WHAT WOULD YOU DO?

Residual Type B Aortic Dissection With Worsening Renal Malperfusion

MODERATOR: JOSEPH LOMBARDI, MD
PANELISTS: ADAM BECK, MD; G. CHAD HUGHES, MD; AND DARREN SCHNEIDER, MD

CASE PRESENTATION

A 63-year-old man presents after type A dissection repair (ascending tube graft with valve resuspension) performed 6 months earlier with a residual type B dissection and an obvious entry tear in zone 1, extending from zone 1 to 11. The patient is now experiencing worsening renal failure. Against the advice from nephrology, CTA is performed, which shows a diminutive true lumen with all visceral vessels supplied by the true lumen. The right common iliac artery (CIA) is occluded, and the left seems severely compromised by the dissection, with impaired filling of the external iliac artery (EIA; Figures 1–5). No delayed images are available. The patient is not walking enough for us to know if claudication is present, but he denies rest pain.

Do you believe the renal failure is related to the dissection, and why?

Dr. Hughes: This is an interesting question that is not infrequent in the setting of chronic dissection with compressed distal true lumen. I think the safe (and maybe cop-out) answer is "maybe." Although there are only a few still-frame images to review, what we see of the kidneys suggests that the renal cortices are somewhat shrunken, consistent with chronic hypertension and preexisting chronic kidney disease, which may now simply be getting worse in the setting of recent major surgery and new multiple antihypertensive medications.

However, the left kidney has a lateral filling defect that may represent an area of infarction, possibly related to the original dissection event 6 months earlier, and may implicate some degree of chronic malperfusion contributing to the renal dysfunction. If the patient has difficult-to-control hypertension requiring multiple agents, this would suggest chronic renal malperfusion and implicate the chronic dissection with compressed distal true lumen contributing to the patient's worsening renal function.

Dr. Beck: Although it's difficult to tell for sure with the imaging provided, I would absolutely say that the renal failure has a high probability of being related to the dissection given the collapse of the true lumen at the level of the renal arteries.

Dr. Schneider: Yes, the CTA images show anatomy consistent with visceral and lower extremity malperfusion due to marked true lumen compression. In the absence of any other identifiable causes, the renal failure is most likely being caused by renal hypoperfusion due to the true lumen compression.

An entry tear is noticed in zone 1. Is there a role for thoracic endovascular aneurysm repair (TEVAR)? If so, how would you manage the dissected arch?

Dr. Beck: If it were possible, in the ideal setting, the ultimate goal would be depressurization of the false lumen, allowing true lumen expansion and perfusion of the branch vessels arising from the true lumen in the visceral segment. If this patient's false lumen pressurization and true lumen collapse is indeed related to the entry tear in the ascending aorta, this presents a challenge to the use of an endograft to achieve the goal of false lumen exclusion.

There is a role for TEVAR in very select patients with previous ascending arch reconstruction and nonaneurysmal

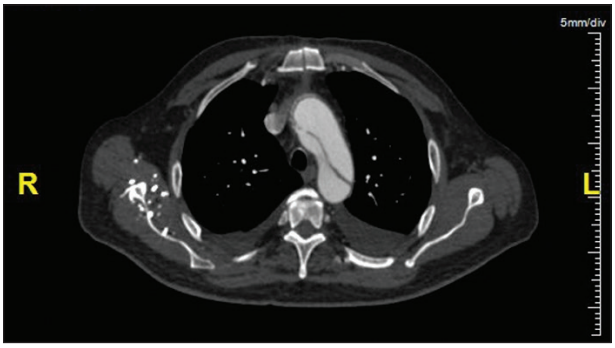

Figure 1. Axial imaging of residual arch involvement with an entry tear at zone 1.

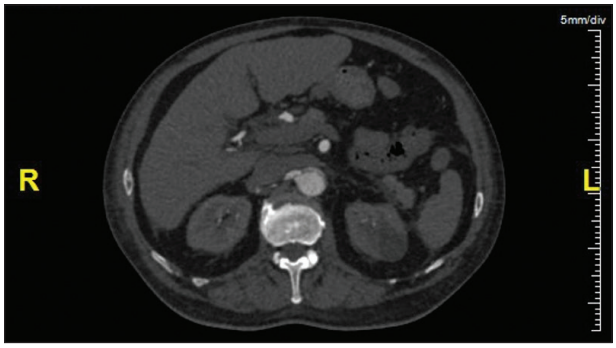

Figure 2. Axial imaging with a severely compressed true lumen and dynamic obstruction of renal arteries.

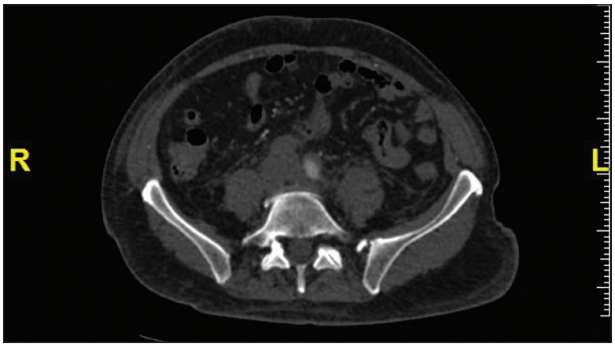

Figure 3. Axial imaging revealing an occluded right CIA and impaired filling of left iliac artery.

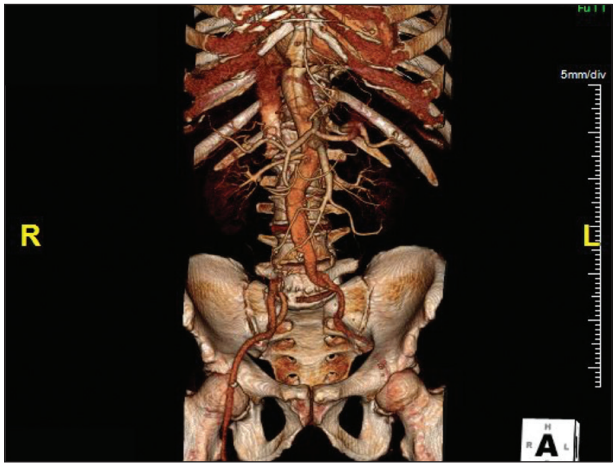

Figure 4. Three-dimensional rendering demonstrating an occluded right CIA and impaired left EIA.

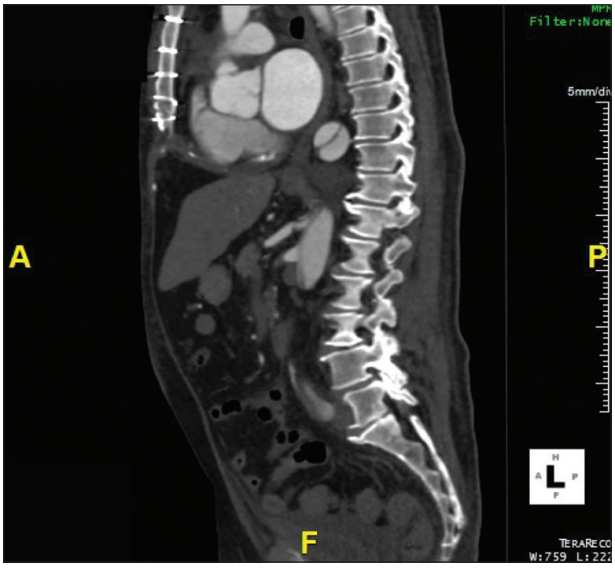

Figure 5. Sagittal imaging revealing severe dynamic obstruction of the visceral segment from residual dissection process.

proximal descending aorta that might allow for TEVAR in the true lumen with balloon expansion of the stent/septum to occlude false lumen flow. Other options would be branched devices that would allow arch deployment/coverage of the entry tear in patients with appropriate anatomy, as well as TEVAR performed in the setting of open arch debranching with subsequent or concomitant stent graft placement.

Given that this patient's arch is not currently aneurysmal, I do not think it is imperative to treat that segment of his aorta if flow to his visceral segment/renal arteries/legs can be established by other means. The only caveat to this is that I would take care to prevent future difficulties with arch reconstruction with whatever treatment was chosen for the current problem.

Dr. Schneider: TEVAR is one possible treatment option to improve flow into the true lumen and decrease flow into the false lumen flow by covering the proximal entry tear. Because the tear is in zone 1, the stent graft would have to cover the origins of the left common carotid artery and the left subclavian artery, assuming common arch anatomy. This would require arch debranching with an extra-anatomic carotid-carotid-subclavian bypass with proximal ligation or

embolization of the left common carotid and left subclavian arteries to prevent retrograde perfusion of the false lumen. If the stent graft must land into zone 0, then total arch debranching via a redo sternotomy would be necessary to maintain brachiocephalic perfusion or an arch branched endograft could be used (although these are not yet commercially available in the United States).

Of course, TEVAR is only one of several possible approaches (as discussed later) to improve true lumen perfusion to the visceral aorta and lower extremities. It is also important to consider that the risk of complications may be higher because this case requires zone 1 or zone 0 coverage. It does not appear that there is aneurysmal dilation of the arch or the thoracic aorta based on the available images, so a thoracic stent graft may not be needed at this time. Moreover, although TEVAR has been shown to be effective for the treatment of malperfusion in acute and subacute dissections, this patient has a chronic dissection and achieving true lumen reexpansion with TEVAR may require additional adjunctive procedures.

Dr. Hughes: Based on the images provided, the patient does not yet appear to have developed aneurysmal dilation of the distal chronically dissected aorta, which would be the far more common reason for reintervention after index type A dissection repair. However, if one is convinced that the worsening renal function is secondary to chronic malperfusion, there may be a role for endovascular repair to exclude the zone 1 reentry tear (this does not appear large enough to have been the primary entry tear for the index type A dissection) and expand the compressed true lumen distally with a secondary improvement in renal blood flow. The endovascular options in this case (at least in the United States) would be an investigational branched arch endograft or a "hybrid" approach to include arch debranching (zone 1 vs zone 0) to create a proximal landing zone for endovascular repair. This latter option especially is predicated on the ascending Dacron graft used at the index repair being of suitable length to serve as an adequate proximal landing zone. As our group has previously published,[1] this generally requires approximately a 4-cm length of Dacron as compared to the generally accepted 2 cm of native aorta, and this length may not be present in cases where a concomitant hemiarch replacement was not performed at the time of the original type A dissection repair. Based on our early experience in clinical trials, it is likely that the branched endografts will require closer to the 2 cm or so required for seal in the native aorta.

If a suitable length of Dacron landing zone does not exist and the patient cannot be treated with a branched endograft, another option is redo sternotomy for total arch replacement to create a long-segment Dacron proximal landing zone followed by second-stage endovascular repair (type II or III hybrid arch repair), another technique we have used extensively in the setting of previous type A dissection repair.[2] However, this option is typically used in late aneurysmal dilation involving the arch and proximal descending thoracic aorta after index type A repair, rather than the clinically ambiguous situation in the case presented.

Finally, another option we have used on occasion for patients who are unsuitable for any of the previously mentioned options (typically due to anatomy not amenable to hybrid repair and being medically unfit for redo open surgery) is a hybrid option involving cervical arch debranching (right common carotid–left common carotid–left subclavian artery bypass) with subsequent zone 0 TEVAR including an innominate artery "snorkel" endograft to maintain inflow to the brain.

The patient is 6 months postrepair with a diminutive and narrowed true lumen. How would you reestablish flow to the kidneys?

Dr. Beck: Endovascular options that I would consider include TEVAR, as I previously outlined, if he was an anatomic candidate; septal fenestration at the visceral segment to provide false to true lumen flow; placement of a bare dissection stent through the visceral segment to force open the true lumen; or, in very select patients, placement of a septal occluder to attempt closure of the small entry tear in the ascending aorta. Also, open aortic reconstruction with open fenestration of the dissection septum in the visceral segment along with an aorto-bi-iliac reconstruction would be high on my algorithm in a patient who is a good candidate for open repair.

My treatment choice would likely be septal fenestration, which I would do along with concomitant pressure measurements in the true lumen and branch vessels to demonstrate improved flow to the end organs/legs. If this was not successful and the patient was a cardiopulmonary/physiologic candidate, I would opt for an open aortic reconstruction, as described.

Dr. Hughes: As previously described, the endovascular options mainly involve excluding the arch and descending thoracic reentry tears with endografts, with a goal of reexpanding the true lumen. In this scenario, we would generally pave down to the level of the celiac axis. It is important to note that in the chronic setting, such as the one presented, the true lumen is unlikely to fully reexpand due to fibrosis and thickening of the dissection membrane over time[3]; however, in our experience, the true lumen will continue to slowly enlarge after TEVAR due to the chronic

radial forces of the endograft within the true lumen. An important caveat is that the distal endograft should not be excessively oversized so as to avoid creating a distal stent graft–induced reentry tear would lead to continued false lumen pressurization.

Another consideration would be whether to extend the degree of pavement further distally beyond the visceral segment using bare-metal stents (BMSs), with or without intentional balloon septal fenestration (STABLE and STABILISE techniques). Finally, as the renal arteries do not look narrowed on the images presented, we would not initially plan renal artery branch stenting but rather would assess the results of true lumen expansion with endografts before progressing to other options.

Open thoracoabdominal repair is an option in this scenario, although it is typically performed for aneurysmal dilation rather than chronic malperfusion. For open repairs involving the visceral segment, we reimplant the renal arteries as individual branches using a multibranch Dacron graft, thereby eliminating the possibility of residual malperfusion.

Dr. Schneider: The patient's current main clinical problem is renal (and possibly also mesenteric) malperfusion due to true lumen compression. TEVAR alone may solve this problem by improving true lumen perfusion, but in a chronic dissection, the need for other adjuncts is likely. One possibility is the addition of BMSs distal to the TEVAR stent graft that can be extended through the visceral abdominal aorta. Lombardi et al reported that the addition of the distal BMS can induce positive true lumen remodeling in acute and subacute dissections.[4] The STABILISE technique takes this a step further, using aggressive balloon dilation after placement of distal BMS to reestablish a single aortic lumen.[5] In this case, all the visceral arteries originate from the true lumen, so the likelihood of needing additional renal or mesenteric artery stents may be lower.

An alternative approach to TEVAR with or without distal BMSs is fenestration to create a large communication between the true and false lumens in the visceral aorta to equalize the pressures between the true and the false lumens, thereby improving true lumen flow. My preferred approach in this case would be to intentionally create a long tear in the septum in the abdominal aorta using a through wire passing from true lumen to false lumen. The key to this "cheese wire" technique is gaining access between the true and the false lumens at the level of the visceral aorta.[6] If a small fenestration is already present in the abdominal aorta, it can usually be identified by CTA or intravascular ultrasound (IVUS) and can be used for passing a wire between the true and false lumens. If there is no fenestration in the visceral aorta, then one can be created using a stiff wire or a reentry device. After through wire access is obtained across the septum, the wire is pulled downward to tear the septum. Use of IVUS guidance and maintaining additional wire access from each groin into the aorta are essential because the torn septum may prolapse or embolize distally, obstructing the infrarenal aorta. This is easily corrected if wire access has been maintained by placing stents or a bifurcated infrarenal endograft. The benefit of this approach, which is an endovascular version of a traditional open surgical visceral aortic septostomy, is that perfusion to the visceral aorta and lower extremities is restored without the need for arch debranching and TEVAR.

The iliac arteries look like a potential access/right lower extremity perfusion problem. How would you manage this complication of the dissection?

Dr. Hughes: The chronically malperfused right side will access the true lumen, and we would initially attempt access from this side using a hydrophilic wire to navigate the area of marked true lumen compression. This technique is successful in many cases. Alternatively, one could access the left side and navigate into the true lumen from below. Another option that is more useful in type B dissection, where the arch is not dissected rather than the presented chronic type A case with a dissected arch, is to pass a wire down from above (typically via right brachial access) and snare it out the groin, thereby securing through-and-through true lumen access.

Regardless of the approach, we have found the use of IVUS invaluable in this setting, as one can use the IVUS probe to precisely locate where a wire may pass from the true to false lumen and subsequently redirect it into the desired true lumen. Using IVUS, we can nearly always access the true lumen via a femoral approach.

With regard specifically to the right common iliac chronic malperfusion, if placing the thoracic endografts with or without distal stenting did not resolve the iliac malperfusion, we would proceed to stent the iliacs. Rarely, patients have required femoral-femoral bypass if a totally endovascular approach was not feasible.

Dr. Schneider: Percutaneous access to the true lumen in both extremities should be relatively simple because the dissection does not appear to extend into the common femoral arteries. The challenge after accessing the true lumen at in the common femoral arteries is then to establish wire access into the aortic true lumen. Oftentimes, this is surprisingly easy. But if not, IVUS can be useful to help guide the wire up the true lumen or to assist with a fenestration technique to cross the septum in the iliac arteries or at the aortic bifurcation. Stents or an infrarenal endograft (as previously mentioned) could then be used to restore normal lower extremity perfusion. If access can only be obtained

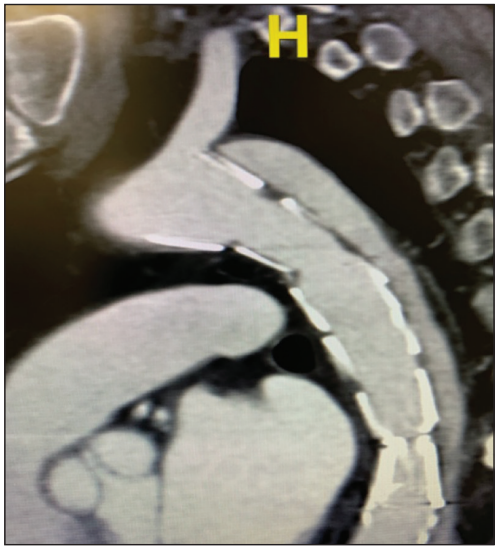

Figure 6. Sagittal imaging revealing placement of endograft, sparing the left subclavian artery.

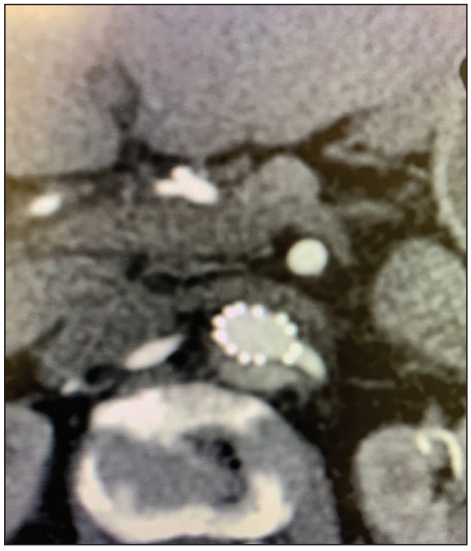

Figure 7. Axial imaging showing reperfusion of the renal arteries with a BMS in place.

from one side, then a femoral-femoral bypass could be performed to correct perfusion to the right lower extremity. Identifiable entry or reentry tears in the iliac arteries also can be used for access to perform false lumen embolization and can be closed using covered stents.

Dr. Beck: After endovascular septal fenestration in the visceral segment, I would obtain an aortogram to determine if reestablishing flow to the visceral vessels also reestablished flow to the legs. If it only successfully established flow to one leg, I would consider stenting the contralateral side if the patient's dissection anatomy allowed or perform a femoral-femoral bypass. If it did not establish flow to either leg, I would opt for an open aortic reconstruction at a separate setting, as long as the patient's condition allowed. In a prohibitively high-risk patient, an axillofemoral bypass would also be an option in the setting of persistent lower extremity malperfusion.

DISCUSSION AND APPROACH OF THE MODERATOR

This patient had worsening renal failure that began after central decompression. Although the etiology, as mentioned by Dr. Hughes, could rationally be attributed to his chronic hypertensive state worsened by the insult of the dissection and subsequent central decompression, the window of opportunity for reversal of an anatomic malperfusion state is finite.

The well-thought-out proposals by Drs. Beck, Schneider, and Hughes—such as arch debranching, single-branch thoracic device, and cervical debranching—all adhere to a staple of type B aortic dissection management: primary entry tear coverage to decompress the false lumen and perhaps restore flow to malperfused beds. Other suggested adjuncts and procedures, such as BMS placement in the aorta and endovascular septal fenestration, focus more directly on the issue of reversing ongoing malperfusion.

The decision-making in this case resides on balancing the risk of any procedure to restore renal function with the understanding that it may not be therapeutic. Therefore, minimizing procedural risk while maximizing renal perfusion was paramount. We elected to perform a straightforward PETTICOAT technique that ignores our first principle of primary entry tear coverage. We placed a thoracic endograft with our proximal seal, starting in the obvious dissected aorta distal to the left subclavian artery in zone 3 (Figure 6) and extending with a BMS (the PETTICOAT technique) across the renal arteries to the midabdominal aorta. One may argue, "Why not just place the BMS alone?" We took advantage of the fact that the ascending aorta was repaired (meaning there was no possibility of retrograde dissection) and used TEVAR (15 cm of coverage) to improve the thoracic aortic diameter, cover any thoracic septal fenestrations, and minimize flap mobility to create a more static environment to the adjacent false lumen. We also did not want

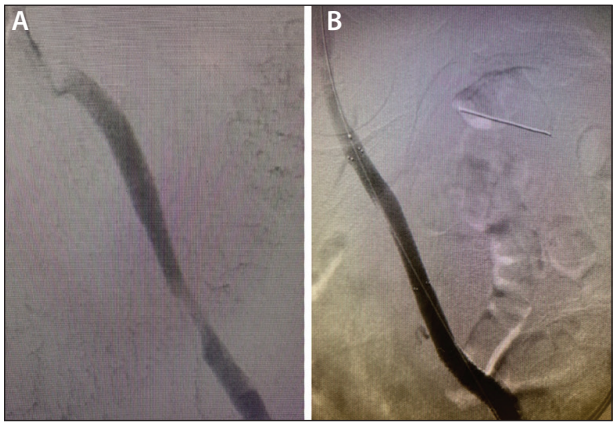

Figure 8. Arteriograms demonstrating continued malperfusion of the left iliac arteries (A) and reperfusion of the left iliac arteries after stent placement (B).

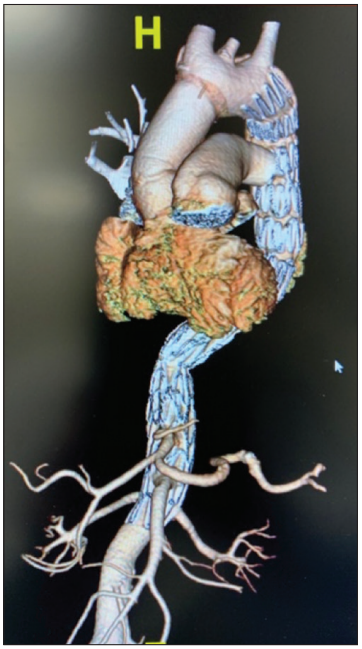

Figure 9. Three-dimensional reconstruction demonstrating the completed thoracoabdominal segment managed after the PETTICOAT technique for residual type B dissection.

the BMS "freely floating," unanchored to a proximal endograft. The BMS nicely transitions the endograft to the remainder of the diminutive and collapsed true lumen, preventing a stent graft–induced entry tear and maximizing the diameter in the remaining aorta. The dynamic obstruction caused by the mobile septum was then alleviated (Figure 7). The proximal aorta remains with persistent false lumen flow and, in the future, if the arch or descending thoracic aorta becomes aneurysmal, both open surgical and endovascular techniques can still be employed for management.

The left iliac occlusion was reevaluated and was found to have evidence of continued dynamic obstruction in the left CIA and EIA. As Dr. Schneider mentioned, these radiographic occlusions are fairly easily traversed and are usually not "thrombosed" as they would appear on CTA. Contrast timing plays a role, as well as the degree of dynamic intimal obstruction influenced by the initial unabated false lumen flow. In our case, the patient had continued left iliac stenosis; a balloon-expandable covered stent was placed in the left CIA, along with a self-expanding stent in the EIA (Figure 8).

The patient's renal function was restored to normal rather quickly and he returned to work with no growth in the arch on follow-up CTA (Figure 9). We will follow up every 6 months to 1 year with gated CTA to monitor for late growth. ■

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Joseph Lombardi, MD
Professor and Chief of Vascular and Endovascular Surgery
Director of the Cooper Aortic Center
Cooper University Health Care
Camden, New Jersey
lombardi-joseph@cooperhealth.edu
Disclosures: Consultant to Cook Medical and Endologix, Inc.

Adam Beck, MD
Holt A. McDowell Jr, MD, Endowed Chair of Vascular Surgery
Division Director and Associate Professor of Surgery
Division of Vascular Surgery and Endovascular Therapy
University of Alabama at Birmingham
Birmingham, Alabama
awbeck@uabmc.edu
Disclosures: Consultant to Cook Medical, Gore & Associates, and Terumo Aortic; receives grant support from Cook Medical, Gore & Associates, and Medtronic. All proceeds to University of Alabama at Birmingham.

G. Chad Hughes, MD
Director, Duke Center for Aortic Disease
Surgical Director, Duke Center for Structural Heart Disease
Associate Professor, Division of Cardiovascular and Thoracic Surgery
Duke University Medical Center
Durham, North Carolina
gchad.hughes@duke.edu
Disclosures: None.

Darren Schneider, MD
Associate Professor of Surgery
Weill Cornell Medical College
Chief, Vascular and Endovascular Surgery
Weill Cornell Medicine
NewYork-Presbyterian Hospital
New York, New York
dbs9003@med.cornell.edu
Disclosures: Consultant to Gore & Associates and Medtronic; receives contracted research grants from Cook Medical, Gore & Associates, and Endologix, Inc.