Indications for Thoracic EndoVascular Aortic Repair (TEVAR): A Brief Review

Frank Manetta, MD^{1,2} Joshua Newman, MS² Allan Mattia, MD^{1,2}

¹Department of Cardiovascular and Thoracic Surgery, Northwell Health, Bay Shore, New York

²Zucker School of Medicine at Hofstra/Northwell, 500 Hofstra University, Hempstead, New York

Address for correspondence Frank Manetta, MD, Department of Cardiovascular and Thoracic Surgery, Southside Hospital, 301 East Main Street, 2 North, Bay Shore, NY 11706
(e-mail: fmanetta@northwell.edu).

Int J Angiol 2018;27:177–184.

Abstract

The utility of Thoracic EndoVascular Aortic Repair (TEVAR) continues to progress at a very rapid rate. Initially implemented for the treatment of thoracic aortic aneurysms, TEVAR has evolved to treat a variety of aortic pathologies and reduce overall morbidity and mortality rates compared with traditional open surgical repair.

Given the rapidly evolving nature of endovascular thoracic intervention, we hereby briefly review the current literature on the evolving applications of TEVAR.

TEVAR continues to rapidly evolve and is being applied to a growing number of aortic pathologies. Given the perioperative, short- and mid-term morbidity and mortality rates, TEVAR is quickly surpassing traditional open surgical intervention as the ideal procedure for patients undergoing intervention of the descending thoracic aorta and applicability to ascending and arch pathologies is being explored. However, as more data becomes available TEVAR may be associated with higher rates of reoperative requirements. Data remains limited on the long-term efficacy of the intervention and should continue to be investigated.

Keywords

- ▶ aneurysm
- ▶ aortic dissection
- ▶ endograft placement
- ▶ endovascular repair
- ▶ TEVAR
- ▶ pseudoaneurysm

Endovascular aortic grafts were initially developed to assist in repair of abdominal aortic aneurysms (AAAs). In the mid-1990s, Dake et al explored the possibility of repurposing these endografts for use in aneurysmal descending thoracic aortas, and in 1994 reported the first successful implantation of an endograft as a Thoracic EndoVascular Aortic repair (TEVAR).¹

Although Dake et al demonstrated a reproducible, safe approach to TEVAR in 1994, it was not approved for human implantation by the United States Food and Drug Administration (US FDA) until 2005, following completion of the Gore TAG pivotal trial. At that time the approval was limited to descending thoracic aortic aneurysms located in aortic zones 3, 4 or 5 (▶ **Table 1**).^{2,3} As graft technology has continued to advance, the variety of pathologies that TEVAR is actively used to treat has increased; however, these additional applications are considered “off-label” with few or no randomized trials comparing open versus endovascular management.⁴ As of only

2008, more than 50% of TEVAR applications in the United States were non-FDA approved or hybrid procedures, and with many major consensus papers having been published greater than five years prior, we hereby offer a brief review of the literature supported indications for TEVAR intervention.⁵

Indications for Thoracic Endovascular Repair

I. Descending Thoracic Aortic Aneurysm

As radiographic studies continue to become more readily available, the prevalence of incidentally diagnosed aneurysms is increasing. Current CHEST guidelines recommend endovascular intervention for asymptomatic, degenerative, or traumatic aneurysms exceeding 5.5 cm as well as all saccular aneurysms.^{6,7} Additionally, all symptomatic thoracic aortic aneurysms should be emergently managed by either open or endovascular techniques, with successful placement of endovascular graft allowing for complete

Table 1 Anatomic aortic landing zones

Zone	Location
0	Distal to the Coronary Ostia -> Proximal edge of the Innominate Artery
1	Distal to the Innominate Artery -> Proximal and involving the Left Common Carotid Artery
2	Distal to the Left Common Carotid Artery -> Proximal and involving the Left Subclavian Artery
3	Distal margin of the Left Subclavian Artery -> 2 cm distal to the Left Subclavian Artery
4	2 cm distal to the Left Subclavian Artery -> Midpoint of the Descending Thoracic Aorta
5	Midpoint of the Descending Thoracic Aorta -> Proximal margin of the Celiac Artery
6	Celiac Artery origin -> Proximal margin of the Superior Mesenteric Artery
7	Superior Mesenteric Artery -> Remainder of Suprarenal Aorta

occlusion of the aneurysm sac even in the presence of a tortuous descending aorta (►Fig. 1).

When endovascular approach is possible, multiple studies have demonstrated reduced morbidity and mortality when compared with open surgical management. Continuing, multiple observational studies have demonstrated reduced morbidity and mortality in stable patients and noninferiority in patients suffering from aortic trauma; however, the absence of significant prospective randomized trials is still noticeable in the literature.^{8,9}

When compared with open procedures, TEVAR grafting has been demonstrated to have the advantage of absence of a thoracotomy scar, reduced need for mechanical circulatory support, and no requirement of aortic cross clamping, thereby offering a resultant reduction in overall hospital morbidity, mortality, and length of stay.¹⁰ Demers et al have demonstrated that an endovascular approach is substantially beneficial in patients who are suboptimal candidates for an open procedure, resulting in greater long-term outcomes. When compared with open surgical repair for descending thoracic aortic aneurysms, TEVAR has been demonstrated to have a greater 30-day survival (98.1vs 94.3%) and one-year all-cause mortality benefit (91.6vs 85.5%). The VALOR trial demonstrated that when compared with a traditional open surgical approach, TEVAR demon-

strated a decrease in blood transfusion requirements (22.7vs 93.7%), reduced average procedure length (154.2 vs 303.3 minute) and a reduction in total length of stay in the intensive care unit (46.8 vs 185.3h).¹¹ Furthermore, whereby a cerebral vascular accident (CVA) is a feared complication of open aortic repair, TEVAR has been demonstrated to have a pooled prevalence of CVA of ~4.1%, with a 7.2% incidence marking the high end of reported CVA occurrences.^{12,13}

Continuing, traditional surgical mentality generally focuses on the removal of foreign surgical prostheses from infected fields; however, emerging evidence demonstrates the efficacy of utilizing TEVAR for management of mycotic aortic aneurysms (MAA). Patients with MAA are frequently higher-risk than those without active infection, with resultant traditional open surgical repair approaches having demonstrated approximately a 36% mortality rate.¹⁴ Although prolonged postoperative antibiotic therapy is indicated in endovascular management of thoracic MAAs, extrapolation of data from sub-diaphragmatic intervention is promising - MAAs in the abdominal aorta demonstrate a 94% survival rate at 12 months, compared with only a 70% survival rate with traditional open surgical abdominal MAA repair.¹⁵

II. Penetrating Atherosclerotic Ulcers and Intramural Hematomas

Penetrating Atherosclerotic Ulcer (PAU) has been identified radiographically in 10–20% of patients that demonstrate the clinical picture appropriate for an acute aortic dissection. (►Fig. 2)⁴ First described by Stanson et al, PAU is an ulceration penetrating the internal elastic lamina with possible resultant intramural hematoma (IMH) formation in the medial layer of the aorta, which is thought to evolve secondary to rupture of an atheromatous plaque.^{16–18} The resultant IMH is likely secondary to either vasa vasorum rupture and hemorrhage or microscopic tears in the aortic intima, and when present increases the rate of rupture to as high as 38%.^{4,17,18} Tittle et al demonstrated that surgical intervention improved 30-day survival with PAU complicated by IMH, indicating the importance of surgical intervention for these “virulent vascular lesions” if the patient’s other comorbidities do not preclude surgical intervention.¹⁸

PAU with IMH has a preferential involvement for the distal arch and descending thoracic aorta, making TEVAR an ideal therapeutic strategy.^{17,19} Current data suggests that asymptomatic PAU without IMH can be managed medically with close radiographic follow-up; however, if IMH is present, the patient is symptomatic, or radiographic growth is documented then urgent operative intervention is indicated.²⁰

Fig. 1 Successful placement of endograft for descending thoracic aortic aneurysm viewed in (A) axial orientation and (B) coronal orientation.

Fig. 2 CT with IV contrast demonstrating PAU in (A) axial and (B) coronal orientations.

Table 2 Grading of blunt aortic injury/trauma⁵⁴

Grade	Description
I	Aortic intimal tear
II	Aortic intramural hematoma
III	Aortic pseudoaneurysm
IV	Aortic Transection/Rupture

As a result of the segmental nature of PAU, short endovascular grafts are frequently effective at covering the lesion; however, long endograft (20–25 cm) utilization has been proposed to ensure coverage of any additional intimal disruption. With this approach, TEVAR to manage PAU has been documented to have a 4% neurologic morbidity rate and early mortality rates as low as 7%, which is significantly reduced from traditional open surgical rates that can approach as high as 21%.^{21,22}

Of note, at present time the Medtronic Talent, Medtronic Vallant, and Cook Zenith TX2 devices are currently approved by the FDA for management of PAU.²³

III. Aortic Pseudoaneurysm

Aortic pseudoaneurysm, also referred to as an aortic false aneurysm, results from a vascular transmural lesion resulting in a leak that is contained by the surrounding structures. Aortic pseudoaneurysm development is most commonly associated with recent trauma, with blunt aortic injury resulting in aortic pseudoaneurysm (►Table 2) and infection or a history of cardiac surgery providing the majority of the remaining pseudoaneurysm presentations.²⁴ When an aortic pseudoaneurysm is secondary to trauma, it is most commonly located in the region of the aortic isthmus, just distal to the left subclavian artery, or the diaphragmatic hiatus.^{25,26}

Given the unpredictability of the natural history of aortic pseudoaneurysm, debate continues regarding optimal medical therapy versus operative management of the pathology. Current suggestions are that for false aneurysms less than 2 cm in size without clinical symptomatology optimal medical management can be pursued. Supporting this approach, in a small study Gandhi et al noticed no statistically significant difference between optimal medical management and TEVAR in this subset of patients.²⁷ In this circumstance, strict blood pressure and impulse control was the mainstay of treatment.

For aortic pseudoaneurysms that are clinically symptomatic, greater than 2 cm in size, increasing in size via radiographic studies or actively ruptured/leaking, operative management is always preferred. In a small case series, TEVAR repair for both ascending and descending aortic pseudoaneurysm has been demonstrated to have a 100% survival rate at one year, with no return of symptoms or evidence of device migration.²⁸ Given the high morbidity associated with traditional open surgical intervention, endovascular repair is a favorable alternative. Demonstrating the versatility of TEVAR in management of this pathology, Pirelli et al demonstrated that in non-surgical candidates, innovative approaches can prove successful and placed an Amplatzer device to isolate an ascending aortic pseudoaneurysm via a direct ascending aortic approach.²⁹

Fig. 3 CT with IV contrast demonstrating Type B Aortic Dissection originating at the left subclavian artery in (A) axial and (B) coronal orientations.

At present time, Medtronic Vallant and Cook Zenith TX2 devices are both approved by the US FDA for management of descending aortic pseudoaneurysms. Although evidence is in support of utility of these devices for frail and unstable patients, caution is still advised in young patients given the absence of data and uncertainty surrounding long-term graft implantation.⁶

IV. Type B Aortic Dissection

Aortic dissection describes a pathology whereby an intimal tear allows blood from the true lumen to form a false lumen between the intimal and medial layers of the aortic wall (►Fig. 3).³⁰ The Stanford classification scheme separates these dissections into two types: Stanford Type A involving the ascending aorta and/or the aortic arch, and Stanford Type B involving the descending thoracic aorta distal to the left subclavian. Temporally, aortic dissections are divided into acute, whereby the diagnosis is made within fourteen days of clinical symptoms, and subacute/chronic whereby diagnosis is delayed beyond fourteen days of symptom onset (►Fig. 4).

A. Acute

In this section, we will focus on those dissections of the descending aorta diagnosed within 14 days of symptom onset (Acute, Type B).

Acute Type B Aortic Dissections (aTBAD) are further subdivided into uncomplicated and complicated subdivisions. Complicated aTBAD describes a scenario whereby there is evidence of malperfusion, aortic rupture, hypotension, shock, neurologic complications, refractory pain, refractory hypertension, aortic dilation, or propagation of the dissection flap. Conversely, an uncomplicated aTBAD is the presence of a dissection flap without evidence of the aforementioned clinical signs or symptoms.³⁰

a. Uncomplicated Acute Type B Aortic Dissection

Uncomplicated aTBAD has traditionally been managed medically, with close blood pressure control, impulse control and negative chronotropic agents. When medical management is pursued, a 90to 100% in-hospital survival rate and a 5-year survival rate of 50to 87% have been demonstrated.^{30–32} When treated conservatively, however, these patients remain at risk for long-term complications including aneurysmal degeneration and rupture. Between 20and 55% of patients treated medically will require operative management within 5 years, at which point traditional open surgical intervention may carry a neurologic morbidity and mortality rate of as high as 3and 5%, respectively.^{31,33–35}

Fig. 4 Type B Aortic Dissection Classification Criteria.

With the primary cause of morbidity and mortality continued expansion of the false lumen, operative intervention is focused at maintaining perfusion of the true lumen and eliminating blood flow into the false lumen by obliterating the primary intimal tear (—Fig. 5).^{36,37} With a mortality rate at 6-year follow-up for aTBAD managed with TEVAR as low as 3.2 deaths per 100 patient-years, there is emerging support for management of uncomplicated aTBAD with TEVAR.³⁸ Continuing, the INSTEAD-XL trial demonstrated lower overall and aorta-specific mortality with TEVAR when compared with medical management at 7 versus 19% and 11 versus 19%, respectively.³⁹ Ray et al have suggested high risk criteria including: aortic diameter greater than 44 mm, age greater than 60, and false lumen diameter greater than 22 mm, may carry a worse prognosis and receive greater benefit from TEVAR when compared with conservative, optimal medical management.⁴⁰

Fig. 5 CT with IV contrast demonstrating Type B Aortic Dissection with endograft placement demonstrating successful obliteration of the intimal tear in (A) axial and (B) coronal orientations.

b. Complicated Acute Type B Aortic Dissection

Complicated aTBADs demand immediate operative intervention with optimal medical therapy demonstrating as high as a 33% in-hospital or 30-day mortality.⁴¹ Traditional open repair of complicated aTBAD has been demonstrated to carry mortality, stroke, and renal failure rates of 15 to 30%, 10, and 20%, respectively.^{35,42} Conversely, TEVAR has been demonstrated to offer as low as a 7.3% in-hospital mortality rate, a 1.9 to 6.4% in-hospital stroke rate and an 8% 30-day mortality rate.^{37,43}

Dissimilar to uncomplicated aTBAD, complicated aTBAD should be managed immediately with optimal medical therapy and then urgently with operative TEVAR when possible. The US FDA has approved the Gore TAG and Medtronic Valiant endografts for management of complicated aTBAD.

B. Subacute/Chronic

In this section, we will focus on those dissections of the descending aorta diagnosed after 14 days of symptom onset (Chronic, Type B).

Chronic Type B Aortic Dissections (cTBAD) were traditionally considered to be stable and therefore managed with optimal medical therapy, focusing on heart rate and blood pressure control. Elective, traditional open surgical repair carrying reported rates of stroke, paraplegia, and mortality of 16, 9, and 6% respectively.

Medical management alone does not remove the risk of aneurysmal growth, refractory pain or recurrent dissection/rupture, and with low morbidity and mortality associated with TEVAR, endovascular management of cTBAD is a growing

consideration.^{33,44} The goal of endovascular intervention in this subgroup is to prevent aortic false lumen growth via exclusion. Technical success has been reported as high as 90 to 96% for cTBAD endovascular repairs, and rates of paraplegia and early mortality are as low as 0.4 and 5%, respectively.³⁴ Kang et al have reported that in up to 85% of patients with complicated cTBAD TEVAR can stop or even reverse pathologic aortic growth.⁴⁴ With the STABLE trial demonstrating acute and nonacute placement of an endograft resulting in growth of the true lumen and regression of the false lumen, and the mother registry demonstrating a 6-year mortality rate under 3%, there is growing support in the efficacy of utilizing an endograft for the management of cTBAD.^{30,38,45}

Although traditionally cTBAD has been thought stable, 17% of patients with cTBAD will present with symptoms similar to complicated aTBAD, resulting in the temporal relevance of chronic stability being questioned. With aortic plasticity being demonstrated to exist beyond the 14-day cutoff, a newly recognized subacute phase extending from day 15 to 85 has come into existence whereby operative intervention is suggested to be as appropriate as in the management of complicated aTBAD.^{30,46}

V. Blunt Trauma/Thoracic Aortic Injury

Blunt aortic injury resulting in traumatic aortic rupture occurs most frequently as a result of a deceleration injury most commonly impacting the aortic isthmus or diaphragmatic hiatus, where the aorta is most immobile, and carries a tremendous morbidity and mortality rate.^{25,26} Blunt aortic injury has an incidence of ~0.3% of all traumas entered in the National Trauma Database, and carries between a 23 and 85% mortality rate before completion of the trauma triage, making it the second most common cause of death in trauma presentations.^{47–49} Of those patients that survive triage, mortality rates have been reported as high as 65% for patients not operatively managed and historically as high as 54% for those patients managed with traditional open surgical repair with as high as a 19% risk of paraplegia postoperatively. Further complicating management, 90% of patients will present with comorbid injuries, of which 24% will require intervention prior to aortic repair. Resultantly, recent data continues to demonstrate mortality rates as high as 20 to 40% for traditional open surgical repair of patients surviving the initial resuscitative effort.^{49–53}

Thoracic aortic traumatic injuries fall into 4 categories that are useful in dictating management, and is detailed in ▶Table 2.⁵⁴

Medical management to ensure tight blood pressure and heart rate control is frequently initiated in all patients with blunt aortic injury. In stable patients with grade I injury, optimal medical management alone is sufficient without a significant increased risk of rupture; however, in patients with grade II (▶Fig. 6) or higher injury operative management – endovascular or open – is recommended given the reported 2 to 5% risk of delayed rupture, frequently within one week of the initial insult.⁵⁵

TEVAR is a promising therapeutic strategy for Grade II+ blunt aortic injury in patients who are anatomic candidates. Procedure related mortality rates for endovascular repair have been demonstrated to be as low as 2 to 9%, with overall

Fig. 6 CT with IV contrast demonstrating IMH secondary to trauma extending from the left subclavian artery to the abdominal aorta in (A) axial and (B) sagittal orientations.

30-day mortality reported at ~8%, a significant reduction from traditional operative management.^{49,54,56} Additionally, in a large systemic review, Hoffer et al demonstrated a 0.30 (95% CI, 0.12–0.76, $p = 0.01$) odd ratio for paraplegia following endovascular repair when compared with open repair.⁵⁰ Long-term durability of endografts remains a concern; however, Szostek et al have demonstrated at 14 years following TEVAR for blunt aortic injury up to 82% of patients remain asymptomatic without radiographic evidence of graft migration.⁵⁷

It is important to note that although TEVAR is a promising intervention for patients undergoing a traditionally highly morbid injury, at present time the graft choices designed for traumatic aortic injury are limited and as such, anatomic limitations may prevent endovascular intervention or lead to increased procedural mortality.⁵⁴

VI. Aorto-esophageal and Aortobronchial Fistula

Forty years ago Carter et al recognized the lethal progression of Aorto-esophageal (AE) fistulas through Chiari's triad (thoracic pain, sentinel hemorrhage, a symptom free interval and lethal hemorrhage) as well as the need for operative intervention.⁵⁸ AE and Aortobronchial (AB) fistula describe pathologic fistulae between either the aorta and the esophagus or the aorta and the bronchial tree, respectively.

Traditionally, primary fistula occurs without prior interventions as a result of thoracic aortic aneurysm, PAU, neoplasm, intrapulmonary disease (e.g., tuberculosis, aspergillus) or foreign body aspiration/ingestion. Secondary fistula occurs as postoperative complications of graft placement or surgical procedure.

AE and AB fistulas are exceedingly rare but carry a mortality rate reported as high as 50 and 41%, respectively, when undergoing emergent traditional open surgical repair of a primary fistulae and can approach 78% for patients undergoing repair of a secondary fistula.^{59–63}

Given the high mortality rates, endovascular intervention is an attractive alternative; however, TEVAR alone will not repair the esophageal or bronchial pathology and the lack of sterility of the respiratory tree and esophagus greatly increase the risk of graft infection. Canaud et al have demonstrated the success of primary endovascular intervention for AB fistula with a 6.4% mortality rate and no evidence of paraplegia.⁶³ Jonker et al have reported technical success for endovascular intervention in AB fistula of 90% and AE fistula of 91%; however, given the high risk of sepsis or mediastinitis

the in hospital mortality rate for AE fistula remained high at 19% when compared with AB fistula repair at 3%. Additionally, 4 and 44% of patients undergoing TEVAR for AB or AF fistula, respectively, required additional surgical intervention to repair the esophageal or bronchial pathology.⁶⁰

Data are emerging regarding hybrid interventions combining TEVAR for emergent hemorrhage control followed by surgical repair of the bronchial tree or esophagus. Kahlberg et al have demonstrated that emergent TEVAR followed by open repair has resulted in an 87.5% survival rate at 34 months and no in-hospital mortality.⁶² Continuing, Canaud et al demonstrated endovascular emergent repair followed by open surgical repair for AB fistula resulted in a 100% technical success rate and 80% of patients had an uneventful postoperative course at an average follow-up of 23 months.⁶³

Given the high risk of infection secondary to graft contact with either the bronchial tree or the esophagus, antibiotics are likely beneficial for at least 6 weeks postoperatively.

Summary

TEVAR continues to be a promising modality for managing thoracic aortic pathologies. When compared with open surgical approaches, efficacy has been demonstrated to be similar or better than traditional open surgical procedures, with greatly improved morbidity and mortality in most instances; however, currently available data assesses only short and midterm outcomes. As long-term data becomes available, rates of reintervention are appearing higher with TEVAR compared with traditional open intervention, but no prospective trials currently exist demonstrating long-term outcomes comparing traditional and endovascular interventions. Additionally, the device market continues to rapidly grow, but with a majority of interventions being used "off-label," there is tremendous room for improvement in pathology management. As more devices come to market and FDA approval expands, it is likely the applicability of TEVAR will continue to evolve as well.

As TEVAR and medical technology continues to mature as a field we will continue to see the applicability of the intervention expand to manage additional pathologies. With the continued expansion of electronic data gathering in medicine, currently existing databases will continue to expand and allow for better understanding of the short-, mid-, and long-term outcomes of TEVAR for various aortic pathologies.

References

- 1 Dake MD, Miller DC, Semba CP, Mitchell RS, Walker PJ, Liddell RP. Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms. *N Engl J Med* 1994; 331(26):1729–1734. Doi: 10.1056/NEJM199412293312601
- 2 Makaroun MS, Dillavou ED, Kee ST, et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. *J Vasc Surg* 2005; 41(01):1–9. Doi: 10.1016/j.jvs.2004.10.046
- 3 Fillingim MF, Greenberg RK, McKinsey JF, Chaikof EL; Society for Vascular Surgery Ad Hoc Committee on TEVAR Reporting Standards. Reporting standards for thoracic endovascular aortic repair (TEVAR). *J Vasc Surg* 2010;52(04):1022–1033, 1033.e15
- 4 Hiratzka LF, Bakris GL, Beckman JA, et al; American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines; American Association for Thoracic Surgery; American College of Radiology; American Stroke Association; Society of Cardiovascular Anesthesiologists; Society for Cardiovascular Angiography and Interventions; Society of Interventional Radiology; Society of Thoracic Surgeons; Society for Vascular Medicine. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. *Circulation* 2010;121(13):e266–e369. Doi: 10.1161/CIR.0b013e3181d4739e
- 5 Hughes GC, Daneshmand MA, Swaminathan M, et al. "Real world" thoracic endografting: results with the Gore TAG device 2 years after U.S. FDA approval. *Ann Thorac Surg* 2008; 86(05):1530–1537, discussion 1537–1538. Doi: 10.1016/j.athoracsur.2008.07.089
- 6 Svensson LG, Kouchoukos NT, Miller DC, et al; Society of Thoracic Surgeons Endovascular Surgery Task Force. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. *Ann Thorac Surg* 2008;85(1, Suppl):S1–S41. Doi: 10.1016/j.athoracsur.2007.10.099
- 7 Matsumura JS, Cambria RP, Dake MD, Moore RD, Svensson LG, Snyder S; TX2 Clinical Trial Investigators. International controlled clinical trial of thoracic endovascular aneurysm repair with the Zenith TX2 endovascular graft: 1-year results. *J Vasc Surg* 2008;47(02):247–257, discussion 257. Doi: 10.1016/j.jvs.2007.10.032
- 8 Walsh SR, Tang TY, Sadat U, et al. Endovascular stenting versus open surgery for thoracic aortic disease: systematic review and meta-analysis of perioperative results. *J Vasc Surg* 2008;47(05):1094–1098. Doi: 10.1016/j.jvs.2007.09.062
- 9 Abrahams I, Romagnoli C, Montedori A, Cirocchi R. Thoracic stent graft versus surgery for thoracic aneurysm. *Cochrane Database Syst Rev* 2013;CD006796(09):CD006796. Doi: 10.1002/14651858.CD006796.pub3
- 10 Demers P, Miller DC, Mitchell RS, et al. Midterm results of endovascular repair of descending thoracic aortic aneurysms with first-generation stent grafts. *J Thorac Cardiovasc Surg* 2004;127(03):664–673. Doi: 10.1016/j.jtcvs.2003.10.047
- 11 Fairman RM, Criado F, Farber M, et al; VALOR Investigators. Pivotal results of the Medtronic Vascular Talent Thoracic Stent Graft System: the VALOR trial. *J Vasc Surg* 2008;48(03):546–554. Doi: 10.1016/j.jvs.2008.03.061
- 12 Maldonado TS, Dexter D, Rockman CB, et al. Left subclavian artery coverage during thoracic endovascular aortic aneurysm repair does not mandate revascularization. *J Vasc Surg* 2013;57(01):116–124. Doi: 10.1016/j.jvs.2012.06.101
- 13 von Allmen RS, Gahl B, Powell JT. Editor's Choice - Incidence of Stroke Following Thoracic Endovascular Aortic Repair for Descending Aortic Aneurysm: A Systematic Review of the Literature with Meta-analysis. *Eur J Vasc Endovasc Surg* 2017;53(02):176–184. Doi: 10.1016/j.ejvs.2016.10.025
- 14 Huang YK, Ko PJ, Chen CL, et al. Therapeutic opinion on endovascular repair for mycotic aortic aneurysm. *Ann Vasc Surg* 2014;28(03):579–589. Doi: 10.1016/j.avsg.2013.07.009
- 15 Kan CD, Lee HL, Yang YJ. Outcome after endovascular stent graft treatment for mycotic aortic aneurysm: a systematic review. *J Vasc Surg* 2007;46(05):906–912. Doi: 10.1016/j.jvs.2007.07.025
- 16 Stanson AW, Kazmier FJ, Hollier LH, et al. Penetrating atherosclerotic ulcers of the thoracic aorta: natural history and

- clinicopathologic correlations. *Ann Vasc Surg* 1986;1(01):15–23. Doi: 10.1016/S0890-5096(06)60697-3
- 17 Nathan DP, Boonn W, Lai E, et al. Presentation, complications, and natural history of penetrating atherosclerotic ulcer disease. *J Vasc Surg* 2012;55(01):10–15. Doi: 10.1016/j.jvs.2011.08.005
  - 18 Tittle SL, Lynch RJ, Cole PE, et al. Midterm follow-up of penetrating ulcer and intramural hematoma of the aorta. *J Thorac Cardiovasc Surg* 2002;123(06):1051–1059
  - 19 Harris KM, Braverman AC, Eagle KA, et al. Acute aortic intramural hematoma: an analysis from the International Registry of Acute Aortic Dissection. *Circulation* 2012;126(11, Suppl 1):S91–S96. Doi: 10.1161/CIRCULATIONAHA.111.084541
  - 20 Grabenwöger M, Alfonso F, Bachet J, et al; European Association for Cardio-Thoracic Surgery (EACTS); European Society of Cardiology (ESC); European Association of Percutaneous Cardiovascular Interventions (EAPCI). Thoracic Endovascular Aortic Repair (TEVAR) for the treatment of aortic diseases: a position statement from the European Association for Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI). *Eur J Cardiothorac Surg* 2012;42(01):17–24. Doi: 10.1093/ejcts/ezs107
  - 21 Cho KR, Stanson AW, Potter DD, Cherry KJ, Schaff HV, Sundt TM III. Penetrating atherosclerotic ulcer of the descending thoracic aorta and arch. *J Thorac Cardiovasc Surg* 2004;127(05):1393–1399, discussion 1399–1401. Doi: 10.1016/j.jtcvs.2003.11.050
  - 22 Eggebrecht H, Plicht B, Kahlert P, Erbel R. Intramural hematoma and penetrating ulcers: indications to endovascular treatment. *Eur J Vasc Endovasc Surg* 2009;38(06):659–665. Doi: 10.1016/j.ejvs.2009.09.001
  - 23 Cho JS, Haider SE, Makaroun MS. US multicenter trials of endoprotheses for the endovascular treatment of descending thoracic aneurysms. *J Vasc Surg* 2006;43(Suppl A):12A–19A
  - 24 Atik FA, Navia JL, Svensson LG, et al. Surgical treatment of pseudoaneurysm of the thoracic aorta. *J Thorac Cardiovasc Surg* 2006;132(02):379–385. Doi: 10.1016/j.jtcvs.2006.03.052
  - 25 Neschis DG, Scalea TM, Flinn WR, Griffith BP. Blunt aortic injury. *N Engl J Med* 2008;359(16):1708–1716. Doi: 10.1056/NEJMra0706159
  - 26 Sevitt S. The mechanisms of traumatic rupture of the thoracic aorta. *Br J Surg* 1977;64(03):166–173
  - 27 Gandhi SS, Blas JV, Lee S, Eidt JF, Carsten CG III. Nonoperative management of grade III blunt thoracic aortic injuries. *J Vasc Surg* 2016;64(06):1580–1586. Doi: 10.1016/j.jvs.2016.05.070
  - 28 Steinberg ZL, Don CW, Sun JC, Gill EA Jr, Goldberg SL. Percutaneous Repair of Aortic Pseudoaneurysms: A Case Series. *J Invasive Cardiol* 2016;28(01):E6–E10
  - 29 Pirelli L, Kliger C, Fontana GP, Ruiz CE. Direct percutaneous trans-aortic approach for treatment of aortic pseudoaneurysms. *Interact Cardiovasc Thorac Surg* 2015;20(05):680–681. Doi: 10.1093/icvts/ivv027
  - 30 Alfson DB, Ham SW. Type B. Type B Aortic Dissections: Current Guidelines for Treatment. *Cardiol Clin* 2017;35(03):387–410. Doi: 10.1016/j.ccl.2017.03.007
  - 31 Fleerackers J, Schepens M. How should we manage dissections type B aortic? *Gen Thorac Cardiovasc Surg* 2017;••••. Doi: 10.1007/s11748-017-0818-5
  - 32 Kodama K, Nishigami K, Sakamoto T, et al. Tight heart rate control reduces secondary adverse events in patients with type B acute aortic dissection. *Circulation* 2008;118(14, Suppl):S167–S170. Doi: 10.1161/CIRCULATIONAHA.107.755801
  - 33 Andersen ND, Keenan JE, Ganapathi AM, Gaca JG, McCann RL, Hughes GC. Current management and outcome of chronic type B aortic dissection: results with open and endovascular repair since the advent of thoracic endografting. *Ann Cardiothorac Surg* 2014;3(03):264–274. Doi: 10.3978/j.issn.2225-319X.2014.05.07
  - 34 Khoynezhad A, Toluie S, Al-Atassi T. Treatment of the Chronic Type B Aortic Dissection: The Pro-endovascular Argument. *Semin Thorac Cardiovasc Surg* 2017;29(02):131–136. Doi: 10.1053/j.semtcvs.2017.03.001
  - 35 Parsa CJ, Schroder JN, Daneshmand MA, McCann RL, Hughes GC. Midterm results for endovascular repair of complicated acute and chronic type B aortic dissection. *Ann Thorac Surg* 2010;89(01):97–102, discussion 102–104. Doi: 10.1016/j.athoracsur.2009.09.029
  - 36 Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med* 1999;340(20):1546–1552. Doi: 10.1056/NEJM199905203402004
  - 37 Nauta FJ, Trimarchi S, Kamman AV, et al. Update in the management of type B aortic dissection. *Vasc Med* 2016;21(03):251–263. Doi: 10.1177/1358863 × 16642318
  - 38 Patterson B, Holt P, Nienaber C, Cambria R, Fairman R, Thompson M. Aortic pathology determines midterm outcome after endovascular repair of the thoracic aorta: report from the Medtronic Thoracic Endovascular Registry (MOTHER) database. *Circulation* 2013;127(01):24–32. Doi: 10.1161/CIRCULATIONAHA.112.110056
  - 39 Nienaber CA, Kische S, Rousseau H, et al; INSTEAD-XL trial. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. *Circ Cardiovasc Interv* 2013;6(04):407–416. Doi: 10.1161/CIRCINTERVENTIONS.113.000463
  - 40 Ray HM, Durham CA, Ocazonez D, et al. Predictors of intervention and mortality in patients with uncomplicated acute type B aortic dissection. *J Vasc Surg* 2016;64(06):1560–1568. Doi: 10.1016/j.jvs.2016.07.111
  - 41 Zeeshan A, Woo EY, Bavaria JE, et al. Thoracic endovascular aortic repair for acute complicated type B aortic dissection: superiority relative to conventional open surgical and medical therapy. *J Thorac Cardiovasc Surg* 2010;140(6, Suppl):S109–S115, discussion S142–S146
  - 42 Trimarchi S, Nienaber CA, Rampoldi V, et al; IRAD Investigators. Role and results of surgery in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). *Circulation* 2006;114(1, Suppl):I357–I364. Doi: 10.1161/CIRCULATIONAHA.105.000620
  - 43 Virtue Registry I; Virtue Registry Investigators. The VIRTUE Registry of type B thoracic dissections—study design and early results. *Eur J Vasc Endovasc Surg* 2011;41(02):159–166. Doi: 10.1016/j.ejvs.2010.08.016
  - 44 Kang WC, Greenberg RK, Mastracci TM, et al. Endovascular repair of complicated chronic distal aortic dissections: intermediate outcomes and complications. *J Thorac Cardiovasc Surg* 2011;142(05):1074–1083. Doi: 10.1016/j.jtcvs.2011.03.008
  - 45 Lombardi JV, Cambria RP, Nienaber CA, et al; STABLE investigators. Aortic remodeling after endovascular treatment of complicated type B aortic dissection with the use of a composite device design. *J Vasc Surg* 2014;59(06):1544–1554. Doi: 10.1016/j.jvs.2013.12.038
  - 46 Steuer J, Björck M, Mayer D, Wanhainen A, Pfammatter T, Lachat M. Distinction between acute and chronic type B aortic dissection: is there a sub-acute phase? *Eur J Vasc Endovasc Surg* 2013;45(06):627–631. Doi: 10.1016/j.ejvs.2013.03.013
  - 47 Clancy TV, Gary Maxwell J, Covington DL, Brinker CC, Blackman D. A statewide analysis of level I and II trauma centers for patients with major injuries. *J Trauma* 2001;51(02):346–351
  - 48 Cowley RA, Turney SZ, Hankins JR, Rodriguez A, Attar S, Shankar BS. Rupture of thoracic aorta caused by blunt trauma. A fifteen-year experience. *J Thorac Cardiovasc Surg* 1990;100(05):652–660, discussion 660–661
  - 49 Xenos ES, Abedi NN, Davenport DL, et al. Meta-analysis of endovascular vs open repair for traumatic descending thoracic aortic rupture. *J Vasc Surg* 2008;48(05):1343–1351. Doi: 10.1016/j.jvs.2008.04.060
  - 50 Hoffer EK, Forauer AR, Silas AM, Gemery JM. Endovascular stent-graft or open surgical repair for blunt thoracic aortic trauma:

- systematic review. *J Vasc Interv Radiol* 2008;19(08):1153–1164. Doi: 10.1016/j.jvir.2008.05.012
- 51 Arthurs ZM, Starnes BW, Sohn VY, Singh N, Martin MJ, Andersen CA. Functional and survival outcomes in traumatic blunt thoracic aortic injuries: An analysis of the National Trauma Databank. *J Vasc Surg* 2009;49(04):988–994. Doi: 10.1016/j.jvs.2008.11.052
  - 52 Cardarelli MG, McLaughlin JS, Downing SW, Brown JM, Attar S, Griffith BP. Management of traumatic aortic rupture: a 30-year experience. *Ann Surg* 2002;236(04):465–469, discussion 469–470. Doi: 10.1097/01.SLA.0000028968.68441.A5
  - 53 Pate JW, Fabian TC, Walker W. Traumatic rupture of the aortic isthmus: an emergency? *World J Surg* 1995;19(01):119–125, discussion 125–126
  - 54 Lee WA, Matsumura JS, Mitchell RS, et al. Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the Society for Vascular Surgery. *J Vasc Surg* 2011;53(01):187–192. Doi: 10.1016/j.jvs.2010.08.027
  - 55 Tehrani HY, Peterson BG, Katariya K, et al. Endovascular repair of thoracic aortic tears. *Ann Thorac Surg* 2006;82(03):873–877, discussion 877–878. Doi: 10.1016/j.athoracsur.2006.04.012
  - 56 Tang GL, Tehrani HY, Usman A, et al. Reduced mortality, paraplegia, and stroke with stent graft repair of blunt aortic transections: a modern meta-analysis. *J Vasc Surg* 2008;47(03):671–675. Doi: 10.1016/j.jvs.2007.08.031
  - 57 Szostek M, Jakuczun W, Oseka M, et al. Endovascular treatment of traumatic rupture of thoracic aorta: long term results. *Int Angiol* 2016;35(02):129–139
  - 58 Carter R, Mulder GA, Snyder EN Jr, Brewer LA III. Aortoesophageal fistula. *Am J Surg* 1978;136(01):26–30
  - 59 Dorweiler B, Weigang E, Duenschede F, Pitton MB, Dueber C, Vahl CF. Strategies for endovascular aortic repair in aortobronchial and aortoesophageal fistulas. *Thorac Cardiovasc Surg* 2013;61(07):575–580. Doi: 10.1055/s-0033-1347294
  - 60 Jonker FH, Heijmen R, Trimarchi S, Verhagen HJ, Moll FL, Muhs BE. Acute management of aortobronchial and aortoesophageal fistulas using thoracic endovascular aortic repair. *J Vasc Surg* 2009;50(05):999–1004. Doi: 10.1016/j.jvs.2009.04.043
  - 61 Chiesa R, Kahlberg A, Tshomba Y, Marone EM, Marrocco-Trischitta MM, Melissano G. Endovascular repair of aortoesophageal and aortobronchial fistulae. *Tex Heart Inst J* 2011;38(06):655–657
  - 62 Kahlberg A, Tshomba Y, Marone EM, Castellano R, Melissano G, Chiesa R. Current results of a combined endovascular and open approach for the treatment of aortoesophageal and aortobronchial fistulae. *Ann Vasc Surg* 2014;28(07):1782–1788. Doi: 10.1016/j.avsg.2014.06.003
  - 63 Canaud L, D'Annunzio T, Ozdemir BA, Marty-Ané C, Alric P. Combined endovascular and surgical approach for aortobronchial fistula. *J Thorac Cardiovasc Surg* 2014;148(05):2108–2111. Doi: 10.1016/j.jtcvs.2014.01.018