Preoperative selective intercostal angiography in patients undergoing thoracoabdominal aneurysm repair

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Objective: **This study was designed to test the hypothesis that detection of the location of the major artery supplying the spinal cord, that is, the artery of Adamkiewicz or the great radicular artery (GRA), with angiography would help prevent paraplegia. Knowing which intercostal artery provides this important branch would enable prompt, focused revascularization.**

Method: **The surgical outcome in 131 patients with Crawford extent 1 and 2 degenerative aneurysms and 69 patients with descending thoracic aortic dissection was correlated with findings on selective intercostal arteriograms. Angiographic maneuvers were done with care, and the procedures were aborted if there was loose or "shaggy" mural thrombus, significant tortuosity, or difficulty entering each dissection channel. No attempts were made to find major contributions proximal to T6. Subarachnoid drains were placed in all patients, and all but five patients underwent distal aortic perfusion with controlled cooling to 32°F. Five patients underwent cold circulatory arrest, enabling replacement of the distal aortic arch. We defined paraplegia simply as the inability to walk at hospital discharge, paraparesis as impaired ambulation, and both as having spinal cord dysfunction (SCD).**

Results: **A GRA was found in 65 (43%)of the 151 patients studied. Of the 65 patients with the GRA identified, SCD developed in 3 (4.6%) patients. Thirteen of 135 (9.6%) patients in whom the GRA was not identified, either because they were not studied or were studied and the GRA was not found, developed SCD ($P = .35$) However, when the GRA was identified, SCD occurred only in the group with aortic dissection. None of the 45 patients with degenerative aneurysms with the GRA identified had SCD, compared with 9 of 55 (16%) patients studied but without a GRA found ($P = .01$).**

Conclusion: **The approach with selective intercostal angiography did not improve overall results. One third of our patients were not studied, and they fared as well as patients who were studied and the GRA was localized (not studied, 4 of 49, 8% with SCD; GRA localized, 3 of 65, 5% with SCD; $P = .8$). However, when the GRA was found, SCD occurred only in patients with aortic dissection. The studies confirmed the concept that the existence of mural thrombus in degenerative aneurysms results in the occlusion of many intercostal arteries, leaving those remaining patent to supply rich vascular watersheds through acquired collateral channels. As a result, in the group of patients with degenerative aneurysms, the identification of the critical intercostal artery allows focused reimplantation with uniform success. This is not the case in patients with aortic dissection. In those patients, most intercostal vessels remain patent, such that the insertion of one pair is insufficient to supply the paravertebral plexus and the spinal cord. Finally, failure to identify the GRA angiographically with our methods does not provide assurance that the GRA does not exist. Therefore negative findings did not provide license to ligate all intercostal arteries. (J Vasc Surg 2004;39:314-21.)**

Despite the application of adjunctive measures, spinal cord dysfunction (SCD) resulting in paraplegia and paraparesis continues to be a leading cause of morbidity attending repair of thoracoabdominal aneurysms.[1-8] While adjunctive measures have had a role in reducing the incidence of SCD, there remain 5% to 10% of patients with Crawford extent I and II degenerative and DeBakey type III dissecting aneurysms in whom SCD develops despite the application of appropriate adjuncts. Although multiple compounding mechanisms likely are responsible, it is clear that the common denominator is an initial period of ischemia and the need to attach frequently diseased intercostal arteries to the new graft. We reasoned that ischemia would be reduced by the ability to identify precisely which intercostal arteries were important providers of blood to the spinal cord. We recognized from the onset that spinal angiography entailed some risk for producing direct toxic injury to the spinal cord by overenthusiastic injection of iodinated compounds and that atheroemboli released with manipulation within the aneurysm sacs could lead to serious sequelae. Angiographic procedures were to be aborted in the presence of loose, shaggy mural thrombus, extreme tortuousity, or difficulty in engaging an important channel in aortic dissection. We focused attention on Crawford extent I and II degenerative aneurysms and DeBakey III aortic dissections. In all such cases surgical repair entails replacement of the aorta in the area most likely to provide significant blood supply to the spinal cord. This communication

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deals with correlations between radiographic findings and surgical outcome.

MATERIAL AND METHODS

Patients. Operative and postoperative management has been standardized for the last 10 years,[9,10] and we chose this period of time to develop correlations between radiographic findings and patient outcome. All patients with Crawford extent I or II degenerative aneurysms and DeBakey III extent aortic dissections, including those with acute presentation, were included. We tried to perform angiography in all patients, but this was logistically difficult in many instances; thus 31 of 131 patients with degenerative aneurysms and 18 of 69 with aortic dissection were not studied.

Angiography. The standard Seldinger technique was used. Descending thoracic aortography with a pigtail catheter provided a rough outline of the location of patent intercostal arteries. This was followed by selective hand injection of individual intercostal arteries from T6 to L2. Simmons type I and II catheters and the Cobra Glide catheter were used. The great radicular artery (GRA) was identified when an intercostal branch was found making a cephalad hairpin turn to enter the spinal canal and supply a midline longitudinal artery (Fig 1). The studies were curtailed at the discretion of the testing angiographer.

Spinal fluid drainage. In all patients spinal fluid was drained intraoperatively by means of lumbar puncture and insertion of an indwelling subarachnoid catheter. The catheter was connected to a drainage system set to sustain a pressure of 10 cm of water above the level of the ear. For 1 year we reduced the pop-off pressure to 5 cm of water, which may have been associated with a higher proportion of patients with epidural hematomas, and therefore we returned to using the 10 cm pressure. In cases of delayed-onset paraplegia or paraparesis we reduced the pressure to 5 cm of water or instituted new drainage if SCD occurred after the third postoperative day.

Surgical technique. A posterolateral thoracoabdominal incision was made resecting the sixth rib and notching the fifth rib, and in some cases performing a double thoracotomy to gain proximal exposure by resection of the fifth rib and distal exposure through the ninth or tenth interspace.[9,10] The diaphragm was always divided peripherally, and the subdiaphragmatic aorta was exposed extraperitoneally behind the left kidney. By displacing the kidney anteriorly the entire aorta and proximal common iliac vessels were exposed. Distal aortic perfusion was achieved in most instances with cannulation of the left inferior pulmonary vein or the left atrial appendage for outflow. Of the two techniques, the former is preferred, because of its ease. An 8-mm Dacron graft was sutured to the side of the left common femoral artery, and cannulated to serve as the distal inflow. The advantages of attaching a graft were twofold. It enabled the passage of a catheter to record the distal aortic pressure proximal to the inflow catheter, and the left leg was always perfused during bypass, eliminating peripheral ischemia as the cause of intraoperative deterioration of evoked potentials.

The intercostal arteries were implanted as one or two pairs, in later years including as little aorta as possible in the anastomosis.[11] At times, when the important intercostal orifice was very close to the celiac axis an 8- mm Dacron graft was used to anastomose to the pair of intercostal arteries and to the side of the Dacron graft. The Crawford inclusion technique was used in all cases for the rapid restoration of blood supply to the celiac axis, superior mesenteric artery, and right renal artery. The anastomoses were always done in sequence, as described previously,[9] working from proximal to distal. This reduced the ischemic interval to the specific time needed for revascularization of the part to the graft. In no case was cold perfusion of the superior mesenteric artery or renal artery used, but all patients were cooled with heat exchanger to 32°F before any ischemic insult.

When the GRA was not found at arteriography or the patient was not studied, the surgeon exercised judgment about which intercostal pair or pairs should be put back into the circulation. Full cardiopulmonary bypass and cold circulatory arrest were used in five patients who underwent concomitant distal arch repair.[10]

After the patient awakened, leg movement was checked hourly during the 3 days that the patient stayed in the intensive care unit having spinal fluid drained. Any loss of neurologic function in the legs required neurologic consultation and magnetic resonance imaging (MRI) of the spine. The exception to this protocol was in patients subjected to cold hypothermic arrest. All of these patients had temporary disturbances in brain function or delirium. Patients who died in the operating room or in the intensive care unit before neurologic assessment were excluded from the study.

RESULTS

Spinal angiography. There were no neurologic or embolic complications in the 151 patients studied. The number of patent intercostal arteries varied widely. In general, the presence of thrombus on computed tomography (CT) scans foretold occlusion of the intercostal vessels at that location. In addition to variation in number, there was variation in the size of the intercostal vessels. Without exception, when there was a limited number of patent intercostal arteries the injection of one that was patent provided flow to several that were occluded at the orifice (Fig 2). Thus the circulation was sustained to radicular and intercostal arteries by a rich paravertebral collateral network (Fig 1). In the case of aortic dissection, occlusion of intercostal vessels was the exception rather than the rule (Fig 3).

The GRA (Fig 2) varied not only in location but in size. In the case of a large artery, flow sometimes proceeded in both cephalad and caudad directions within the spinal cord, thus clearly establishing this as a critically important vessel. At other times the hairpin vessel was very small and hard to detect, which created skepticism about its importance.

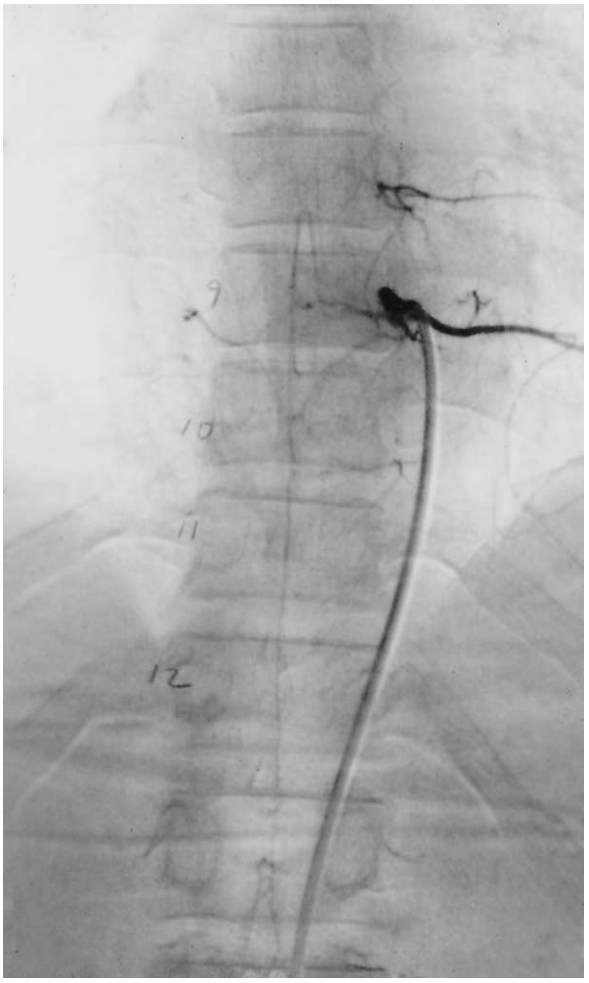

Fig 1. Selective injection of the intercostal artery at T9 demonstrates well the great radicular artery. Such vivid demonstration was rare, and most arteriograms outlined a much less distinct hairpin turn, with less visualization of the anterior spinal artery.

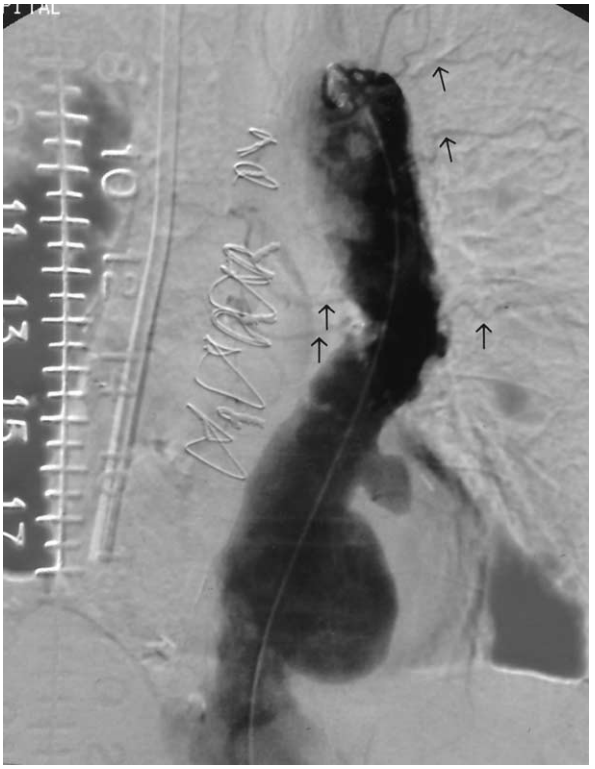

Fig 2. Flush thoracic aortogram in a patient with a fusiform extent 2 aneurysm. Note the limited distribution of intercostal arteries *(arrows)*. In such cases the patent intercostal arteries at T6 and T7 were selected for study and reimplantation, and those arising superiorly were oversewn.

However, in all cases there was agreement between radiologist and surgeon that the branch was or was not the GRA.

The ability to delineate the artery is shown in Table I. Of 53 patients studied with degenerative aneurysms of Crawford extent I, the artery was found in 26 patients (49%). In the 47 patients with extent II the GRA was detected in 19 (40%) patients. In the case of aortic dissection the artery was found in 20 of 51 (39%) patients. Neither the cause nor the extent of the aneurysm influenced the ability of the radiologist to detect the important artery. One third of the patients in each group were not studied. Standard risk factors were evaluated to determine whether they influenced whether patients were studied or, if studied, the GRA was found. Our patients were grouped by type of aneurysm, whether the GRA was found (Tables II, III), and whether they were studied. It seemed likely that patients with acute presentation would constitute a good proportion of the group not studied. However, acute presentation occurred in only 4% of our patients, and did not explain why one third of our patients were not studied. No other factor evaluated provided an explanation. The groups were closely matched. Hypertension and smoking were common in all groups. It was surprising that diabetes and renal disease were rare.

Spinal cord dysfunction. SCD occurred in 10 of 131 (7.6%) patients with degenerative aneurysms, and 6 of 69 (8.7%) patients with aortic dissection. The features that characterized these 16 patients are shown in Table IV. Of the patients with SCD, 12 had paraplegia, 3 had paraparesis, and 1 had monoplegia. Nine of the 16 patients had early-onset SCD, while 7 had delayed-onset dysfunction despite attention to every adjunct. Two of the 7 patients with delayed onset of dysfunction were of particular interest.

One patient with a Crawford extent II aneurysm underwent implantation of the T10 intercostal pair as a best guess. Delayed-onset left leg monoparesis developed. MRI of the spine yielded negative findings. Four weeks later while in a nursing home the patient had a myocardial infarction associated with hypotension, which resulted in dense paraplegia.

The second patient was operated on because of threatened rupture of a DeBakey IIIA aortic dissection. Three

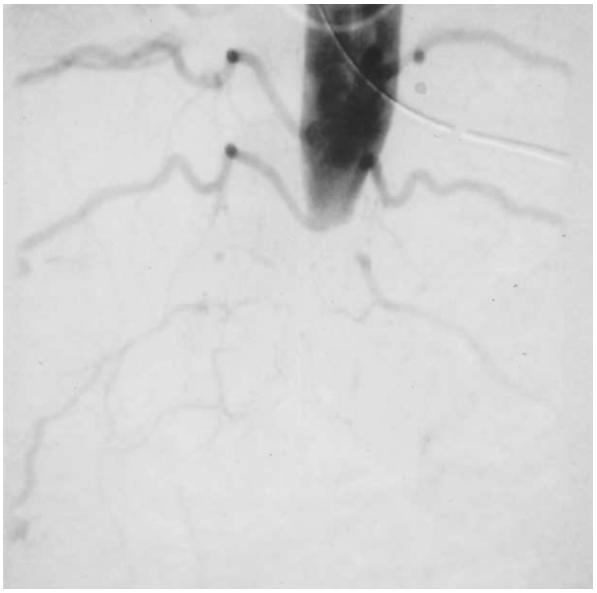

Fig 3. Flush thoracic aortogram in a patient with subacute aortic dissection. The channel studied ended blindly, and patency was maintained with open intercostal arteries. Note the limited collateral vessel development between patent arteries and the beginning collateral vessel development distal to the occlusion.

pairs of intercostal arteries, T9 to T11, were included in the distal anastomosis. On the eighth postoperative day, without any precipitating event, severe bilateral leg weakness developed, which was treated with insertion of a subarachnoid drain, with nearly complete restoration of neurologic function.

The principal finding in this study is that SCD developed in none of 45 patients undergoing repair of extensive degenerative aneurysms with the GRA origin identified. In contrast, NMD developed in 10 of 81 (12%) patients after degenerative aneurysm repair in whom the origin of the GRA was not identified ($P = .01$). The detection of the origin of the GRA was not so helpful in the case of aortic dissection, because NMD developed in 3 of 20 (15%) patients when the origin of the artery was known, compared with 3 of 49 (6%) patients when the origin of the GRA was unknown. We believe the cause of NMD in the three failures with the GRA known is significant. In all 3 patients reliance was placed on establishing perfusion to a single pair of intercostal arteries, whereas most of the intercostal arteries were patent and ligated.

In one of these 3 patients a single intercostal pair at T10 known to supply a reasonable GRA was attached to the graft with a short segment of Dacron graft. The anastomosis included the aorta, encompassing the single intercostal pair. When checked with Doppler scanning there was sluggish flow in this graft, but vigorous flow in the intercostal vessels themselves, which were graft-dependent. Motor evoked potentials were lost, and did not return after completion of this anastomosis. The patient awoke with dense paraplegia, and subsequent magnetic resonance angiograms (MRA) failed to demonstrate graft patency.

Table I. Great radicular artery found, by aneurysm type and extent

Aneurysm type	*Found*	*Studied, not found*	*Not studied*	
			n	*%*
1*	26	27	18	34
2†	19	28	13	35
Dissection	20	31	18	35

*Forty-nine percent of those studied were found.
†Forty percent of those studied were found.

A second patient with a IIIB aneurysm had a critical intercostals artery at T11. There were collateral vessels from T12 to T11, and we reasoned that if we ended our graft and started our tailoring[12] at T12 these collateral vessels would sustain spinal cord perfusion. Onset of dense paraplegia occurred on day 2.

The third patient was similar to the second, with a Dacron graft extending to a tailored section. However, the tailored section contained only the critical T12 intercostal artery and poor lumbar arteries.

Despite our intention to implant some intercostal arteries in each case, in 8 of the 13 patients with NMD, with unknown origin of the blood supply to the spinal cord, no intercostal vessels were implanted. In 4 patients the operation was done on an emergency basis, and a sense of urgency to reestablish aortic continuity may have been primary. In two instances motor evoked potentials were normal at the conclusion of the procedure, which suggests that collateral circulation was adequate.

DISCUSSION

Spinal cord angiography in patients with extensive aortic aneurysms has been reported by two other groups. Keiffer et al[13] reported detailed studies in which the GRA was found in 86% of 487 patients studied. This group also sought the middle dorsal and superior dorsal contributions. Their success in finding the GRA was twice as high as ours, which is related to the diligence of their radiologists and our philosophy of not struggling to find the artery in the presence of loose thrombus, tortuousity causing high torque in the interrogating catheter, and when one dissection channel could not be entered easily. However, they reported only six complications in this large series, not including aneurysm rupture in 2 patients at 3 days postprocedure. From their study and others, the GRA arose as a branch of an intercostal artery most commonly located at T9 to T11. However, 43% of the GRAs arose from arteries higher or lower. There was no report of surgical correlations. Minatoya et al[14] did report surgical correlations with angiography. Paraplegia developed in 3 patients with the GRA identified, giving rise to the conclusion that angiography was of no benefit. The patency of the reattached intercostal arteries was not reported, thereby failing to differentiate between surgical and radiologic failure.

Table II. Fusiform aneurysms

	GRA found (n = 45)		*GRA not found (n = 55)*		*Not studied (n = 31)*	
	n	*%*	*n*	*%*	*n*	*%*
Age (y)	66		69		65	
Hypertension	40	89	45	82	23	74
Diabetes	2	4	4	7	0	0
Renal disease	3	7	7	16	1	3
COPD	10	22	15	27	8	26
CAD	18	40	27	49	11	35
PVD	18	40	23	42	8	26
Smoker	39	87	40	73	21	67
Stroke	6	13	12	22	5	16
SCD	0	0	9	16*	1	3†

GRA, Great radicular artery; *COPD,* chronic obstructive pulmonary disease; *CAD,* coronary artery disease; *PVD,* peripheral vascular disease; *SCD,* spinal cord dysfunction.

*Found vs not found or studied, $P < .01$.

†Not studied vs not found, $P = .08$ (Fisher exact test).

Table III. Aortic dissection

	GRA found (n = 20)		*GRA not found (n = 31)*		*Not studied (n = 18)*	
	n	*%*	*n*	*%*	*n*	*%*
Age (y)	60.4		60		51	
Hypertension	19	95	28	90	14	78
Diabetes	2	10	1	3	0	
Renal disease	3	15	5	17	1	6
COPD	1	5	3	10	3	17
CAD	7	35	12	39	5	28
PVD	4	20	7	23	4	22
Smoker	10	50	18	58	11	61
Stroke	1	5	2	6	1	6
Marfan syndrome	4	20	3	10	8	44
SCD	3	15	0	0	3	17

GRA, Great radicular artery; *COPD,* chronic obstructive pulmonary disease; *CAD,* coronary artery disease; *PVD,* peripheral vascular disease; *SCD,* spinal cord dysfunction.

Table IV. Patients with spinal cord dysfunction

				Paralysis	
Fusiform	*Studied*	*GRA identified*	*Implanted*	*Delayed*	*PP/P*
Extent 1					
1	Yes	No	None	No	P
Extent 2					
2	Acute		None	Yes	P
3	Yes	No	None	No	PP
4	Yes	No	None	No	P
5	Yes	No	T11	Yes	PP-P
6	Yes	No	T11	Yes	P
7	Yes	No	None	No	P
8	Yes	No	T10	Yes	P
9	Yes	No	T9	No	P
10	Yes	No	None	Yes	PP
Aortic dissection					
11	Acute		None	No	P
12	Acute		None	No	P
13	Acute		None	Yes	PP-N
14	Yes	Yes (T10)	T10	No	P
15	Yes	Yes (T12)	T12 Tailored[12]	No	P
16	Yes	Yes (T11)	T12 Tailored[12]	Yes	P

P, Paraplegia; *PP,* paraparesis; *N,* normal.

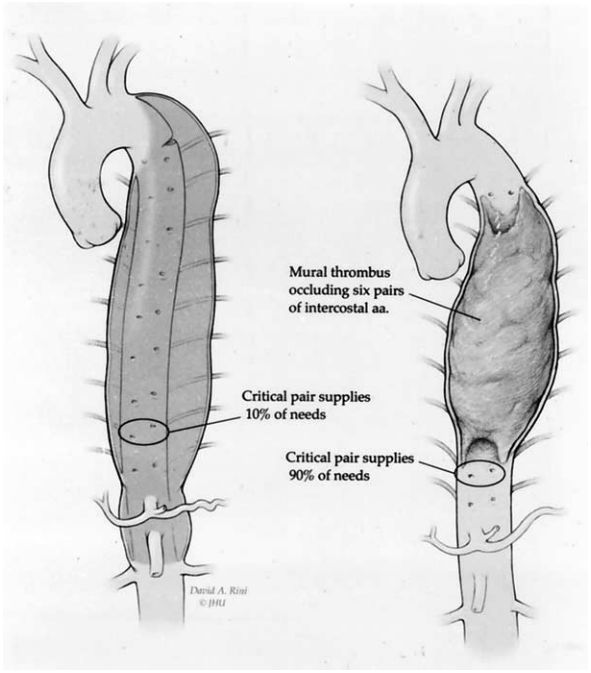

Fig 4. Drawings demonstrate the differences in intercostal circulation between fusiform *(left)* and chronic *(right)* dissecting aneurysms.

Inasmuch as our overall rate of SCD was 8%, which was achieved without angiography, we cannot support our hypothesis that finding the artery would improve results. There are three principal reasons for this. First, failure to identify the origin of the GRA as a result of our philosophy of doing no harm did not equate with the absence of this vessel. However, we were probably influenced by a negative finding to ligate rather than reconstruct the vessels[15]; intercostals arteries were reattached in only 4 of 9 patients. The rate of paraplegia was highest in the group of patients with degenerative aneurysms who were studied but the GRA was not found (Table II). The rate in that group (16%) was higher than in the group not studied (3%; $P = .08$). Second, identifying the location of the GRA and focused attachment was not helpful in the case of chronic expanding aortic dissection. Third, the cohort of patients with degenerative aneurysms and the GRA found was too small (23%) to have an effect on results overall, despite our finding that no patient in this group had SCD. Thus the "value" of the present angiographic-surgical correlations is not so much to provide a solution to the complication of paraplegia as to gain insight into the intercostal-paravertebral circulation.

Positive identification of the GRA in patients with degenerative aneurysms enables successful reattachment of limited bits of aorta, including the orifices of the intercostal pair providing the GRA branch. Indeed, uniform success was achieved. We believe this is the result of mural thrombus occluding many intercostal arteries in degenerative aneurysms. Arteries remaining patent supply a rich network of adjacent intercostal arteries and numerous paravertebral arteries. Outflow is excellent, and almost any form of revascularization will function well. This is not the case in aortic dissection when almost all intercostal arteries are open and collateral vessels have not formed (Fig 4). When we tried to revascularize one T12 pair with a side graft, the procedure failed. When we ligated all but T12, and T12 was the critical artery, paraplegia resulted.

Thus, in the case of aortic dissection, several intercostal branches should be attached. This is not a trivial job, because major backbleeding always occurs. This not only depletes blood volume, but turns the paravertebral arterial system into one having just atmospheric pressure. Further, attempts to reduce backbleeding run the risk of injuring these small and fragile vessels, and adjacent structures such as the thoracic duct. Nevertheless, the lesson learned is that it is best to gain control of the intercostal arteries with careful dissection before aortotomy, with use of microvascular clamps to occlude the vessels to be preserved. A cluster of three should be sutured as a very narrow button to prevent subsequent patch aneurysm.[11]

It is possible to localize segments of aorta with patent intercostal arteries with thin-section CT. We have not been able to follow the vessels into the spinal canal with any consistency, but knowing where to look will make finding the GRA easier. There are reports of finding the GRA with MRA.[16,17] It is hoped we will have less invasive methods in the future. However, at this time postoperative evaluation of intercostals artery patency is possible with CT, and should be done routinely to evaluate methods of revascularization. Finally, the techniques of motor evoked potentials advocated by Jacobs et al[18] need to be used by other groups to determine whether persistence at intercostal implantation to the point of restoring electrical normality before finishing the operation will reduce SCD to their remarkably low levels.

The information presented in this article was made possible by the contributions of many interventional radiologists, led by Dr Floyd Osterman, and including Drs Sally Mitchell, Stephen Kaufman, Anthony Venbrux, Gunnar Lund, Arvind Arepally, Lawrence Hofmann, and many fellows.

REFERENCES

1. Estrera AL, Miller CC III, Huynh TT, Porat E, Safi HJ. Neurologic outcome after thoracic and thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 2001;72:1225-30; discussion 1230-1.
2. Cheung AT, Weiss SJ, McGarvey ML, Stecker SMM, Hogen MS, Escher A, et al. Interventions for reversing delayed-onset postoperative paraplegia after thoracic aortic reconstruction. Ann Thorac Surg 2002; 74:413-9; discussion 420-1.
3. Coselli JS, Lemaire SA, Koksoy C, Schmittling ZC, Curling PE. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: results of a randomized clinical trial. J Vasc Surg 2002;35:631-9.
4. Zvara DA. Thoracoabdominal aneurysm surgery and the risk of paraplegia: contemporary practice and future directions. J Extracorporeal Technol 2002;34:11-7.
5. Gloviczki P. Surgical repair of thoracoabdominal aneurysms: patient selection, techniques and results. Cardiovasc Surg 2002;10:434-41.

6. Wada T, Yao H, Miyamoto T, Mukai S, Yamamura M. Prevention and detection of spinal cord injury during thoracic and thoracoabdominal aortic repairs. Ann Thorac Surg 2001;72:80-4; discussion, 85.
7. Maniar HS, Sundt TM III, Prasad SM, Chu CM, Camillo CJ, Moon M, et al. Delayed paraplegia after thoracic and thoracoabdominal aneurysm repair: a continuing risk. Ann Thorac Surg 2003;75:113-9; discussion, 119-20.
8. Safi HS, Miller CC, Carr C, Iliopoulus DC, Dorsay DA, Baldwin JC. Importance of intercostal artery reattachment during thoracoabdominal aneurysm repair. J Vasc Surg 1998;27:58-66; discussion, 66.
9. Frank SM, Parker SD, Rock P, Gorman RB, Kelly S, Beattie C, et al. Moderate hypothermia with partial bypass and segmental sequential repair for thoracoabdominal aortic aneurysm. J Vasc Surg 1994;19:687-97.
10. Stone CD, Greene PS, Gott VL, Frank S, Williams GM. Single-stage repair of distal aortic arch and thoracoabdominal dissecting aneurysms using aortic tailoring and circulatory arrest. Ann Thorac Surg 1994;57:580-7.
11. Dardik A, Perler BA, Roseborough GS, Williams GM. Aneurysmal expansion of the visceral patch after thoracoabdominal aortic replacement: an argument for limiting patch size? J Vasc Surg 2001;34:405-10.
12. Williams GM. Treatment of chronic expanding dissecting aneurysms of the descending thoracic and upper abdominal aorta by extended aortotomy, removal of the dissected intima, and closure. J Vasc Surg 1993;18:441-9.
13. Kieffer E, Fukui S, Chiras J, Koskas F, Bahmini A, Cormier E. Spinal cord arteriography: a safe adjunct before descending thoracic or thoracoabdominal aortic aneurysmectomy. J Vasc Surg 2002;35:262-8.
14. Minatoya K, Karck M, Hagl C, Meyer A, Brassel F, Harringer W, et al. The impact of spinal angiography on the neurological outcome after surgery on the descending thoracic and thoracoabdominal aorta. Ann Thorac Surg 2002;74(suppl):S1870-2; discussion, S1892-8.
15. Acher CW, Wynn MM, Iloch JR, Popic P, Archibald J, Turnipseed WD. Combined use of cerebral spinal fluid drainage and naloxone reduces the risk of paraplegia in thoracoabdominal aneurysm repair. J Vasc Surg 1994;19:236-46; discussion, 247-8.
16. Yamada N, Okita Y, Minatoya K, Tagusari O, Ando M, Takamiya M, et al. Preoperative demonstration of the Adamkiewicz artery by magnetic resonance angiography in patients with descending or thoracoabdominal aortic aneurysms. Eur J Cardiothorac Surg 2000;18:104-11.
17. Kawaharada N, Morishita K, Fukada J, Yamada A, Muraki S, Hyodoh H, et al. Thoracoabdominal or descending aortic aneurysm repair after preoperative demonstration of the Adamkiewicz artery by magnetic resonance angiography. Eur J Cardiothorac Surg 2002;21:970-4.
18. Jacobs MJ, Elenbaas TW, Schurink GW, Mess WH, Mochtar B. Assessment of spinal cord integrity during thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 2002;74(suppl):S1864-6; discussion, S1892-8.

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DISCUSSION

Dr Hazim J. Safi (Houston, Tex). This is a great paper, and it presents wonderful results. But my question to you is, How do you convince your radiologists to do it? Short of using a machine gun, I couldn't.

Dr G. Melville Williams. They're very tractable. And as I said, they all take turns. Some, I think, are much more fastidious in trying to find this artery than not. I think they're going to like these data, because they show that persistence is going to be important in some of these patients with fusiform aneurysms.

Dr Safi. What do you think of the work of Peter de Haan, from Amsterdam, where he showed in the pig that if you ligate the lower intercostal arteries the upper intercostal arteries become important? Hence, I change my practice, where if I open the aneurysm and the lower intercostal arteries are occluded I reimplant 6, 7, and 8.

Dr Williams. I think that's important to do. I think the more intercostals you can put in the better, running the risk always that what you implant of native aorta may degenerate in the case of patients who have aneurysms, particularly those with connective tissue disorders.

Dr Richard P. Cambria (Boston, Mass). Thanks for a good paper, Mel. Certainly I've quoted your earlier work with Dr Roseborough often, in terms of writing about patterns of intercostals in patients with thoracoabdominal aneurysms.

I have three questions. What is your strategy when in fact you haven't found the greater radicular artery angiographically and you open the aorta and there are three or four pairs of intercostals in the critical segment?

Second, what was the cumulative morbidity and complications of doing this angiography in patients, who often have renal dysfunction and a lot of mural debris in their visceral aortic segment?

And third, I've come to look carefully at the pattern of mural thrombus on axial CT scans, because I think one can use this to predict that intercostals in the critical segment will or won't be open.

Dr Williams. Thank you for your perceptive questions, and they're all good ones.

What do we do when we open the aneurysm and can't find anything to put in? Well, I think these data have encouraged us to keep looking and perhaps do, as Dr Jacobs has recommended, an endarterectomy, trying to free up intercostals and reimplanting those that backbleed.

Dr Cambria. The question was, when you haven't found the artery preoperatively, but you open and you find multiple open pairs, how do you handle that?

Dr Williams. I think we would go for the lower three at the present time. But you're taking a guess.

With respect to the morbidity of doing the angiographic procedures, we have not had a single major complication from doing it. We have always erred on the side of terminating the exam if we feel that we're getting into trouble with this excess contrast or if we can see some of the thrombus being loose and waving with the heartbeat.

Axial CT, I think, is coming into its own. I wouldn't be surprised if within the next several years improvements in either CT or MRA are going to help us look at the aorta quite differently. We're already able from CT scans to identify which intercostal arteries are open and which are occluded. So that we know basically the information that we would have from a flush aortogram with the CT scan. And I think it's a good clue to go from that into what we ought to revascularize.

I think also that it's going to be critically important when we put in these intercostal vessels to have follow-up studies to know whether they're open or not. Because until we can show one-to-one correlation between here is the artery we think goes to the spinal cord, we put it in, and yes it's good or no it's not, we really won't know the success or failure of our procedures.

Dr Peter Gloviczki (Rochester, Minn). I enjoyed very much your presentation.

Tell me, can you identify on the arteriogram if the artery of Adamkiewicz is really critical, if reimplantation of that single artery is going to protect the cord? I'm just wondering, like in the aortic dissection cases, where you have to implant three or four arteries, it almost appears that there is not a critical artery but there are multiple arteries. So can you maybe make your decision on the preoperative arteriogram anatomy?

And my second question that always returns is, How do you explain the results of those who oversew all intercostal arteries during thoracoabdominal repair?

Dr Williams. I think that's the sticking point for all of us. And it happens, and I don't know why. I think one of the problems that we have with this study is that in the 16 paralyzed patients in whom we did not find an anterior spinal artery supply, we only put intercostals vessels in 9 of them, because it was sort of our hope that was the group of patients whom Dr Ascher and those deploying stent grafts indicate as not needing their intercostal arteries in. I think our data make us suspicious that this is not the case and that intercostals ought to be reimplanted in everybody.