Article published online: 2024-11-15

Endovascular Repair of Zone 0 Ascending Aortic Aneurysm: A Review of Current Knowledge and Developing Technology

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Aorta (Stamford) 2024;12:13–19.

Abstract

Aortic aneurysms represent the 15th leading cause of death in men and women over 55 years of age. Where historically these lesions were all addressed via an open approach, endovascular aortic repair has entirely altered the way that surgeons approach aortic lesions. Although it was initially employed for patients who were poor surgical candidates, endovascular repair is now standard for abdominal aortic aneurysms and aneurysms in the descending thoracic aorta. Open surgery remains the gold standard for management of ascending aneurysms, in part due to the limitations portended by the anatomy of the ascending aorta, although increasing evidence suggests that endovascular approaches are feasible and may sometimes be optimal for patient outcomes. Here, we present some of the anatomical and technical challenges of the endovascular approach to these "Zone 0" aneurysms, the associated complications, and the current state of device development.

Keywords

- ascending aorta
- thoracic endovascular aortic repair
- stent grafting
- landing zone

Background

The Centers for Disease Control and Prevention report that aortic aneurysm, both thoracic and abdominal, represent the 15th leading cause of death in men and women >55 years of age, and approximately the 19th leading cause of death overall.[1] Although the overall prevalence of thoracic aortic aneurysm (TAA) is lower than that of abdominal aortic aneurysm (AAA), some of this may be secondary to underreporting, given the lethal nature of acute presentations coupled with variable autopsy rates.[1] Their true prevalence is further obscured by misdiagnosis, as many patients who suffer a fatal rupture are mistakenly diagnosed with myocardial infarction.[2] Given that the general populace is now undergoing more diagnostic imaging than ever before, the overall prevalence of TAA has also been increasing between 2002 and 2014 from a prior estimate of 3.5 to 7.6/100,000 persons.[2]

Approximately 60% of TAA present at the aortic root and/or the ascending aorta, 10% in the arch, and 30% in the

received August 3, 2023
accepted after revision September 5, 2024
article published online November 15, 2024

DOI https://doi.org/10.1055/s-0044-1791670.
ISSN 2325-4637.

descending aorta. Open surgery was the only option for ascending and descending TAA until the advent of thoracic endovascular aortic repair (TEVAR) in the 1990s.[3] However, open surgery conveys an approximate mortality risk of 12%, which further increases in the setting of an acute rupture or dissection.[3] Although TEVAR is now first-line therapy for descending thoracic aneurysms, open repair remains the standard for ascending aneurysms due to the limitations portended by the anatomy of the ascending aorta; specifically the presence of the aortic valve and related pathology, the location of the coronary ostia, the brachiocephalic artery, and the highly dynamic nature of the aorta during the cardiac cycle.[4] Endovascular options for the management of AAAs were introduced in the 1990s, with current data suggesting a perioperative survival advantage over open surgical repair, although outcomes become equivocal with regard to long-term survival at 6 years, based on the results of the DREAM2 trial.[5] This technique gradually became adopted for management of descending TAA, introduced in 1994 and ultimately approved by the U.S. Food and Drug Administration (FDA) in 2005. Currently available long-term data derived from studies sourcing from the Medicare database suggest the initial survival benefit becomes equivocal at 2 years, further limiting the ability for extant guidelines to provide information outside of absolute anatomical contraindications or an inability to tolerate open surgery.[5] However, options for endovascular management of Zone 0 TAA have remained controversial. This is largely due to a lack of sufficient data demonstrating clear indications for endovascular management relative to open surgery, no currently commercially available dedicated graft for Zone 0 TEVAR, and no guidelines on adopting currently available techniques and graft materials to this location.[5] This report describes the specific anatomical considerations of the ascending aorta, relevant technical endovascular considerations, and potential complications when undertaking TEVAR for the ascending aorta.

Anatomy

As mentioned above, the aorta is grossly divided into ascending, arch and descending portions. It can then further be divided into distinct zones, as shown in ►**Fig. 1**.

The first part of the ascending aorta is described as Zone 0, anatomically described as between the aortic valve annulus and the origin of the brachiocephalic artery,[6] can then be itself subdivided based on the location of the sinotubular junction, with subzone A located below the sinotubular junction, subzone B extending from the sinotubular junction to the mid-ascending aorta, and subzone C including the mid-ascending to distal ascending aorta.[6] The ascending aorta is highly variable with respect to its morphology and patient-specific anatomy. Given the lack of a dedicated graft being commercially available, there is a discordance with regard to the design of implants relative to the anatomy of the ascending aorta.[6] Currently employed grafts have a straight and tubular design, in contrast to the native aorta, which has a slight curve. Furthermore, the posterior wall of the aorta and lesser curve of the aorta are therefore shorter in

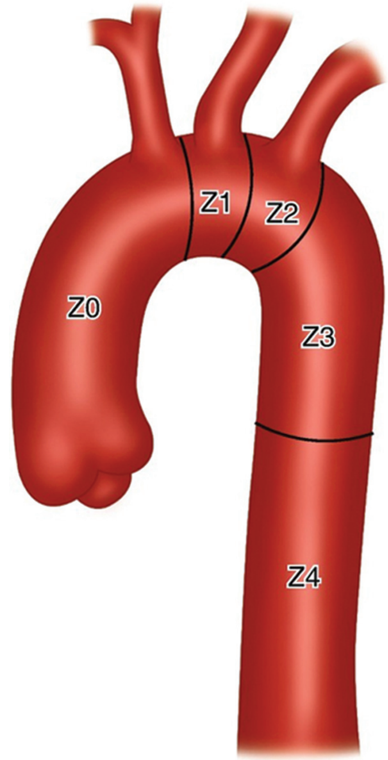


Fig. 1 Aortic zones. Reproduced with permission from Rodriguez et al 2019.[54]

linear distance when compared with the greater curve and corresponding anterior wall, which have a longer length.[7]

The arterial wall comprises three layers: the intima, media, and adventitia. The tunica media is composed of lamellar units interconnected by a network of elastic fibers and collagen.[7] These lamellar units decrease along the more distal aorta in terms of absolute number, and the number of units positively correlate with physiological pressure and elasticity. The ascending aorta, namely Zone 0, is highly dynamic during the cardiac cycle.[7] This is perhaps epitomized by the Windkessel effect, in which approximately 50% of left ventricular stroke volume is stored in the proximal aorta during systole.[8] Physiologically, this allows for continuous blood flow to the periphery during diastole and reduced left ventricular afterload, simultaneously improving coronary blood flow and diastolic relaxation of the left ventricle.[7]

The elastic fibers exhibit low rates of turnover, and progressively accumulate age-related fragmentation, calcification, and decay due to the basal activity of matrix metalloproteinases found within the arterial wall.[8] In contrast, collagen found within all three layers of the arterial wall increases with age.[9] As the ratio of collagen: elastin fibers continues to increase with age, these fibers become progressively glycated via a nonenzymatic reaction predicated on the presence of reducing sugars.[9] As these glycated end products accumulate, they become mechanically cross-linked and work to decrease the availability of nitric oxide availability, further impacting the maintenance of vascular tone via mechanical and chemical changes.[10] This ultimately results in an age-related decrease in the Windkessel effect, increased systolic resistance, and negative impact on coronary blood flow with decreased myocardial perfusion.[10]

TAA is the pathophysiological result of medial degeneration, as illustrated above.[10] It remains poorly understood, with animal modeling and tissue analyses extrapolated from AAA specimens proving misleading resulting from anatomical heterogeneity throughout the aorta, specifically above and below the diaphragm.[11] The combination of anatomical changes such as changes in extracellular matrix microfibril density, small molecules such as homocysteine, hypertension resulting in constant exposure to angiotensin II, and genetic predisposition ultimately increase risk of TAA development.[12] Current guidelines recommend intervention for ascending TAA at a diameter of 5.5 cm or greater, with a lower threshold for intervention in those with documented rapid growth, a known genetic aortopathy, or a family history of aortic rupture or dissection.[13]

These physiological and anatomical considerations are critical with respect to postoperative physiological changes after placement of a synthetic graft.[14] Synthetic grafts are typically made of polyethylene terephthalate (Dacron), Polyester, or polytetrafluoroethylene.[15] Regardless of material chosen, there will be alteration of arterial compliance, contributing to antegrade and retrograde hemodynamic changes.[16] The alteration in mechanical distension of the aorta places focal stress at the proximal anastomosis, leading to intimal hyperplasia, aneurysmal degeneration, and potential pseudoaneurysm formation.[16] The risk of pseudoaneurysm formation is currently listed at between 2 and 6% in available literature[16] and may present with increasing diameter, rupture, fistula formation, or thrombosis.[16] These complications occur along a spectrum, dependent on the absolute reduction in aortic compliance.[16] In pediatric and younger patients who have an elevated risk of an underlying connective tissue disorder or aortic valve pathology being an indication for surgical intervention, the risk of pseudoaneurysm formation is approximately 8%.[17] Mattens et al[18] reported that in addition to these antegrade changes, grafts are known to have a 31.4% dilation relative to their packaged diameter, with successive 3% annual increase in diameter over 2-year follow-up. These physical changes in the acute postoperative period along with subsequent material degradation lead to loss of compliance and further impact on normal aortic physiology.[18]

The impact of graft presence also may have deleterious effects on cardiac and aortic valve function.[19] Afterload is noted to increase following loss of aortic compliance and the Windkessel effect and can lead to left ventricular hypertrophy or alterations in coronary flow.[19] Increased coronary flow velocity as small as 1 m/s may be associated with as much as a 15% increase in overall cardiovascular risk.[20] Younger patients with aortic root aneurysms are often treated with an aortic-valve-sparing operation.[21] As a result, a rigid and less compliant ascending aorta may increase the rate of degeneration in the native aortic valve.[21] Helder et al[22] noted that the proximity of Dacron to the aortic valve can result in cumulative trauma to the cusps as the valve opens, which has been somewhat mitigated with recent advancements in technique but remains a known complication. Furthermore, lack of compliance and increased hemodynamic pressure may result in dilation of the aortic annulus resulting in aortic insufficiency, especially in patients with an underlying connective tissue disorder.[23]

Given these considerations, the approach to Zone 0 TEVAR requires consideration of the unique patient anatomy, comorbid conditions, and graft location. Especially in the setting of acute presentation, such as with an ascending dissection, the international registry of acute aortic dissection found that 28% of patients were judged unfit for open surgery and were medically managed.[24] When determining candidacy for TEVAR, it is critically important to understand the underlying anatomy and physiology, including anticipated postoperative changes. The adequacy of proximal and distal landing zones, involvement of coronary artery ostia, presence or absence of myocardial infarction, aortic valve insufficiency, and degree of tortuosity of the ascending aorta are all critical when considering an endovascular approach.

Techniques

Given the many potential anatomical variants and considerations, high-quality imaging of the aorta is critical for operative planning and graft selection. Currently, there are no FDA-approved devices for use in the ascending aorta. However multiple techniques, including chimney or periscope techniques, in situ fenestration, and branched grafts have met with some success.[25] There is also evidence that Zone 0 TEVAR is feasible for patients who have undergone prior sternotomies.[26]

Chimney graft deployment involves retrograde catheterization of an aortic branch vessel with subsequent retrograde deployment of a covered stent and thoracic endograft. The technique allows for the branch vessel involved to remain patent and has been demonstrated to result in low rates of complications including postoperative stroke and death.[27] However, the technique is vulnerable to the development of frequent endoleaks due to the configurations of the chimney and thoracic grafts, with rates as high as 18.5%.[27] Thoracic endografts can also be placed with fenestration for branch vessels performed after deployment, by radiofrequency, laser, or sharp means.[28] As opposed to an ex vivo approach, in situ technique decreases the potential for incorrect alignment of fenestrations with branch vessels. Branched endografts for Zone 0 lesions are less readily available, and thus data on this approach are sparse. One small study of 28 high-risk patients by Kudo et al[29] demonstrated a 100% success rate with no perioperative endoleaks or required conversions. However, the report also cited a high perioperative stroke rate of 14.3%, and this has led others to caution against the use of this approach without careful patient selection.[30]

Multiple pitfalls have been identified with using these approaches. For example, the tortuosity of the aorta can make it difficult to advance catheters into the ascending portion, and the multiple large catheters, sheaths, and wires

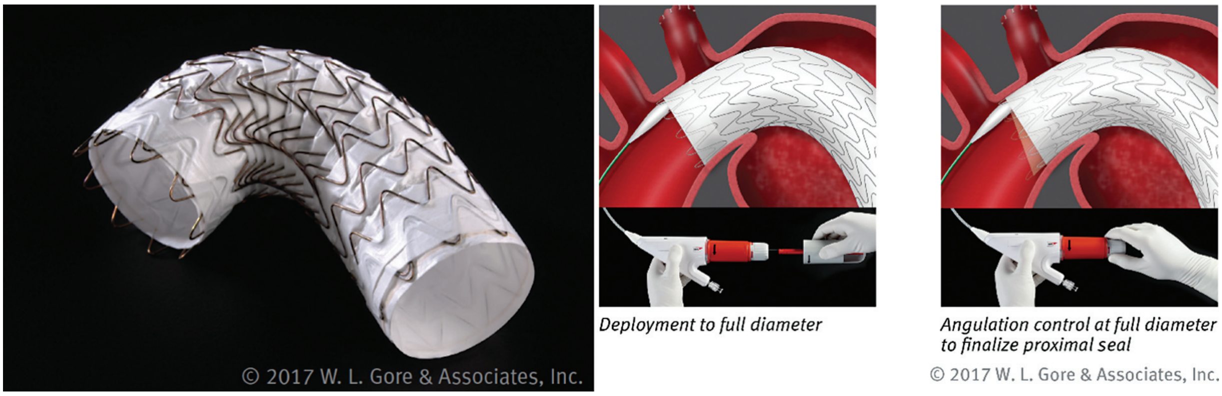


Fig. 2 The GORE TAG endoprosthesis (W.L. Gore and Associates, Flagstaff, AZ). The device is authorized for Zone 2 TEVAR procedures and is approved to maintain flow to the left subclavian artery. It remains investigational for Zone 0 landing sites. TEVAR, thoracic endovascular aortic repair. These images are provided courtesy of the GORE Company.

required leaves the aorta and its branches susceptible to inadvertent injury or occlusion.[31]

Currently Available Graft Options

Most of the experience and data using grafts in this context is extrapolated from the management of aortic dissection. This is because aneurysm development results in focal weakening of the aortic wall intima, and predisposes the patient to subsequent dissection. Multiple manufacturers produce grafts, each possessing unique advantages and disadvantages, namely Cook Medical, GORE, and Terumo. It is critical to emphasize that each of these devices remains investigational in the setting of Zone 0 deployment. There has been no formal data resulting in FDA approval for Zone 0 application, and each device in this setting is therefore being used off-label in the United States. The GORE TAG (W.L. Gore and Associates, Flagstaff, AZ) endoprosthesis is currently authorized for Zone 2 TEVAR procedures and is approved to maintain flow to the left subclavian artery (►**Fig. 2**). It remains an investigational device when employed with a Zone 0 landing site.[32] When evaluated for patients with a Zone 0 aneurysm, it was deployed only when the landing zone was at least 2.5 cm proximal to the brachiocephalic artery.[33] Cook Medical produces the Zenith Alpha Thoracic Endovascular Graft (Cook Medical, Bloomington, IN), which is a two-piece cylindrical endovascular graft composed of a proximal and overlapping distal component. These components are constructed of self-expanding nitinol stents and densely woven polyester material.[34] It is also specifically FDA approved to treat descending aortic pathology, requires a proximal and distal landing zone of at least 20 mm, and can only be deployed in a situation where the aortic diameter is between 20 and 42 mm. As a result, its applicability in Zone 0 situations remains very limited, with no current data demonstrating its efficacy nor risk profile. However, Cook Medical is currently developing a dedicated stent-graft for the ascending aorta, called the Zenith Ascend (Cook Medical, Bloomington, IN), which has shown promising early results in selected high-risk patients (►**Fig. 3**).[35] Terumo Aortic manufactures a recently approved family of devices commercially known as the Relay (Terumo, Tokyo, Japan) and

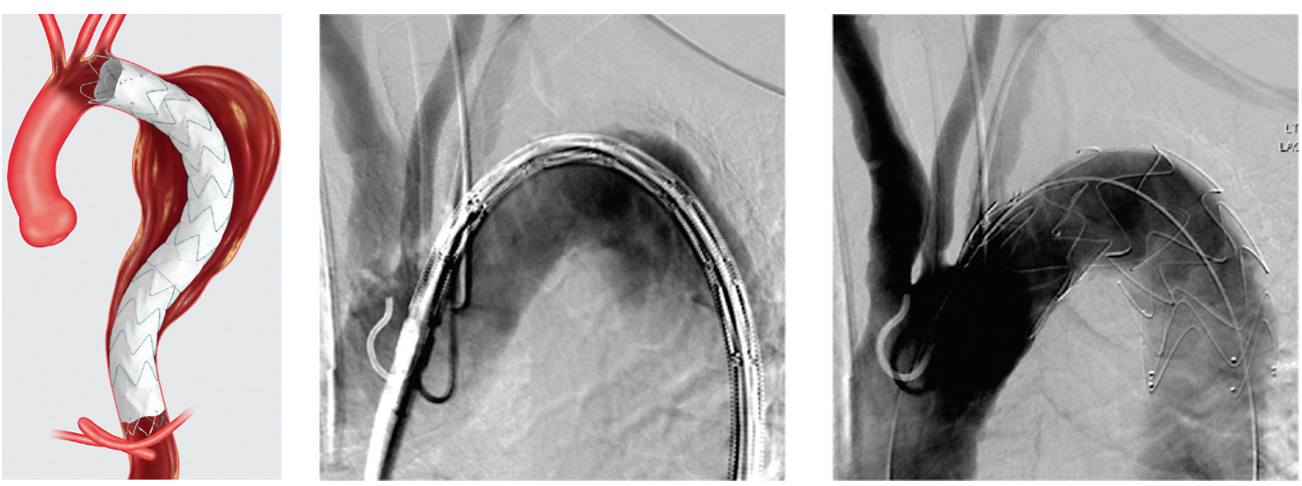
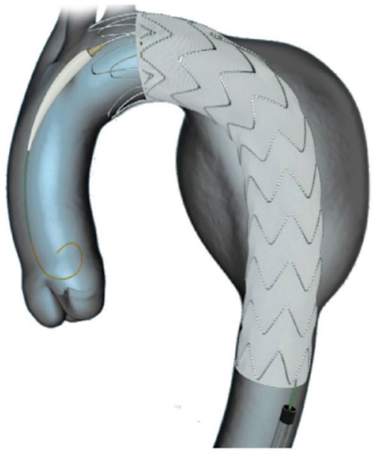


Fig. 3 The Zenith Alpha Thoracic Endovascular Graft (Cook Medical, Bloomington, IN). This is a two-piece cylindrical endovascular graft composed of proximal and overlapping distal components constructed of self-expanding nitinol stents. It is FDA (U.S. Food and Drug Administration) approved to treat descending aortic pathology. Its applicability in Zone 0 situations remains very limited, with no current data demonstrating its efficacy or risk profile. These images are provided courtesy of Cook Medical.



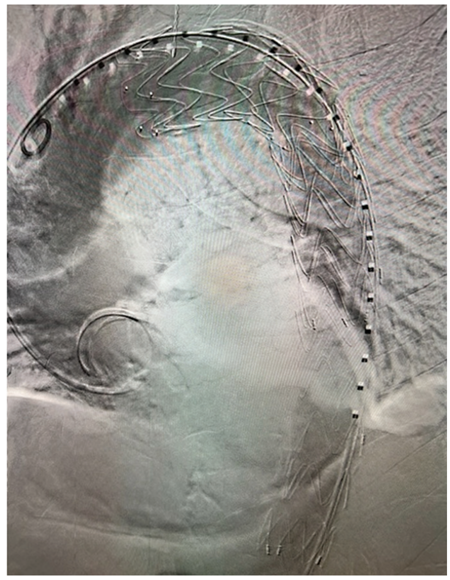


Fig. 4 The Relay (Terumo, Tokyo, Japan) and Relay Pro (Terumo, Tokyo, Japan) are constructed of polyester with the potential for custom-created devices, which may be constructed over a period of 3 weeks based on preoperative imaging. This device is currently undergoing feasibility studies in the United States. TAA, thoracic aortic aneurysm. These images are provided courtesy of the Terumo Company.

Relay Pro (Terumo, Tokyo, Japan) variety of endovascular devices (►**Fig. 4**). These are constructed of polyester with the potential for custom-created devices, which may be constructed over a period of 3 weeks based on preoperative imaging. This device is currently undergoing feasibility studies in the United States.[36] Still, there remains a paucity of data regarding the efficacy and risk profile of these devices for treating Zone 0 pathology.

Complications

Acute aortic dissection, a life-threatening emergency that involves the separation of layers in the aortic wall, can occur as a complication of TEVAR. Dissections of the ascending aorta, or Type A, are often catastrophic, and can be caused by injury to the aorta, malfunction or mispositioning of a stent/endograft, or retrograde blood flow.[37] Mortality of patients with spontaneous Type A dissection has historically been high, with mortality in up to 26% of surgically treated patients.[24] Studies have also identified an association between Type A dissection and proximal landing zone, particularly Zone 0, although the reason remains debated. Proximal stent-graft configuration and oversizing have been posited as plausible contributors, although definitive supportive evidence is needed.[38] For example, in one meta-analysis, Chen et al[38] evaluated 50 publications including a total of 8,969 patients who underwent TEVAR and found a pooled incidence of retrograde Type A dissection of 2.5%, with a mortality rate of 37.1%. They also identified that dissection occurrence rate in Zone 0 was higher than other landing zones. This incidence of dissection is consistent with other reports. In one case series, Czerny et al[39] retrospectively studied 66 patients with Zone 0 pathology who underwent TEVAR and observed an incidence of Type A dissection in 3% of patients. In-hospital mortality for this group was 9% and 5-year mortality was 28%, although 5-year aorta-related mortality was 4%.[39]

There is evidence that the use of rapid cardiac pacing during device deployment may potentially decrease this catastrophic complication. Rapid pacing dramatically decreases ventricular ejection fraction and systolic blood pressure as an endograft expands, and it is routinely used during transcatheter aortic valve replacement to improve proper device placement. In one series by Chassin-Trubert et al,[40] retrograde aortic dissection was significatively reduced when comparing groups of rapidly paced patients to those who were not 17 to 0% ($p < 0.038$). These results are encouraging, although further targeted study is needed.

Currently published rates of mortality in patients presenting with an acute dissection range from 13 to 38% in open surgery in patients with high-risk features, such as advanced age or prior open cardiac or thoracic surgery.[41] As a result, these patients are often denied surgical intervention. This has led to recent studies exploring interest in endovascular management. To date, only the ARISE trial has been conducted evaluating the impact of TEVAR in the context of acute Type A dissection,[42] and no study has been conducted demonstrating the efficacy of one device over another.

Another category of complications includes the potential occlusion of coronary arteries and great vessels, with the associated sequelae. Not only does the inserted endograft itself have the potential to cause constant occlusion or decreased cerebral perfusion if malpositioned, but the large access sheaths and devices used during these procedures (20–26 Fr) may result in intraprocedural occlusion that can lead to limb ischemia and increase the risks of stroke, paraplegia, or spinal cord ischemia.[43,44] Any manipulation of the aortic arch entails a risk of embolization and subsequent stroke.[43] This risk is magnified in patients with proximal seal zones, mural thrombus, or a history of prior strokes[45] and is also increased for Zone 0 and Zone 1 procedures.[46] Spinal cord ischemia occurs in 3 to 10% of TEVAR patients.[31] Identified risk factors include length of coverage of the aorta, prior abdominal aortic surgery, pelvic occlusive disease, perioperative hypotension, and renal failure, as well as unintentional device displacement during deployment.[31,43]

Another complication from these procedures is the migration of endografts. Migration of stents after placement is not uncommon and is influenced by aortic tortuosity, undersizing and rarely the passage of other endografts and delivery system removal.[31,47–49] Migration rates are overall fairly low, with incidence at around 1 to 2.8%, although occasional series have cited rates as high as 19%.[43,47,49,50] The role of aortic zone is unclear in these cases, with some studies identifying an association with migration rates and others that do not support this association.[47,51]

Thoracic Aorta Team

Multiple studies have recently been conducted demonstrating the importance of comprehensive multispecialty evaluation of patients prior to surgical intervention for various cardiac and aortic pathology, including aneurysms, dissection, coronary artery disease, and aortic valve insufficiency.[32,33,42,52] This allows for informed and multidisciplinary decision making along the spectrum of medical management, surgical intervention, and open compared with endovascular intervention where applicable. This is critically important in the setting of Zone 0 TEVAR, especially with an aging patient population who will likely have multiple comorbidities and complex disease. At our own institutions, the routine evaluation of all patients by a team of cardiothoracic surgeons, interventional structural cardiologists, interventional radiologists, and sometimes vascular surgeons has allowed for truly optimized care for each patient, including appropriate risk stratification and treatment selection, interpretation of relevant radiology, predicting prognosis, and medical optimization. These teams may also expand to include other specialists, depending on each individual patient's needs. The use of similar teams is currently a Class I recommendation for patients with aortic stenosis in the decision point for open versus transcatheter aortic valve replacement[53] and will also be requisite for determining candidates amenable to TEVAR versus open aortic surgery.

Conclusion

TEVAR for ascending aortic pathology is becoming more common, and a growing body of research has demonstrated that it can be safely and successfully performed in select cases. This practice is likely to become even more prevalent, and outcomes are likely to further improve, with the development and approval of dedicated grafts for deployment in the ascending aorta. As this frontier in cardiac surgery progresses, the role of the Thoracic Aorta Team will undoubtedly continue to be critically important to optimizing patient outcomes, as it promotes a multidisciplinary and individualized approach to what are often technically complex and unique pathologies.

Conflict of Interest
None declared.

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