{\beta} = 3.70$]). The association between later procedure date and higher early mortality was initially thought to be related to treatment of an increasing number of patients with more severe or potentially irreversible peripheral ischemic complications as our experience with these techniques progressed. Close examination of the data, however, revealed 1 year (1996) during which 4 of the 5 treated patients died within 30 days of the procedure, likely skewing the statistics.

It is difficult to make strict comparisons between the surgically and percutaneously treated patients with peripheral vascular complications because the patient populations and eras of treatment differ substantially. Type A dissections were present in only 25% of the percutaneously treated patients in our series (and all save one had already undergone surgical repair of the ascending aorta), compared with 55%¹ to 73%² of patients in the earlier surgical series. When surgically treated patients with type B dissections who have peripheral vascular complications similar to those in this current series are considered, however, the 25% $\pm$ 7% early death rate after percutaneous treatment compares favorably with the earlier reported operative mortality rate of 64% for patients with acute type B dissections.² Again, strict comparisons between these series are not possible.

Surgical fenestration of the aortic intimal flap to relieve peripheral vascular ischemic complications has recently resurged as a viable therapeutic approach in the view of some surgical groups and is fairly well tolerated by most patients. Small series of patients who underwent surgical fenestration of the abdominal aorta either after graft replacement of the thoracic aorta or as an isolated primary therapeutic procedure have shown that it can be effective in relieving lower extremity, visceral, and renal ischemia, with early mortality rates ranging from 0% to 21%.^{13,14} As was the case in this report after percutaneous treatment, some patients

also required amputation¹³ or bowel resection¹⁴ despite restoration of branch vessel perfusion. Surgical fenestration is reported to improve symptoms of spinal cord ischemia rarely, something that was not observed in our experience after percutaneous interventions.

The severity and the duration of visceral ischemia before intervention undoubtedly have an important impact on outcomes and patient survival. By the time the clinical diagnosis of renal or mesenteric ischemia can be made with confidence, irreversible gut infarction has often already occurred. The catastrophic consequences associated with delayed diagnosis of ischemic intra-abdominal complications resulting from acute aortic dissection underscore the need for a high level of clinical suspicion, close monitoring, and a low threshold for performing diagnostic studies to assess the arterial perfusion status in patients with acute aortic dissection.

The occurrence of procedure-related complications in 9 of the 40 patients (23%) treated percutaneously is relatively high. Only 3 of these complications (8%), however, had long-term clinical sequelae. The frequency with which stents were misplaced was related to difficulty in choosing the appropriate stent size. The optimal stent diameter required in a dissected vessel is poorly characterized. Arbitrary oversizing may increase the risk of rupture in an already weakened artery; however, impaired elastic recoil of the arterial wall may not secure a stent expanded to less than the transverse diameter of the combined lumina.

Although the potential for successful reperfusion of ischemic vascular beds using percutaneous techniques has been demonstrated in patients with acute aortic dissections, debate continues concerning the most appropriate timing for surgical repair of the thoracic aorta and percutaneous treatment of peripheral ischemic vascular complications. Percutaneous and medical treatment of branch vessel ischemic complications with delayed surgical repair of the ascending aorta in patients with acute type A dissections has been advocated by the University of Michigan surgical group, in an attempt to decrease the very high surgical mortality rates seen earlier in similar patients in their center.⁶ Immediate percutaneous treatment of jeopardized downstream end-organs, followed without delay by surgical graft replacement of the thoracic aorta, has been suggested as one possible approach to prevent additional (and possibly irreversible) damage of ischemic end-organs during the surgical procedures on the thoracic aorta.¹⁵ Nine of the 10 patients with an acute type A dissection in this report had just undergone surgical repair of the thoracic aorta before percutaneous intervention. Inasmuch as mortality risk in untreated patients with acute aortic dissections approximates 1% per hour (with death usually being

due to aortic rupture causing pericardial tamponade or exsanguination into the chest or acute aortic valvular insufficiency),¹⁶ we continue to believe that immediate surgical repair of the ascending aorta is the most prudent course for almost all patients with acute type A aortic dissections. Repair of the thoracic aorta usually relieves the peripheral ischemia, but close postoperative surveillance is crucial to identify quickly any persistent or new distal ischemia or "malperfusion" such that percutaneous treatment may be undertaken expeditiously.

A smaller proportion of our patients with acute type B dissections had prior surgical repair of the thoracic aorta before percutaneous intervention. In untreated patients with acute type B dissection, early death is most frequently due to pleural rupture or peripheral ischemic complications. The likelihood of aortic rupture can be markedly reduced with appropriate medical treatment, and the peripheral ischemic complications can be treated with percutaneous techniques. If successful, this approach reduces the need for surgical repair of the descending thoracic aorta through a left thoracotomy, which carries substantial morbidity.

This study has several limitations. Our definitions of peripheral ischemia may have prompted percutaneous treatment of vascular beds that were not actually in critical jeopardy. This aggressive approach in treating potentially ischemic regions is consistent with our desire to avoid any delay in treatment, which may portend irreversible damage. Our follow-up data are only subjective, retrospective, and based on patient recollection. More information could have been gleaned from this study if we had had objective laboratory or imaging data or both for confirmation of prolonged patency of treated arteries and relief of ischemia. Finally, the number of patients is relatively small, and the available follow-up is relatively short; additional evaluation out to 5 to 10 years in larger numbers of patients will be necessary before the real therapeutic effectiveness of percutaneous fenestration and stenting in patients with peripheral ischemic complications resulting from acute aortic dissections can be known with certainty.

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Discussion

Dr Scot H. Merrick (*San Francisco, Calif*). I thank Dr Slonim and her colleagues for updating us on the Stanford experience. This experience is the largest in the world and is very impressive.

The traditional teaching for aortic dissection is that acute type A dissections should be treated surgically, whereas acute type B dissections can be either operated on or observed depending on the patient and perhaps the surgeon's philosophy. In most cases those patients do reasonably well. The patients that you have just discussed are the most difficult to treat and have the highest mortality. In fact, treatment options have really plateaued over the past decade in this population; perhaps your strategy at this point is opening the door a bit for new treatment therapies.

I do have several questions. The first relates to the outcome of organ ischemia. The data on mortality are useful, but do you have any objective data, clinical or biochemical, that

would indicate what happens to the organ ischemia before and after the procedure?

Dr Slonim. I do not have objective data; I have subjective clinical data. We have monitored weight gain and loss, as well as gastrointestinal symptoms, in patients with mesenteric ischemia. In patients with renal ischemia, we know whether they have begun using dialysis, and we know how many medications it takes to control their blood pressure. A few patients with renal ischemia have had nuclear medicine renal scans to evaluate renal perfusion. Overall, however, objective follow-up information is minimal. The follow-up we do have suggests that patients who survive the acute crisis are likely to do well in the long term.

Dr Merrick. I imagine these procedures take time in your interventional suite and a certain amount of contrast material is delivered. Did you find any correlation between the length of the procedure and/or the amount of contrast material delivered and the morbidity of the procedure?

Dr Slonim. We have not evaluated the length of the procedure or the amount of contrast material used during the procedure. In terms of contrast load, in patients in whom we suspect renal ischemia, we perform portions of the diagnostic study with carbon dioxide arteriography with digital subtraction. Also, if we have a high-quality computed tomographic scan beforehand, we can limit the amount of contrast medium used during arteriography. Obviously the computed tomographic scan involves a contrast load, but the benefit of knowing the anatomy of the dissection before we begin allows us to limit the time and contrast material for the interventional procedure. Alternatively, intravascular ultrasound can be used to define the anatomy of the dissection before we intervene.

Dr Merrick. I have a theoretical question regarding the patient whose angiogram you presented. If you have a patient with a type B dissection with a severe collapse of the true lumen and you stent the aorta, the proximal entrance point is still open, and yet you may be occluding the exit points down in the distal aorta. Do you think that could result, either immediately or over the long term, in an increased risk of rupture, extension of the dissection, or aneurysm formation?

Dr Slonim. I do not think we occlude the re-entry tear. There may be re-entry tears at multiple levels along the length of the aorta, related to the intercostal and visceral arteries. The stents we use have fairly large interstices. In fact, we have stented over the origins of visceral vessels that maintain flow through the stents, so I do not think that we occlude re-entry tears.

We do not know, however, what happens in the long term to patients who are treated with aortic stents. It is true that we are leaving the entry tear open, and the false lumen does stay patent. It seems reasonable that patients whose blood pressure is inadequately controlled would be at risk of aneurysmal enlargement and potential rupture of the false lumen.

Dr Merrick. Of the 11 patients with acute type A dissection, 2 were treated percutaneously first. Does that represent a change in either Dr Miller's or Dr Mitchell's philosophy on treating type A dissection?

Dr Slonim. One of those patients had a tiny dissection flap in the arch, just proximal to the left subclavian artery, with no involvement of the ascending aorta. The other patient had a heparin-induced antibody, and therefore surgery was withheld. I do not believe that the aggressive surgical approach to patients with a type A dissection at Stanford has been altered.

Dr Merrick. I am relieved. Finally, do you prescribe long-term anticoagulation for those patients with stents?

Dr Slonim. No, we do not. In rare instances we have noticed clot forming during the procedure and have used

heparin or urokinase. However, we are usually dealing with an acutely dissected aorta, and we are reluctant to use anticoagulation. In general, stents placed in a patient with an aortic dissection are dilated larger than expected for nondissected vessels, and the amount of flow through them is sufficient to keep them patent without anticoagulation. One renal stent thrombosed immediately on deployment. Otherwise, we have not had any stents thrombose.

Dr Merrick. I look forward to the long-term imaging studies on these patients.

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