Management of acute type B aortic dissection with malperfusion via endovascular fenestration/stenting

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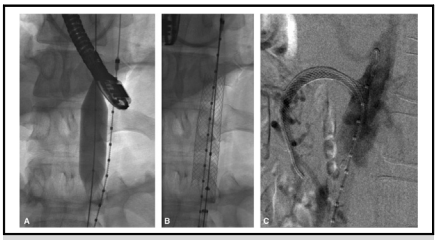
ABSTRACT

Objective: The study objective was to evaluate the management of malperfusion in acute type B aortic dissection with endovascular fenestration/stenting.

Methods: From 1996 to 2018, 182 patients with an acute type B aortic dissection underwent fenestration/stenting for suspected malperfusion based on imaging, clinical manifestations, and laboratory findings. Data were obtained from medical record review and the National Death Index database.

Results: The median age of patients was 55 years. Signs of malperfusion included abdominal pain (61%), lower-extremity weakness (27%), nonpalpable lower-extremity pulses (24%), and abnormal lactate, creatinine, liver enzymes, and creatine kinase levels. Confirmed hemodynamically significant malperfusion affected the spinal cord (2.7%), celiac (24%), superior mesenteric (40%), renal (51%), and iliofemoral (43%) arterial distributions. Of the 182 patients, 99 (54%) underwent aortic fenestration/stenting, 108 (59%) had 1 or multi-branch vessel fenestration/stenting, 5 (2.7%) had concomitant thoracic endovascular aortic repair, 17 (9.3%) had additional thrombolysis or thromboembolectomy, and 48 (26%) received no intervention. After fenestration/stenting, 24 patients (13%) required additional procedures for necrotic bowel or limb and 9 patients (4.9%) had subsequent aortic repair (thoracic endovascular aortic repair, open repair) before discharge. The new-onset paraplegia was 0%. The in-hospital mortality was 7.7% over 20+ years and 0% in the last 8 years. The 5- and 10-year survivals were 72% and 49%, respectively. The significant risk factors for late mortality were age and acute paralysis (hazard ratio, 3.5; both $P < .0001$). Given death as a competing factor, the 5- and 10-year cumulative incidence of reintervention was 21% and 31% for distal aortic pathology, respectively.

Conclusions: Patients with acute type B aortic dissection with malperfusion can be managed with endovascular fenestration/stenting with excellent short- and long-term outcomes. This approach is particularly helpful to patients with static malperfusion of aortic branch vessels. (J Thorac Cardiovasc Surg 2020;160:1151-61)



A, Aortic flap balloon fenestration. B, Thoracic aortic true lumen. C, SMA stenting.

Central Message

Endovascular fenestration/stenting can effectively resolve dynamic and static malperfusion in ATBAD with favorable short- and long-term outcomes (survival and reoperation).

Perspective

Endovascular fenestration/stenting effectively and timely resolves dynamic and static malperfusion in ATBAD with minimal risk of paraplegia and retrograde type A dissection, and excellent in-hospital mortality, cumulative incidence of reintervention, and long-term survival in this patient population as combined with TEVAR or open repair when indicated.

See Commentaries on pages 1162 and 1164.

Malperfusion, a feared complication of aortic dissection, is present in approximately 20% of type B aortic dissections[1,2] and is a risk factor for mortality[2] (mortality ranging from 17% to 64%).[2-5] An International Registry of Acute Aortic Dissection study showed 28% mortality in acute type B aortic dissection (ATBAD) with malperfusion compared with 9.6% in those without malperfusion.[2] Malperfusion is defined as inadequate

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Abbreviations and Acronyms

ATAAD = acute type A aortic dissection
ATBAD = acute type B aortic dissection
CI = confidence interval
CT = computed tomography
HR = hazard ratio
IR = interventional radiology
IVUS = intravascular ultrasound
MPS = malperfusion syndrome
OR = odds ratio
SMA = superior mesenteric artery
TEVAR = thoracic endovascular aortic repair

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flow to a vascular territory, whereas malperfusion syndrome (MPS) is decreased flow to a vascular territory resulting in tissue/organ necrosis and end-organ dysfunction, both because of dissection-related obstruction of the aorta and its branch vessels. Patients with malperfusion more often undergo thoracic endovascular aortic repair (TEVAR) than open repair[6-8] to alleviate the end-organ ischemia. However, TEVAR in ATBAD cannot reliably resolve static malperfusion of aortic branch vessels, which results from thrombosis of the false lumen and compression of the true lumen of the aortic branch vessels, has the potential risk of acute paraplegia due to false lumen thrombosis of the descending thoracic aorta and its intercostal arteries when the entire descending thoracic aorta is covered, and carries approximately a 2% to 5%[9,10] risk of retrograde type A dissection.

Since 1996, we have adopted the approach of endovascular reperfusion via fenestration/stenting by interventional radiology (IR) of the critically malperfused organ systems for patients with ATBAD and malperfusion, which can resolve both dynamic and static malperfusion of aortic branch vessels. Previously we reported our 10-year experience of treating malperfusion in ATBAD.[11] In this study, we report the short- and long-term outcomes of endovascular fenestration/stenting to treat malperfusion in ATBAD over the past 20 years.

MATERIALS AND METHODS

This study was approved by the Institutional Review Board at the University of Michigan, Michigan Medicine (Ann Arbor, Mich).

Study Population

From February 1996 to May 2018, 182 patients presented with spontaneous ATBAD and suspected malperfusion and proceeded to angiography for diagnosis and fenestration/stenting. Patients with ATBAD and malperfusion due to trauma (n = 4) or malperfusion treated with isolated TEVAR (n = 8) or open surgical repair were excluded. ATBAD was defined as onset within 14 days of admission with dissection confined to the aorta distal to the left subclavian artery. Investigators used medical record review to obtain preprocedural, intraprocedural, and postprocedural characteristics. Reinterventions included open or endovascular aortic repair of the aorta distal to the left subclavian artery and were collected from a thorough medical record review. The National Death Index database through December 31, 2015,[12] and medical record review were used to determine survival. Loss of follow-up was treated as a censor during the time-to-event analysis.

Diagnosis of Dynamic and Static Malperfusion

Malperfusion, inadequate blood flow to the end organs, could be diagnosed with radiographic findings consistent with reduced or absent flow to an end-organ or complete true lumen collapse on computed tomography, including disappearance of the aortic double lumen indicating elimination of the true lumen with the dissection flap being pushed against the aortic wall causing obstruction of flow to branch vessels, continuation of dual lumen patency with absent flow in a branch vessel (dynamic malperfusion), and dissection into a branch vessel or a thrombosed false lumen in a branch vessel (static malperfusion), with or without clinical evidence of end-organ dysfunction (Figure 1). MPS involves tissue/organ necrosis and end-organ dysfunction as a result of inadequate blood flow (malperfusion) and requires clinical features (abdominal pain, bloody diarrhea, tenderness to palpation, decreased urine output, absence of peripheral pulses, motor or sensory deficit of the lower extremity) and laboratory findings (elevated lactate, liver enzymes, metabolic acidosis, elevated creatinine) in addition to radiographic findings. The etiology of the malperfusion can be static, dynamic, or both static and dynamic obstruction of a branch vessel.[13] Dynamic malperfusion results from the dissection flap of a collapsed true lumen prolapsing across the origin of the branch vessel and obstructing flow and can vary in severity depending on variations of pressure in the false lumen. Dynamic obstruction can usually be resolved with restoration of the true lumen with a TEVAR endograft covering the intimal tear or aortic fenestration/stenting. Static obstruction results from extension of the dissection flap into a branch vessel, frequently accompanied by false lumen thrombosis due to no or very small reentry tear and occlusion of the true lumen, and is present throughout the cardiac cycle. Total occlusion of a vessel, like the superior mesenteric artery (SMA), by a thrombosed false lumen can lead to thrombosis of the true lumen distal to the dissection. Furthermore, collateral flow to an obstructed SMA is often compromised by dissection-related compromise of the celiac trunk or inferior mesenteric artery. Static obstruction usually requires stenting or other intervention (fenestration/thromboembolectomy/thrombolysis) of the affected branch vessel to restore flow. Both static and dynamic obstruction can be resolved with endovascular reperfusion via fenestration/stenting. In contrast to our management in type A aortic dissection, where MPS is the indication for IR procedures, in type B aortic dissection suspected malperfusion (not MPS) is an indication for IR.

Endovascular Techniques

Angiography was completed a median of 1 day after hospital admission. The angiographic evaluation of malperfusion has been described in detail.[13-16] In angiography, treatable malperfusion was indicated by ongoing arterial obstruction and was confirmed by a systolic blood pressure gradient greater than 15 mm Hg between the ascending aorta and the branch vessel. If a branch artery is dissected, branch artery manometry is performed distal to the dissection (confirmed by intravascular ultrasound [IVUS]). The gradient of 15 mm Hg was chosen

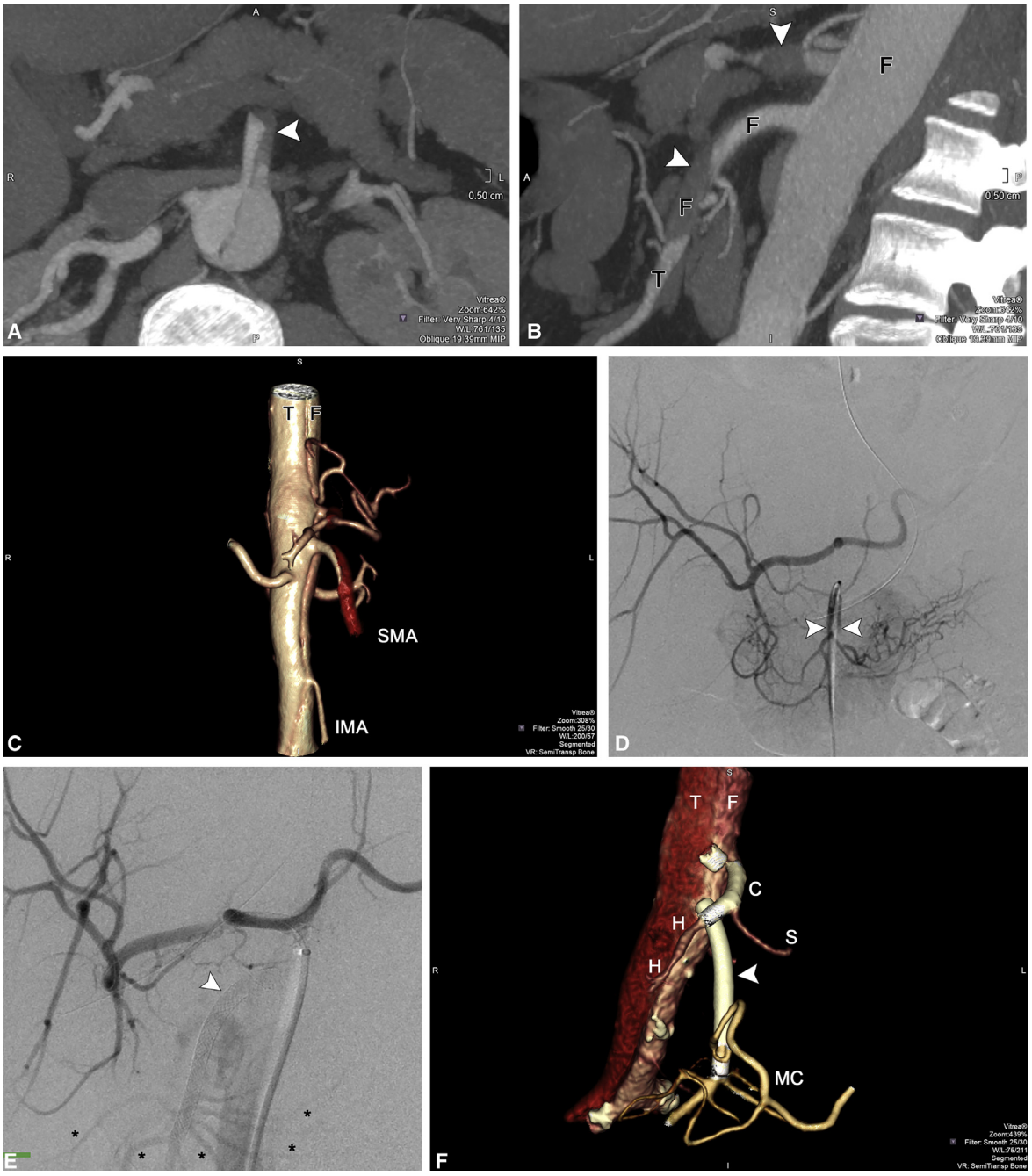


FIGURE 1. Computed tomography (CT) angiogram of a 55-year-old man with an ATBAD and static malperfusion of the celiac artery and SMA before and 8 years after endovascular fenestration/stenting. Axial CT at the level of the SMA (A) shows thrombosed false lumen (*arrowhead*) just beyond the SMA origin. Sagittal CT of the upper abdomen through the false lumen of the aorta (B) again shows thrombus in the terminal portion of the false lumen of the celiac trunk and the SMA, resulting in arterial occlusion. Three-dimensional reconstruction of the abdominal aorta (C) shows the dissection flap cleaving the celiac and SMA origins with thrombosed false lumen (*red*), with the inferior mesenteric artery (IMA) supplied by the false lumen. Superior mesenteric arteriogram (D), approximately 28 hours after symptom onset, shows proximal occlusion of the SMA trunk (*arrowheads*), absent filling of jejunal and ileal branches, and retrograde filling of the celiac distribution through pancreatic collaterals. After stenting of the SMA and celiac trunk, distal SMA pressure was 57/43 mm Hg, 32 mm Hg lower than aortic true lumen pressure. Sheath injection at the celiac origin after stenting (E) fills hepatic and splenic arteries and refluxes into the abdominal aorta, filling stented SMA (*arrowhead*) with jejunal and ileal branches (*asterisks*). CT with 3-dimensional reconstruction 8 years later (F) shows the celiac artery (*C*) stent extending into the common hepatic artery (*H*) with jailed but patent splenic artery (*S*). The SMA stent (*arrowhead*) is patent down to the ileocolic artery. Several small jejunal and ileal branches continue to fill through stent interstices. A prominent IMA (not shown) supports flow through the middle colic (*MC*) artery. *F*, False lumen; *T*, true lumen; *SMA*, superior mesenteric artery; *IMA*, inferior mesenteric artery; *H*, hepatic artery; *S*, splenic artery.

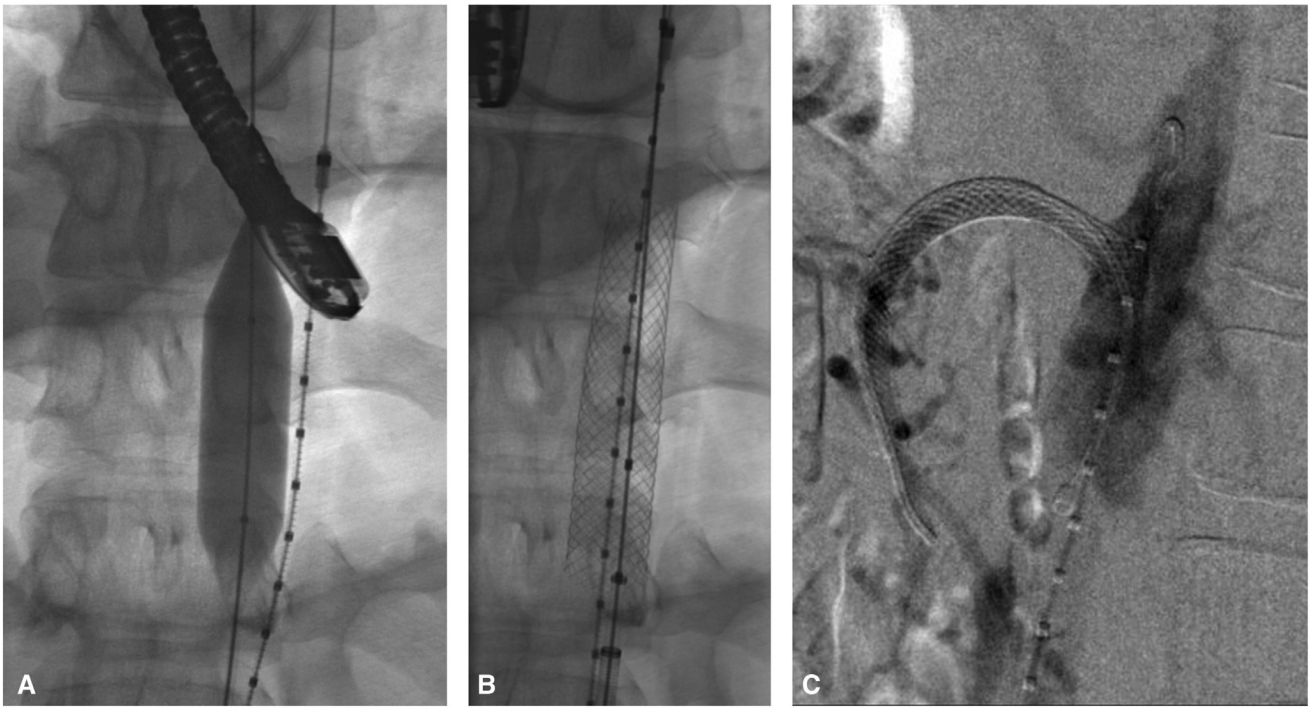


FIGURE 2. A, Aortic flap balloon fenestration. B, Thoracic aortic true lumen. C, SMA stenting.

as the criterion for malperfusion based on the customary acceptance of a blood pressure differential of greater than 20 mm Hg as indicating hemodynamic significance in patients with aortic coarctation.[17] Fenestration and stenting were performed by creating a tear in the dissection flap 2 to 4 cm above the celiac artery using a 16-mm diameter balloon, thereby permitting flow between the true and false lumens, followed by deployment of a 16- to 18-mm diameter closed-cell self-expanding stent (Wallstent, Boston Scientific Corporation, Marlborough, Mass, off-label application) exclusively in the aortic true lumen (Figure 2), as previously described,[13,16,18,19] if the true lumen remains collapsed or a gradient between the aortic root and the abdominal aorta persists after aortic fenestration. Blood pressure gradients between the aorta and the branch vessels were determined both before and after fenestration/stenting (Wallstents). If after aortic fenestration/stenting a significant gradient persisted between the aorta and a branch vessel, then branch vessel fenestration/stenting, thrombolysis, or thromboembolectomy was performed as appropriate, based on angiographic and IVUS findings (Figure 2). Complete resolution of malperfusion was defined as blood pressure gradient decreased to less than 15 mm Hg. In dissected vessels with thrombosed false lumens, gradients after stenting might exceed 15 mm Hg, defined as partial resolution of malperfusion, but as long as absolute perfusion pressure was viable (ie, >60 mm Hg), postdilation of stents was not performed.

Concomitant (n = 5) or post-IR TEVAR (n = 4) or open aortic repair (n = 5) was indicated for pending rupture, refractory back pain, uncontrollable severe hypertension, and aortic aneurysm. Concomitant TEVAR includes patients who initially had TEVAR for back pain/impending rupture with persistent postoperative static malperfusion subsequently treated by fenestration/stenting. All open procedures were performed before 2004. Postprocedure management consisted of aspirin therapy, blood pressure control, standard management of end-organ dysfunction, and adequate analgesia and sedation. When bowel ischemia or extremity ischemia was present, general or vascular surgery was consulted, respectively, to determine if exploratory laparotomy or fasciotomies were indicated.

Statistical Analysis

Continuous variables were summarized by median (25%, 75%), and categoric variables were reported as n (%) in frequency tables. Crude survival curves since admission were estimated using the nonparametric Kaplan–Meier method. Multivariable logistic regression was performed to calculate the odds ratio (OR) of risk factors for in-hospital mortality adjusting for age, gender, coronary artery disease, acute myocardial infarction, acute renal failure on dialysis, acute paralysis, celiac malperfusion, mesenteric malperfusion, renal malperfusion, and extremity malperfusion. Cox proportional hazard regression was performed to calculate the hazard ratio (HR) for late mortality by stepwise selection of variables including age, gender, coronary artery disease, chronic renal failure on dialysis, acute myocardial infarction, acute paralysis, acute renal failure requiring dialysis, MPS found, bowel resection, amputation, and fasciotomy. Because patients may experience death before reintervention was indicated, cumulative incidence curves adjusting for death as the competing risk were generated to assess cumulative incidence of reintervention over time. Cox regression was used to calculate the risk factors of reintervention adjusting for age, gender, connective tissue disease, aortic flap fenestration without TEVAR or open aortic repair, and hypertension. All statistical calculations used SAS 9.4 (SAS Institute, Inc, Cary, NC).

RESULTS

Demographics and Preprocedural Data

The median age was 55 years, and most patients (88%) had hypertension. The majority (93%) of patients were transferred from another hospital. Patients frequently

TABLE 1. Demographics and characteristics before procedures by interventional radiology

Variables	Total (n = 182)
Patient age (y)	55 (48, 65)
Sex (male)	139 (76)
BMI	30 (26, 33.5)
Hypertension	160 (88)
Hyperlipidemia	47 (26)
Diabetes	15 (8.2)
COPD	23 (13)
CAD	40 (22)
PVD	18 (9.9)
History of smoking	
None	61 (34)
Former	43 (24)
Current	78 (43)
History of MI	17 (9.3)
History of stroke	12 (6.6)
History of renal failure	20 (11)
On dialysis	3 (1.6)
Connective tissue disorder	12 (6.6)
Acute MI	25 (14)
Acute stroke	1 (0.5)
Worsening renal function	91 (50)
Requiring dialysis	5 (2.7)
Acute paralysis	12 (6.6)
Prior cardiac surgery	30 (16)
Prior aortic intervention	29 (16)
Location of initial admission	
University of Michigan	12 (6.6)
Outside hospital	170 (93)
Admission to IR (d)	1 (0, 2)
Presenting signs and symptoms	
Abdominal pain	111 (61)
Lower-extremity weakness	49 (27)
Lower-extremity pulses nonpalpable	43 (24)
Laboratory values	
Creatinine on admission (mg/dL)	1.2 (0.9, 1.6)
Maximum creatinine before IR (mg/dL)	1.5 (1.0, 2.1)
Maximum lactate before IR (mmol/L)	1.5 (1.0, 2.2)
Elevated AST/ALT	66 (37)
Max CK (U/L)	124 (58.5, 232)
Procedures during hospital stay before IR	
Exploratory laparotomy for suspected ischemia	0 (0)
Vascular surgery for suspected ischemia	6 (3.3)
Thrombolysis/stenting of SMA	1 (0.5)
Thrombectomy/embolectomy	2 (1.1)
Fem-fem bypass	4 (2.2)
Fasciotomy	2 (1.1)
Amputation	0 (0)
Aortic surgery	3 (1.6)
TAVR	1 (0.5)
Median sternotomy*	1 (0.5)
Open TAA	1 (0.5)

Data presented as median (25%, 75%) for continuous data and n (%) for categoric data. *BMI*, Body mass index; *COPD*, chronic obstructive pulmonary disease; *CAD*, coronary artery disease; *PVD*, peripheral vascular disease; *MI*, myocardial infarction; *IR*, interventional radiology; *AST*, aspartate aminotransferase; *ALT*, alanine aminotransferase; *CK*, creatine kinase; *SMA*, superior mesenteric artery; *TAVR*, transcatheter aortic valve replacement; *TAA*, thoracic aortic aneurysm. *ATBAD noted intraoperatively during aortic root repair.

presented with abdominal pain (61%), lower-extremity weakness (27%) with nonpalpable pulses (24%), and elevated creatinine and liver enzymes. Some already had certain vascular procedures for limb ischemia before presentation (Table 1).

Procedural Data

Renal, extremity, and mesenteric malperfusions were the most frequently suspected and confirmed malperfusions in ATBAD. Multiple vascular beds were frequently affected. Overall, 74% of patients had interventions in the angiography suite, including aortic fenestration/stenting (54%), branch vessel fenestration/stenting (59%), and thrombolysis/thrombectomy/embolectomy (9.3%) for static malperfusion (Table 2, Figure 1). Branch artery stenting was performed in the iliac, renal, superior mesenteric, and celiac arteries 56, 47, 37, and 8 times, respectively. Overall, 93% of malperfusion was completely resolved and 5% was partially resolved (Table 3). After endovascular fenestration/stenting, 13 patients required laparotomy for suspected bowel ischemia, including 9 patients who had bowel resection, and 12 patients required additional vascular procedures during the current hospital stay. Nine patients required additional TEVAR (n = 4) or open aortic repair (n = 5) because of aortic aneurysm, pending rupture, and persistent symptoms (Table 2).

Postoperative Outcomes

Postprocedural new-onset paraplegia (0%), retrograde type A dissection (0%), and new-onset acute renal failure requiring dialysis (1.6%) were low. In 5 patients with spinal cord malperfusion, 2 had complete resolution and 1 had partial resolution of paraplegia. Overall in-hospital mortality was 7.7% over 20 years, 11.3% in the first decade (1996-2007), 3.5% in the second decade (2008-2018), and 0% in the last 8 years (Table 4). The significant risk factors for in-hospital mortality were age (OR, 1.15), acute myocardial infarction (OR, 8.6), acute paralysis (OR, 11.5), and extremity malperfusion (OR, 8.8) (Table 5). Detailed

TABLE 2. Interventional radiology indications and procedures and subsequent procedures during hospital stay

Variables	Total (n = 182)
IR indications	
Malperfusion suspected	182 (100)
Spinal cord	8 (4.4)
Celiac	22 (12)
Mesenteric	81 (45)
Renal	119 (65)
Extremity	71 (39)
Malperfusion found	140 (77)
Spinal cord	5 (2.7)
Celiac	43 (24)
Mesenteric	73 (40)
Renal	93 (51)
Extremity	79 (43)
IR procedures	
No intervention in IR	48 (26)
Aortic fenestration/stenting	99 (54)
Branch vessel fenestration/stenting	108 (59)
Concomitant TEVAR	5 (2.7)
Additional procedures*	17 (9.3)
Subsequent procedures during hospital stay	
Exploratory laparotomy for suspected ischemia	13 (7.1)
Bowel resection	9 (4.9)
Vascular surgery for suspected ischemia	12 (6.6)
Thrombectomy/embolectomy	6 (3.3)
Fem-fem bypass	2 (1.1)
Fasciotomy	5 (2.7)
Amputation	3 (1.6)
Aortic surgery	9 (4.9)
TEVAR	4 (2.2)
Open TAA/A	4 (2.2)
Open AAA	1 (0.5)
Time from IR (d)	12 (9, 14)

Data presented as n (%). TEVAR was performed by both IR faculty and cardiac surgeons together. *IR*, Interventional radiology; *TEVAR*, thoracic endovascular aortic replacement; *TAA/A*, thoracic aortic aneurysm or thoracoabdominal aortic aneurysm; *AAA*, abdominal aortic aneurysm. *Thrombolysis, thrombectomy, embolectomy of aortic branch vessels.

causes of death included aortic rupture, extensive necrotic intestine, arrhythmia, and stroke (Table E1).

Long-Term Outcomes

The 5- and 10-year survivals were 72% (95% confidence interval [CI], 64-78) and 49% (95% CI, 39-58), respectively (Figure 3, *A*). The significant risk factors for late mortality were age (HR, 1.04; 95% CI, 1.02-1.06) and acute paralysis (HR, 3.5; 95% CI, 1.8-6.8; $P < .001$).

Of the 182 patients, 14 died in the hospital before discharge and 12 (6.6%) were lost to follow-up for reintervention. The mean follow-up time was 6.2 years. The 5- and 10-year cumulative incidence of reintervention for pathology of the descending or thoracoabdominal aorta adjusted for death as a competing factor was 21% (95% CI, 15-28) and 31% (95% CI, 23-40), respectively (Figure 3, *B*). The significant risk factors for reintervention were connective tissue disease (HR, 3.4; 95% CI, 1.4-8.3; $P = .007$) and male gender (HR, 3.2; 95% CI, 1.3-8.0; $P = .014$). Fenestration without TEVAR or open repair was not a significant risk factor (HR, 0.8; 95% CI, 0.4-1.5; $P = .49$). The 5- and 10-year cumulative incidence of reintervention for patients undergoing fenestration/stenting only, without TEVAR or open repair during the hospital stay, was 20% (95% CI, 13-28) and 31% (95% CI, 21.5-41), respectively (Figure 3, *C*). The primary indication for reintervention was aortic aneurysm (91%) and primarily done through open repair (76%). The median interval time to reintervention was 2 years.

DISCUSSION

In this study, we managed ATBAD complicated by malperfusion with endovascular fenestration/stenting, which enables resolution of both dynamic and static malperfusion. The in-hospital mortality was 7.7% over 20+ years and 0% in the last 8 years; the cumulative incidence of reintervention was 21% at 5 years and 31% at 10 years; and the 5- and 10-year survivals were 72% and 49%, respectively.

Aortic dissection, both type A and B, can be complicated by malperfusion due to dissection-related obstruction of aortic branch vessels. In acute type A aortic dissection (ATAAD), because of the risks of aortic rupture, acute heart failure and aortic insufficiency, acute myocardial infarction, pericardial effusion and tamponade, and neurologic complications,[20,21] we pursue upfront angiography with endovascular reperfusion only for patients with MPS (malperfusion with tissue/organ necrosis and end-organ dysfunction). In patients with ATBAD, we extend this strategy to patients with malperfusion unresponsive to blood pressure and heart rate control and patients with a documented history of poor compliance with antihypertensive medication in addition to patients with MPS. Patients with ATBAD have a lower risk of aortic rupture with adequate blood pressure management[11,22] and a small risk of the proximal aortic complications commonly seen with type A dissections.[23] Malperfusion due to dynamic obstruction, which is generally corrected by proximal aortic repair in patients with ATAAD, may persist in patients with ATBAD unless dealt with directly by fenestration/stenting or TEVAR. Malperfusion, in addition to MPS, is an indication for angiography for diagnosis and potential treatment in patients with ATBAD. Because clinical manifestation of mesenteric and renal malperfusion may lag their computed tomography demonstration and unsuspected vascular beds with malperfusion are frequently identified when we investigate malperfusion of suspected vascular beds, we consider the endovascular evaluation of patients with ATBAD an angiographic emergency and have a low threshold for performing it. The angiographic evaluation of these patients typically involves manometry, IVUS examination, and

TABLE 3. Detailed interventional radiology procedures

Level of aortic fenestration/stenting	Aortic fenestration (n = 87)	Aortic stenting (n = 89)	Branch vessel fenestration (n = 2)	Branch vessel stenting (n = 105)	Malperfusion in vascular bed (n = 182)	Malperfusion completely resolved*	Malperfusion partially resolved*
Descending thoracic	6	5	-	-	-	-	-
Supraceliac	22	12	-	-	-	-	-
Celiac	17	2	0	8	43	42	1
Supramesenteric	17	45	-	-	-	-	-
Mesenteric	15	0	0	37	73	65	8
Suprarenal	1	0	-	-	-	-	-
Renal†	7	5	0	47	93	87	2
Infrarenal	44	62	-	-	-	-	-
Iliac†	-	-	2	56	79	74	4

In columns 2 to 5, n is the number of patients. In column 6, n is the total malperfusion found in different vascular beds, including celiac artery, SMA, renal arteries, and common iliac arteries and their branches (external iliac arteries, femoral arteries). Aortic stenting: If there was a compression of the true lumen by the thrombosed false lumen in the descending aorta, we placed a 16-mm self-expanding bare stent in the descending aorta (descending thoracic aortic stenting). After fenestration of the aortic dissection flap, we place the same self-expanding bare stent in the distal descending thoracic aorta if needed to keep the true lumen open. The distal descending thoracic aortic stent was placed frequently above the SMA (supramesenteric aortic stenting). If we had to place stents in the SMA, then we placed the aortic stent above the celiac artery (supraceliac aortic stenting). The goal of aortic stenting is to achieve adequate expansion of the true lumen of dissected aorta and eliminate blood pressure gradient between the distal aorta and the ascending aorta. *Complete resolution of malperfusion was defined as the systolic blood pressure gradient between the branch vessel and the ascending aorta less than 15 mm Hg after endovascular fenestration/stenting. Partial resolution of malperfusion was defined as systolic blood pressure gradient greater than 15 mm Hg. †Among 47 patients receiving renal artery stenting, 38 (81%) received unilateral stenting (right renal, 38%; left renal, 43%) and 9 (19%) had bilateral renal artery stenting. Among 56 patients receiving iliac stenting, 42 (75%) had aorto-iliac stenting, 32 (57%) had common iliac artery stenting, 33 (59%) had external iliac artery stenting, and 7 (12.5%) had common femoral artery stenting. Four renal artery malperfusions and 1 iliac malperfusion were unable to be treated because of an inability to cannulate the specific branch vessel (n = 1, renal artery), anatomy unsuitable for stenting (n = 3, renal artery), or a significant gradient without symptoms of malperfusion (n = 1, iliac).

hand injection of 7 mL of contrast material diluted 1:1 with normal saline into the SMA, bilateral renal arteries, and external iliac arteries. In the case of SMA or renal artery dissection associated with a significant pressure deficit, IVUS examination is performed to determine radiographic landmarks of the dissection terminus to aid in stent placement.

Management options for ATBAD complicated with malperfusion have generally included open surgical repair and more recently TEVAR. Although TEVAR has reduced early mortality to approximately 10% in all ATBAD,[1,7,24-26] we still use fenestration/stenting as our mainstream treatment for malperfusion in ATBAD for the following reasons: (1) TEVAR has risks of retrograde type A dissection[27] (1.6% in large registry[10]), spinal cord ischemia, and paraplegia (2%-10%[28-30]); (2) TEVAR alone cannot reliably resolve static malperfusion; (3) TEVAR sometimes has to cover the left subclavian artery to cover the primary intimal tear, which requires additional procedures (eg, left carotid artery-subclavian artery bypass) to preserve blood flow to the left subclavian artery; and (4) when patients have necrotic bowel or limb and sepsis, TEVAR has a higher risk of graft infection. With endovascular fenestration/stenting, we can avoid all the risks from TEVAR and adequately treat static malperfusion with branch vessel stenting, fenestration, thromboembolectomy, or thrombolysis. In this study, 59% of patients had fenestration/stenting of an aortic branch vessel and 9.3% had thrombolysis or thromboembolectomy for static malperfusion that could not be resolved by TEVAR alone. Because we did not cover any intercostal arteries, protecting the spinal cord from ischemic injury, postprocedural new-onset paraplegia due to ischemic spinal cord injury was 0%, which is lower than in those treated with TEVAR alone (2%-10%[28-30]). The in-hospital mortality rate was 7.7% with this approach in this subpopulation with ATBAD and malperfusion with 0% mortality in the last 8 years as we became more experienced with fenestration/stenting and with use of TEVAR for aortic

TABLE 4. Postprocedural outcomes

Variables	Total (n = 182)
Stroke	10 (5.5)
Continued acute renal failure requiring new dialysis	14 (7.7)
New-onset renal failure	10 (5.5)
Requiring dialysis	3 (1.6)
Dialysis at discharge	2 (1.1)
New-onset paraplegia	0 (0)
Preoperative paraplegia resolved*	3 (60)
GI bleed	1 (0.5)
Groin hematoma	8 (4.4)
Length of stay (d)	11 (8, 18)
In-hospital mortality	14 (7.7)

Data presented as median (25%, 75%) for continuous data and n (%) for categoric data. *GI*, Gastrointestinal. *Five patients had preoperative paraplegia due to spinal cord malperfusion, 2 patients' paraplegia resolved completely, and 1 patient's paraplegia resolved partially.

TABLE 5. Risk factors for in-hospital mortality (multivariable logistic regression)

Variables	Odds ratio (95% CI)	*P* value
Age	1.15 (1.06-1.25)	.0009
Male gender	3.31 (0.68-16.09)	.14
CAD	0.65 (0.10-4.43)	.66
Acute MI	8.59 (1.35-54.42)	.02
Acute renal failure requiring dialysis	1.80 (0.11-28.88)	.68
Acute paralysis	11.5 (1.83-72.38)	.009
Found celiac malperfusion	1.38 (0.20-9.61)	.74
Found mesenteric malperfusion	3.16 (0.43-23.05)	.26
Found renal malperfusion	2.88 (0.58-14.28)	.19
Found extremity malperfusion	8.83 (1.43-54.78)	.02

CAD, Coronary artery disease; *MI*, myocardial infarction.

pending rupture or rupture, which is lower than that seen with open repair and with TEVAR alone,[1,6,7,24-26,31] possibly because TEVAR alone does not reliably correct static obstruction. Other reasons for improved mortality include better imaging, prompt diagnosis, treating suspected malperfusion in an acute dissection as an angiographic emergency, and better intensive care unit management (blood pressure control). Endovascular fenestration/stenting of ATBAD with malperfusion combined with TEVAR and open repair achieved favorable survival (5- and 10-year survivals: 72% and 49%, respectively), which was better than or similar to those treated with TEVAR or open repair alone.[7,8,24,25,31,32] The significant risk factors for late mortality were age and acute paralysis (HR, 3.5). By decreasing the risk of new-onset paraplegia, endovascular fenestration/stenting could decrease the late mortality.

Our approach is based on risk stratification to determine the best management. In the setting of ATBAD with malperfusion without signs of rupture (persistent or increasing back pain), we think the malperfusion is the most immediate concern and elect to treat the malperfusion with percutaneous fenestration/stenting. This approach accomplishes the goal of resolving the malperfusion and essentially "converts" a complicated ATBAD to an uncomplicated ATBAD and allows patients to recover with medical management afterward. If patients had rupture/pending rupture, refractory back pain, uncontrollable hypertension, or large aortic aneurysm, a concomitant or delayed TEVAR or open aortic repair was performed as is seen in a small portion of this cohort (n = 14, 7.7%) (Table 2). Managing ATBAD with malperfusion via fenestration/stenting does not prevent aortic rupture, as would open surgery or TEVAR. In this study, 7 patients (3.8%) possibly died of aortic rupture a median of 3 days (interquartile range, 2-4.5 days) after angiography with fenestration/stenting, which all

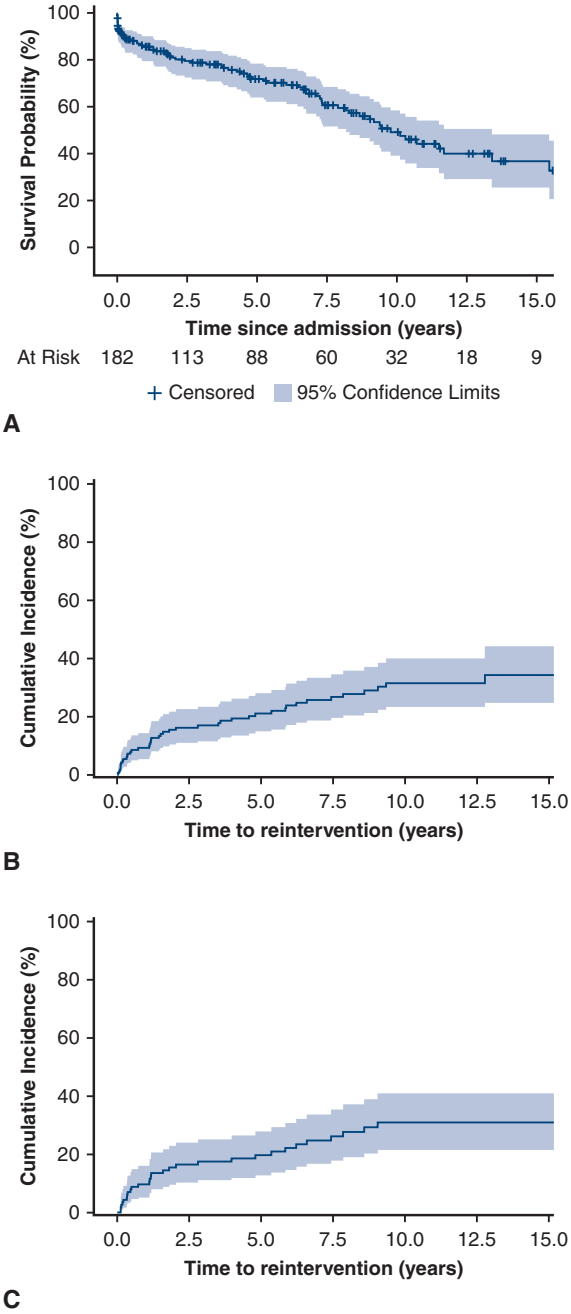


FIGURE 3. A, Kaplan–Meier survival analysis of patients with an ATBAD and MPS undergoing endovascular fenestration/stenting. The 5- and 10-year survivals were 72% (95% CI, 64-78) and 49% (95% CI, 39-58), respectively. B, The cumulative incidence of reintervention of the whole cohort (n = 182) for pathology of the descending thoracic or thoracoabdominal aorta after hospital discharge, adjusting for death as the competing event. The 5- and 10-year cumulative incidence of reintervention was 21% (95% CI, 15-28) and 31% (95% CI, 23-40), respectively. C, The cumulative incidence of reintervention for patients who had only fenestration/stenting (n = 125) after hospital discharge, adjusting for death as the competing event. The 5- and 10-year cumulative incidence of reintervention was 20% (95% CI, 13-28) and 31% (95% CI, 21.5-41), respectively.

happened before 2011 when TEVAR was not commonly used at our institution. Aortic rupture may have been prevented in these patients if they had undergone TEVAR, although cases of rupture have been reported during[24] and after TEVAR.[1,24,25,33] After 2010, we applied endovascular fenestration/stenting, combined with TEVAR and open repair as appropriate, to all patients with ATBAD and malperfusion, with a 0% aortic rupture rate and 0% in-hospital mortality. Endovascular fenestration/stenting is an effective tool to treat malperfusion (dynamic and static) in ATBAD and is a valuable adjunct to both medical and surgical therapy (TEVAR and open repair). The fenestration/stenting approach does not exclude TEVAR or open repair of the aorta. If needed, all 3 approaches can be used to treat ATBAD with different complications.

For patients with MPS (late-stage malperfusion with tissue/organ necrosis and dysfunction), endovascular fenestration/stenting resolves the malperfusion with minimal operative trauma and provides the opportunity for patients to recover from MPS. Additional intervention may be needed to recover from severe MPS. In our study, 24 patients (13%) required general or vascular surgery intervention for bowel and extremity necrosis after angiography, including bowel resection, fasciotomy, and amputations (Table 2). Of these 24 patients, 20 had branch vessel stenting during angiography for static malperfusion, which could not have been treated with open repair or TEVAR alone. This highlights the gravity of the MPS; despite initial reperfusion of affected vascular territories, patients may still have complications of the preexisting malperfusion and subsequent reperfusion. With prolonged static malperfusion, as would be with initial open repair or TEVAR, it is suspected that more patients would experience irreversible, unsalvageable end-organ death.

One concern about leaving a patent false lumen after fenestration/stenting is that it could increase the risk of reintervention. TEVAR could facilitate aortic remodeling by thrombosing and stabilizing the size of the thoracic false lumen due to closure of the primary intimal tear with a covered stent graft.[33] We think the aortic flap fenestration/stenting approach achieves the same goal by creating a distal fenestration as outflow for the false lumen to decompress and prevent the dilation of the false lumen even though the proximal primary intimal tear is open. Burris and colleagues[34] found that in chronic type B aortic dissection the false lumen dilates quickly with a large proximal primary intimal tear and a small distal reentry tear due to high pressure in the false lumen during diastole evident as regurgitant blood flow from the false into the true lumen through both proximal and distal intimal tears. If the distal reentry tear is large, there is no regurgitant flow from false to true lumen and there is minimal growth of the false lumen and the dissected aorta.[34] Our approach, endovascular fenestration of the distal aortic flap, serves exactly the same purpose. As a result, the 5- and 10-year cumulative rate of reintervention with fenestration/stenting alone was 20% and 31%, respectively, adjusting for death as a competing factor (Figure 3, *B* and *C*), which is similar if not better than that reported with TEVAR[24,32,35] and open repair[7] alone, and we had longer follow-up than most studies because TEVAR is a more recent technology. Most of the studies using TEVAR in the literature use freedom from reintervention and do not adjust for late death as a competing factor, which could underestimate the rate of reintervention. We do not think endovascular fenestration/stenting alone treating malperfusion in ATBAD increases the risk of reintervention compared with TEVAR.

Study Limitations

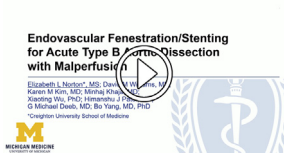
Our study is limited by a single-center and retrospective experience. The management strategy of angiography evaluation and endovascular fenestration/stenting has a learning curve. Because the follow-up of reintervention was 93.4% complete, we could underestimate the rate of reintervention. This study is also limited by lack of a direct comparison group, such as TEVAR alone.

CONCLUSIONS

In patients with ATBAD complicated by malperfusion, endovascular fenestration/stenting can effectively resolve the malperfusion and achieve favorable short- and long-term results with additional indicated TEVAR or open aortic repair. We recommend endovascular fenestration/stenting when treating ATBAD with malperfusion, especially in patients with static malperfusion.

Webcast

You can watch a Webcast of this AATS meeting presentation by going to: https://aats.blob.core.windows.net/media/19%20AM/Saturday_May4/205BD/205BD/S28%20-%20Aortic%20Dissection%20Essentials/S28_6_webcast_024733170.mp4.



Conflict of Interest Statement

Dr Williams is on the Medical Advisory Board of Boston Scientific. Drs Williams and Patel are consultants with WL Gore and Associates on an unrelated device. All other authors have nothing to disclose with regard to commercial support.

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Key Words: type B aortic dissection, malperfusion, malperfusion syndrome, endovascular fenestration/stenting

Discussion

Dr Gabriele Di Luozzo *(New York, NY)*. The Michigan group has over the years provided us with great data for the management of malperfusion in type A and B dissections. As we all agree, and we have discussed this earlier, it carries high morbidity, which also leads to high mortality. This group provides us with 1 approach for malperfusion in type A dissections.

I have a few questions. Can you explain how you determine malperfusion by computed tomography (CT) angiography and magnetic resonance angiography? Do you use imaging as the only evidence of malperfusion, do you use laboratory studies like creatinine, liver function tests, and how does that determine your treatment paradigm? You also mentioned that you use CT angiography as a way of determining reduced flow in an end organ. Can you explain that?

The second question is, you also mentioned that you use a gradient between the ascending aorta and the branch vessel

of greater than 15 mm Hg as an indicator for stenting. Can you clarify that?

Ms Elizabeth L. Norton *(Omaha, Neb)*. When evaluating malperfusion, we are looking at the whole clinical picture. Using radiographic evidence when looking at the CT, which can be seen with dynamic malperfusion, when you lose the double lumen in the aorta and you get occlusion of the true lumen, or when you see dissection extending into the branch vessels with a thrombosed false lumen, that could be static malperfusion. We are also looking at laboratory data such as liver function tests, lactate, or creatinine to evaluate organ function, and just looking at those together to get a whole picture of the malperfusion. However, in type B aortic dissection, I would say that we have a more relaxed criteria as opposed to type A where it is MPS. As we previously discussed in type B dissection, it is just malperfusion not MPS that is the criterion, and because we often find vascular beds that are not suspected to have malperfusion having malperfusion, we have a lower threshold to study this subset of patients in the angiosuite by IR.

The gradient we use is 15 mm Hg, and we established that based on aortic coarctation where a gradient of 20 mm Hg is considered significant, so we chose 15. But from what I understand it is not a rigid 15, and it depends on the patient as well.

Dr Di Luozzo. I believe most centers treat type B aortic dissection with malperfusion with sort of a top-to-bottom approach where you cover the intimal tear followed by interventions to the lower thoracic aorta or abdominal aorta, especially the branch vessels. Has the team at Michigan attempted this approach, and if so, have you looked at this subgroup of patients? If you use this type of intervention, do you need less branch vessel fenestration or stenting?

Ms Norton. At the University of Michigan, if a patient presents with acute type B and malperfusion, our first line of treatment is endovascular fenestration and stenting. However, if we have signs of pending rupture, then we will do TEVAR. In our study, we had 5 patients who had concomitant TEVAR, and they had the TEVAR first and then fenestration and stenting after for persistent malperfusion. So we don't have a comparison group.

Dr Hanni Shennib *(Phoenix, Ariz)*. I noticed that the numbers of TEVARs that you have put in there were very low; you are not putting a lot of TEVARs for this particular situation. I also saw that you had an anecdote of the notion that if you keep the entrance point when you do a fenestration, you have a lower potential for growth of the false lumen. Do you have more data to support this, because you are going sometimes against the principles of dealing with dissection?

And the last question has to do with what type of stents do you put in the mesenteric vessels. Are these covered stents or open stents?

Ms Norton. Yes, we do have a limited amount of TEVAR, because our primary approach in the setting of type B with malperfusion is endovascular fenestration and stenting, and in this study we excluded patients who did have an isolated TEVAR, because we were just looking at the endovascular fenestration and stenting.

Regarding the primary entry tear and the distal reentry tear in the false lumen to equalize the pressure, we do not have any current data on that, but Dr Yang can add more information if he has that.

Dr Bo Yang *(Ann Arbor, Mich)*. For the malperfusion in type B, we still use endovascular fenestration and stenting as the mainstream treatment. We do sometimes use TEVAR when the TEVAR is easy, which means the primary intimal tear is in the middle descending aorta and you just drop a short stent graft and cover that primary tear. If the primary tear is too proximal and surgeons have to cover the subclavian artery, then you have to do more interventions, such as left carotid artery to left subclavian artery bypass, and have an increased risk to create a retrograde type A dissection. A thrombosed false lumen after TEVAR does increase the risk of spinal cord ischemia and paraplegia. So we do not use TEVAR as a first-line approach for malperfusion in ATBAD.

Dr Shennib. What is your preferred stent for the mesenteric vessels, covered or uncovered?

Dr Yang. Uncovered.

Dr David Spielvogel. To answer the second part of your question, there are data about decompressing the false lumen. About 30 years ago, there was a series published just for that where they created a tear on the visceral segment to decompress the thoracic false lumen, and it showed that it slowed the rate of growth of the dissection. So there are data on that.

Dr Ourania Preventza *(Houston, Tex)*. Do you have any experience with the Zenith dissection system with a stent graft that is covered proximally and distally is open and potentially can help in this situation?

Ms Norton. That's a great question. Unfortunately we do not. However, the upper portion of the Zenith system is a covered stent graft. It could have the same risk as placing a regular stent graft, such as retrograde type A dissection and thrombosis of the false lumen and intercostal arteries causing spinal cord ischemia and paraplegia.

TABLE E1. Detailed cause of death in patients with in-hospital mortality

Case	Age, y	Year of treatment	Locations of malperfusion	Cause of death
1	56	1999	Celiac, mesenteric, renal	Unstable after IR, returned to cardiac care unit for continued dialysis, correction of coagulopathy, and correction of acidosis. Given grave prognosis, family decided to designate patient DNR with comfort measures.
2	53	2000	Celiac, mesenteric, renal, extremity	After bowel resection, family decided to place on comfort care.
3	51	2001	Celiac, mesenteric, renal, extremity	Second exploratory laparotomy revealed extensive necrosis - nothing to be done; no resection. Family withdrew treatment.
4	61	2002	Celiac, mesenteric, renal	Mechanical ventilation, continued dialysis for ARF, many old and new lacunar strokes, withdrawal of life support.
5*	71	2005	Extremity	Probably secondary to ruptured dissection (transferred to general care floor, reported back pain, and then coded [PEA arrest]).
6*	76	2005	Extremity	Became bradycardic, unresponsive, and no pulse was palpated after having left sternal chest pain
7*	74	2005	Renal, extremity	Evaluated for hypotension and left neck discomfort, stabilized with limited volume infusion, ~ 1 h later she suffered bradycardia and unresponsiveness and a code was carried out. Patient died.
8	67	2006	Spinal cord, celiac, mesenteric, renal, extremity	After finishing amputation but before leaving the OR, patient went into ventricular tachycardia then ventricular fibrillation with return of pulsatile rhythm and taken to surgical intensive care unit where the patient again entered a pulseless rhythm. Regained a BP for a brief period of time, and emergency hemodialysis was attempted. He again entered into a pulseless rhythm, and angiocaths were inserted. Pulse could not be regained. Patient died.
9*	85	2007	Renal	Patient suddenly became unresponsive with PEA arrest. Large amount of bloody drainage came out of the chest tube.
10*	73	2007	Mesenteric, renal, extremity	Loss of consciousness with acute decrease in BP, with subsequent PEA arrest. CPR and ACLS protocols were initiated, a stat TEE showed a large, false aortic lumen and a large, echo-free region posterior to the descending aorta, which was likely free blood due to aortic rupture. CPR was stopped; patient died.
11	71	2007	Mesenteric, renal, extremity	Ischemia of bilateral lower extremities, became hypotensive, made DNR
12*	74	2009	Spinal cord	Became unresponsive and went into PEA arrest, unable to resuscitate
13	65	2010	Celiac, mesenteric, renal, extremity	Became pulseless, resuscitated with CPR but suffered anoxic Brain injury. Little hope for meaningful recovery. Comfort care initiated.
14*	60	2010	Mesenteric, renal, extremity	PEA arrest, ACLS initiated + CPR started, unsuccessful–>Patient died

IR, Interventional radiology; *DNR*, do not resuscitate; *ARF*, acute renal failure; *PEA*, pulseless electrical activity; *OR*, operating room; *BP*, blood pressure; *CPR*, cardiopulmonary resuscitation; *ACLS*, advanced cardiac life support; *TEE*, transesophageal echocardiogram. *Death due to possible rupture.