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SOCIETY FOR VASCULAR SURGERY CLINICAL PRACTICE GUIDELINES FOR THORACIC ENDOVASCULAR ANEURYSM REPAIR (TEVAR)

(June 11, 2020)

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ABSTRACT

Thoracic aortic diseases, including disease of the descending thoracic aorta (DTA), are significant causes of death in the United States. Open repair of DTA is a physiologically impactful operation with relatively high rates of mortality, paraplegia, and renal failure. Thoracic endovascular aneurysm repair (TEVAR) has revolutionized the treatment of DTA, and has largely supplanted open repair due to lower morbidity and mortality. These Society for Vascular Surgery (SVS) Practice Guidelines are applicable to the use of TEVAR for descending thoracic aortic aneurysm (TAA) as well as other rarer pathologies of the DTA. Management of aortic dissections and traumatic injuries will be discussed in separate SVS documents. In general, there is a lack of high-quality evidence across all TAA pathologies, highlighting the need for better comparative effectiveness research. Yet, large single center experiences, administrative databases and meta-analyses have all consistently reported beneficial effects of TEVAR over open repair, especially in the setting of rupture. Many of the strongest recommendations from the present guideline focus on imaging either prior to, during or after TEVAR and include: 1) in patients considered at high risk for symptomatic TAA or acute aortic syndrome, we recommend urgent imaging, usually Computed Tomography Angiography (CTA) due to its speed and ease of use for pre-operative planning. Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate), 2) if TEVAR is being considered, we recommend fine cut (less than or equal to 0.25 mm) CTA of the entire aorta, as well as the iliac and femoral arteries. CTA of the head/neck is also needed to determine the anatomy of the vertebral arteries. Level of recommendation: Grade 1 (Strong), Quality of Evidence: A (High), 3) we recommend routine use of three-dimensional centerline reconstruction software for accurate case planning and execution in TEVAR. Level of recommendation: Grade 1 (Strong), Quality of Evidence: B

(Moderate), and 4) we recommend contrast-enhanced CT scanning at one and 12 months after TEVAR, and then yearly for life, with consideration of more frequent imaging if an endoleak or other abnormality of concern is detected at one month. Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate). Finally, based on our review, in patients who could undergo either technique (within the criteria of the device's IFU), we recommend TEVAR as the preferred approach to treat elective DTA aneurysms given its reduced morbidity and length of stay, as well as short term mortality. Level of recommendation: Grade 1 (Strong), Quality of Evidence: A (High). Given the benefits of TEVAR, treatment using a minimally invasive approach is largely based on anatomic eligibility, rather than patient-specific factors as is the case in open TAA repair. Thus for isolated DTA, TEVAR should be the primary method of repair in both the elective and emergent setting based on improved short- and mid-term mortality, as well as decreased morbidity.

SUMMARY OF RECOMMENDATIONS

Practice recommendations were made using the GRADE (Grades of Recommendation Assessment, Development and Evaluation) system.[4]

Recommendation 1: In patients considered at low or intermediate risk for a thoracic aortic aneurysm based on their history and physical examination, we suggest chest X-ray as the first radiographic test, as it may identify an alternate diagnosis for symptoms and may obviate the need for additional aortic imaging. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 2: In patients considered at high risk for symptomatic TAA or acute aortic syndrome, we recommend urgent imaging, usually Computed Tomography Angiography (CTA) due to its speed and ease of use for pre-operative planning. Magnetic resonance angiography (MRA) and transesophageal echocardiography (TEE) are also adequate for screening to identify thoracic aortic pathology, but have limited applicability in certain scenarios (discussed further below). **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 3: For elective TEVAR cases, we suggest assessment of left ventricular function by transthoracic echocardiogram in a patient with dyspnea of unknown origin or in a patient with known congestive heart failure with worsening dyspnea. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 4: If TEVAR is being considered, we recommend fine cut (less than or equal to 0.25 mm) CTA of the entire aorta, as well as the iliac and femoral arteries. CTA of the head/neck is also needed to determine the anatomy of the vertebral arteries. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: A (High)**

Recommendation 5: We recommend routine use of three-dimensional centerline reconstruction software for accurate case planning and execution in TEVAR. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 6: We suggest contrast enhanced MRA for pre-operative planning for patients with severe iodinated contrast allergy. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 7: We recommend IVUS use in TEVAR for TAA to assess landing zones when cross-sectional imaging is of poor quality, a more detailed evaluation of landing zones or branch vessel origins are needed, or if a decrease in contrast use is desired. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 8: As hypertension is a modifiable risk factor for the development of aortic aneurysms and is associated with accelerated aortic growth and rupture, we recommend that blood pressure be managed to the adherence of the ACC/ AHA guidelines.[56] **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 9: We recommend interventions for smoking cessation in patients with thoracic aortic pathology, as even passive exposure may increase the risk of aortic rupture. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: A (High)**

Recommendation 10: In patients who could undergo either technique (within the criteria of the device's IFU), we recommend TEVAR as the preferred approach to treat elective DTA aneurysms given its reduced morbidity and length of stay, as well as short term mortality. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: A (High)**

Recommendation 11: We recommend TEVAR in asymptomatic patients with a descending TAA when the maximum aneurysm diameter exceeds 5.5 cm in "low risk" patients with

favorable aortic anatomy. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 12: We suggest using higher aortic diameter thresholds for TEVAR in patients deemed to have a particularly high-risk of death, renal failure or paraplegia from the procedure, where the benefit of treatment is lower than the risk posed by the natural history of the TAA. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 13: Due to the dynamic nature of isolated IMH and its known association with AD, we recommend close observation and hypertension control with follow-up imaging as the initial management of patients with asymptomatic IMH. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 14: We recommend TEVAR in patients with IMH and/or PAU who have persistent symptoms, complications or show evidence of disease progression on follow-up imaging following a period of hypertension control. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 15: We suggest TEVAR in selected cases of asymptomatic PAU who have at-risk characteristics for growth or rupture. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**

Recommendation 16: We suggest TEVAR for symptomatic mycotic/infected TAA as a temporizing measure, but data are lacking demonstrating long-term benefit. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 17: We recommend increasing perfusion pressure via controlled hypertension (mean arterial pressure of greater than 90) as a component of a spinal cord protection protocol in patients at high risk of SCI due to extensive coverage length (>15cm), poor hypogastric perfusion (occluded or significantly stenosed hypogastric arteries), coverage of important collaterals (subclavian/hypogastric arteries). **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 18: We recommend prophylactic CSF drainage for SCI protection in TEVAR cases that are deemed high-risk (covering extensive length of descending aorta, previous aortic coverage, including EVAR or open AAA repair, compromised pelvic perfusion with diseased or occluded common or internal iliac arteries, disease or occluded vertebral arteries, planned left subclavian artery coverage, or deemed high risk by the operating surgeon). **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 19: For elective TEVAR for a TAA where coverage of the LSA is necessary for adequate stent graft seal, we suggest preoperative or concomitant LSA revascularization. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 20: For patients where the anatomy to be treated compromises perfusion to vital structures (see below), we recommend LSA revascularization. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Examples of these circumstances include:

- Presence of a patent LIMA to coronary artery bypass graft
- Termination of the left vertebral artery into the posterior inferior cerebellar artery

- Absent, atretic or occluded right vertebral artery
- Patent left arm arteriovenous shunt for dialysis
- Prior infrarenal aortic operation or EVAR with previously ligated or covered lumbar and middle sacral arteries.
- Planned extensive coverage (≥15cm) of the descending thoracic aorta
- Hypogastric artery occlusion or significant occlusive disease
- Presence of aneurysm disease in the young patient, where future therapy involving the distal thoracic aorta may be necessary

Recommendation 21: For patients with acute thoracic emergencies, where TEVAR is required urgently and coverage of the LSA is necessary, it is suggested that revascularization should be individualized and addressed based on the patient's anatomy and urgency of the procedure. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**

Recommendation 22: We recommend pre-procedural TEVAR planning to include sizing and landing sites before the case to minimize procedural contrast use. If available, intraoperative CTA overlay technology and IVUS should be used to minimize use of contrast. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 23: We recommend non-ionic, hypo-osmolar contrast with attempts at minimizing intra-arterial contrast use, especially in patients at high risk for CIN. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 24: Depending on the patient's corporal density and the capacity of the x-ray equipment available, we suggest diluting contrast in the power injector when possible (typically to 50% or 70%). Adjustments in injection volume and time (faster injection of smaller doses) can

usually compensate when additional visibility is required. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 25: We suggest the use of on-table mapping software options on fixed-imaging X-ray systems, such as roadmapping, CT fusion or overlay reference to aid in locating target landing sites and minimize need for repeated injections. If available, CT overlay capability is extremely useful especially in cases where location and cannulation of branches will be needed. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**

Recommendation 26: To decrease the risk of atheroembolization, we recommend minimizing intraortic wire, catheter and endograft manipulation in the aortic arch and at or above the visceral/renal arteries, especially in patients with significant aortic atheromatous disease or thrombus. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 27: We recommend minimizing the dwelling time of large or occlusive ilio/femoral artery sheaths to decrease the risk of spinal cord ischemia and lower extremity ischemia that can lead to postoperative compartment syndrome or rhabdomyolysis. In cases where a large sheath must be left in place for a prolonged period of time, it can be withdrawn into the external iliac artery to allow antegrade flow into the ipsilateral internal iliac artery. Meticulous postoperative vigilance to detect inadequate lower extremity perfusion and/or compartment syndrome should be routine. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 28: We recommend preemptive SMA stenting with a balloon-expandable stent, in cases of >50% stenosis of the SMA in the following conditions: prior to or after CA coverage or encroachment, TEVAR that is encroaching the SMA origin, or in any patient

otherwise considered as high risk for post-TEVAR mesenteric ischemia. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 29: In anticipation of high risk for CA-territory ischemia (non-visualization of CA collateral branches by CTA or dedicated SMA angiography), we recommend open or endovascular revascularization of the CA before TEVAR. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 30: If an open approach for access is used, we recommend using transverse or oblique incisions when performing open femoral access for TEVAR. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 31: We recommend using ultrasound guidance for percutaneous access to improve procedural success and decrease the rate of major complications. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 32: We recommend that percutaneous access for TEVAR is safe and an acceptable alternative to open common femoral artery exposure if certain anatomic criteria are met (i.e. diameter of common femoral artery, lack of front wall calcium, etc.). **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 33: We recommend the use of iliac conduits or direct iliac/ aortic punctures for TEVAR delivery to facilitate access in patients with small (relative to the chosen device), tortuous or calcified iliac vessels. The decision to perform a conduit should be made in the pre-operative setting, when possible. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 34: We suggest that endoconduits to facilitate access for TEVAR are an acceptable alternative in some cases to an open iliac conduit, but little data comparing them with an iliac conduit or long-term data describing their outcomes over time are available. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 35: We recommend TEVAR over open repair for the treatment of ruptured DTA when anatomically feasible. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 36: We recommend contrast-enhanced CT scanning at one and twelve months after TEVAR, and then yearly for life, with consideration of more frequent imaging if an endoleak or other abnormality of concern is detected at one month. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

1. DEVELOPMENT OF GUIDELINES

The Society for Vascular Surgery (SVS) thoracic endovascular aneurysm repair (TEVAR) Guidelines Committee was created by first soliciting interest among members of the SVS. The committee and Chair were then chosen by the SVS to ensure that the number of authors without documented conflicts of interest was greater than or equal to the number with reported conflicts of interest. Importantly, these guidelines are specific for lesions isolated to the descending thoracic aorta which require coverage of zones 2-6.[6] Those patients with aortic pathology within the aortic arch requiring coverage at or proximal to the left carotid artery (zone 0 or 1) are excluded from these guidelines. Further, while we included management of the celiac artery when requiring coverage for distal seal and fixation, the subject of management of any other visceral arteries was excluded from these guidelines.

An outline was developed by the writing group, which included: the anatomy of the thoracic aorta, aortic pathologies to be covered (i.e.thoracic aortic aneurysms, acute aortic syndromes limited to penetrating aortic ulcer and intramural hematoma, exclusive of traumatic injuries and dissection), diagnostic findings and comparing the advantages and disadvantages of available imaging modalities in various settings. Further topics included the perioperative management of patients with thoracic aortic pathology, specifically mitigation of the perioperative risk of spinal cord ischemia, stroke and renal failure, and evidence-based recommendations regarding the management of the left subclavian and celiac arteries when coverage of those vessels is deemed necessary for “successful” repair. Additional recommendations focused on arterial access, differential management of elective and urgent/emergent TAA, as well as optimal surveillance intervals following TEVAR. Finally, we

considered special problems, including possible volume outcome relationship, related to repair of TAA.

2. DOCUMENT REVIEW AND APPROVAL

The committee developed the practice guideline by assigning members to create primary drafts of each section of the document based on the aforementioned outline, highlighting specific areas where recommendations were needed and appropriate. Each section was then placed into a single document, compiled, reviewed and revised by the writing group, led by the Chair. All guideline recommendations were reviewed by the full committee and finalized via an iterative, consensus process. In considering available treatment modalities to include in the final draft, we evaluated only options currently available to patients and physicians in the United States (U.S.).

The Grades of Recommendation Assessment, Development and Evaluation (GRADE) framework was used for determining the quality of evidence and the strength of recommendation, as previously reported.[4] The quality of evidence is rated as high (A), moderate (B), or low (C). This rating is based on the risk of bias, precision, directness and consistency. The strength of recommendation is graded based on the quality of evidence, balance between benefits and harms, patients' values, preferences, and clinical context. Recommendations are graded as strong (1) or weak (2). The term "we recommend" is used with strong recommendations and the term "we suggest" is used for weak recommendations. Some statements were labeled as good practice statements.[7] These were statements that did not have direct supporting evidence, but had ample indirect evidence and would be considered by many surgeons as surgical principles. Some statements were labeled as implementation remarks. These were technical suggestions that aimed at explaining and implementing the preceding recommendation.

Finally, the SVS Document Oversight Committee peer reviewed the document twice and provided content and methodology expertise. The document was then revised and sent to the Executive Committee and received final endorsement.

3. METHODOLOGY AND EVIDENCE REVIEW

In association with the TEVAR for TAA guideline group document and recommendations, a systematic review and meta-analysis was conducted to evaluate the effectiveness of TEVAR and open repair in patients with isolated TAA.[5] The data sources for this evidence review included PubMed, Ovid MEDLINE, Ovid EMBASE, EBSCO CINAHL, and Scopus, which were searched from each database's inception to January 29th, 2016. Observational studies that compared the two approaches in adults with TAA and reported 30-day mortality or procedure complications were selected. Data were extracted and appraised by two reviewers independently. Random effects meta-analysis was used to estimate odds ratio (OR) and 95% confidence intervals (CI). This document provided evidence that TEVAR reduced the risk of mortality in both intact (OR 0.6; 95% CI, 0.36-0.99) and ruptured (OR, 0.58; 95% CI, 0.38-0.88) settings. In addition, paraplegia risks and pulmonary complication rates were lower with TEVAR compared with open repair for isolated TAA.

a. Glossary/ Definitions of terms and abbreviations used throughout the guideline

   AAA – Abdominal aortic aneurysm(s)

   ABF- Aorto-bronchial fistula

   AD- Aortic dissection

   AEF- Aorto-esophageal fistula

   ASA – Aberrant subclavian artery

CA – Celiac artery

CI – Confidence intervals

COPD - Chronic obstructive pulmonary disease

CTA - computed tomographic angiography

GRADE - Grades of Recommendation Assessment, Development and Evaluation

ICU - Intensive care unit

IFU –Instructions for use (defined by the manufacturer)

IMH - Intramural hematoma

IVUS- Intravascular ultrasound

LIMA- Left internal mammary artery

LSA – Left subclavian artery

MRA - Magnetic resonance angiography

OR – Odds ratio

PAU – Penetrating aortic ulcer(s)

SVS – Society for Vascular Surgery

TAA – Isolated, thoracic aortic aneurysm(s)

TAAA – Thoracoabdominal aortic aneurysm(s) including aneurysms involving the visceral aorta

TAAD- Thoracic aortic aneurysm and dissection

TEVAR – Thoracic endovascular aortic repair

TEE – transesophageal echocardiography

TTE - transthoracic echocardiography

4. EPIDEMIOLOGY AND RISK FACTORS

Thoracic aortic disease is an important public health issue.[1-3,8,9] Although abdominal aortic aneurysms (AAA) and ascending aortic aneurysms are more common, descending TAA and thoracoabdominal aortic aneurysms (TAAA) are not rare, with an estimated incidence of 6-10 cases per 100,000 person-years.[5,10] Olsson examined the prevalence of TAA from 1987 to 2002 in patients with thoracic aortic dissections (AD) or aneurysms in Sweden. Of 14,229 individuals with thoracic aortic disease, the diagnosis was made in 11,039 (78%) before death. The incidence of thoracic aortic disease rose by 52% in men and 28% in women to reach 16.3 and 9.1 per 100,000 per year, respectively. The authors concluded that the prevalence and incidence of thoracic aortic disease were higher than previously reported and has been steadily increasing.[11] The rising prevalence of TAA has been attributed to a number of factors, including improved imaging techniques, an aging population, and increased patient and physician awareness.[12]

A. Population Affected

TAA are primarily a disease of the elderly. The average age of patients with TAA is 65 years at diagnosis, with a male-female ratio of 1.7:1.[10] In contrast, in patients with AAA the mean age is 75 years with a male-female ratio of 6:1.[13,14] TAA clearly have a genetic component with more than 20% of patients having a first-degree relative affected by aneurysm disease.[15-19]

B. Risk Factors for Disease and Rupture

There are many risk factors common to both AAA and TAA patients, including hypertension, smoking, and atherosclerosis in other arterial beds.[10,20-22] Systemic hypertension, especially elevated diastolic blood pressure greater than 100 mm Hg has been

associated with aortic growth and rupture.[23,24] Although most often described as degenerative in etiology, up to 20% of patients have TAA that are the sequelae of chronic aortic dissection. Importantly, for this document, TAA related to chronic type B aortic dissections, and those associated with inherited connective tissue disorders are intentionally excluded and are the subject of future SVS documents.

Natural History and Rupture Rate of TAA

Published data on the natural history of *isolated* TAA is not as readily available as it is for infrarenal AAA, partially related to their much less frequent occurrence. Also, data regarding isolated TAA has historically been combined with TAAA and with aneurysms associated with dissection, which likely each have their own unique natural history, further clouding our knowledge.[25] Importantly, TAAs often occur in patient with multiple co-morbidities, such as hypertension and atherosclerosis, over a wide range of ages. Therefore, patients often succumb to other disease processes, such as cancer or coronary artery disease, highlighting the importance of pre-operative surgical decision-making in the setting of the (largely unknown) natural history of TAA.

Regardless, initial studies from the 1970s by Pressler and McNamara documented that approximately 40% of TAA patients who did not undergo surgical repair died of rupture, whereas 32% died of other cardiovascular diseases, with a mean survival of less than 3 years after TAA diagnosis.[26] During an extended period of observation, more than 90% of patients with unrepaired aneurysms suffered aortic rupture, with 68% of ruptures occurring more than 1 month after the diagnosis.[26,27] A more recent (2002) review found that the 5-year survival rate for patients with a 6.0-cm TAA to be 54%, with a risk for rupture of 3.7%/yr and a risk for death of 12%/yr. They found a similar median survival in patients with untreated TAA of

only 3.3 years.[28] In a natural history study by Crawford and DeNatale of TAA patients who were not candidates for open surgery, the survival rate was just 24% at 2 years, with over half the deaths related to aneurysm rupture. Chronic obstructive pulmonary disease (COPD) was noted in 80% of the subgroup with rupture.[29] Similar studies in patients with small infrarenal AAA have confirmed COPD as a significant risk factor for rupture.[30] Cambria and others followed a series of 57 patients with TAA, including those who were not considered operative candidates. The authors found that an aneurysm >5 cm ($P = 0.05$) and both COPD and chronic renal failure were associated with rupture ($P = 0.06$).[31] Griepp and colleagues studied 165 patients with TAAA who did not undergo surgery, finding that about 20% experience aneurysm ruptured. Significant risk factors included older age, COPD, continued pain and aortic diameter. Patients with AD ruptured at smaller aortic diameters than did those with degenerative aneurysms.[32]

Practice Statement: More research focused on the pathogenesis and clinical care of patients with isolated TAA is required. [25-32] **(Ungraded good practice statement)**

5. THE THORACIC AORTA: ANATOMY AND CLASSIFICATIONS

A. Anatomy of the Thoracic Aorta

The thoracic aorta is divided into the aortic root, ascending aorta, aortic arch, and descending aorta. The size of the thoracic aorta increases from the root to the diaphragm with an average size between 2 and 3 cm, and is approximately 10% smaller in women.[1, 1a] Critically at risk during TEVAR are the multiple spinal cord branches that may be covered by the endograft after emerging as dorsal branches from the intercostal arteries. These critical

branches collateralize as the anterior spinal artery and then travels along the axis of the cord. Multiple vessels supply blood flow to the the spinal cord, including: 1) the subclavian and vertebral arteries, 2) intercostal arteries, 3) the supreme intercostal artery of Adamkiewicz, 4) lumbar arteries, and the 5) iliolumbar branches of the internal iliac (hypogastric) arteries.[33,34]

There are anatomic aortic arch variations. These variations often do not manifest during childhood, but are recognized later in life. Many of these variations are often corrected in childhood if they are incompatible with a normal lifespan. The most common anatomic variations is a "bovine" arch, in which one or more of the great vessels arise from a common trunk. A second common variable, the aberrant right subclavian artery (*arteria lusoria*) arises distal to the left subclavian artery (LSA) and travels posterior to the esophagus to the right arm. The path of these aberrant arteries can vary in their relation to the trachea and esophagus. Other common variants include an aberrant LSA, which often is seen in the setting of a right-sided arch, a thyroidima branch that arises directly from the aortic arch and travels to the thyroid gland. Variations in the origin of the vertebral arteries are also common with the most common variation involving a vertebral artery arising directly from the aortic arch.

B. Classifications of the Zones and Arch

The aorta can be divided into 11 zones, six of which are in the thoracic aorta, which are useful for describing the segment of the vessel and the potential branches that may be covered or replaced during repair (**Figure 1**).[6] The utility of these zones in comparative research is well described in the Society for Vascular Surgery Ad Hoc Committee on TEVAR Reporting Guidelines.[6] Zone 2 is the segment that includes the left subclavian

artery, while Zone 3 is the considered the proximal descending thoracic aorta. Zone 4 is the straight portion of descending aorta. Zone 5 is the segment of the descending thoracic aorta that terminates above celiac artery. The remainder of the aorta lies within the abdomen with zone 6 involving the celiac aorta **(Figure 1).** Aortic arch anatomy also can be critical, especially in the setting of a type III arch.[35]

Practice Statement: Future publications and reporting of TEVAR management should include classifications identifying the location of aneurysms and presence/ absence of PAUs with or without IMH, as well as the zones and arch type to aid comparative studies for the prediction of patient outcomes following interventions. **(Ungraded good practice statement)**

6. THORACIC AORTIC HISTOPATHOLOGY

A. Thoracic Aneurysm and Atherosclerotic Disease

The most common histopathologic feature in TAA is elastic tissue fragmentation and loss of smooth muscle cells resulting in the collection of matrix material in the area of disintegration. These medial degenerative changes are variably associated with wall thinning, loss of elastic and muscle fibers in the aortic media, accumulation of mucopolysaccharide cysts between the fibers and subsequent wall expansion. Common risk factors include hypertension and connective tissue disease. Atherosclerosis on the other hand is typically characterized by intimal plaques composed of variable combinations of fibrous tissue and lipid with calcification. Inflammation manifest by the accumulation of

macrophages and lymphocytes and their secretory products contribute to the progression of disease.

B. Aortic Vasculitides and Inflammatory Diseases

Inflammatory aortitis is characterized by the presence of inflammation of the adventitia and media.[36] Histologic findings may show thickened adventitia with infiltration of adventitia and media with clusters of plasma cells and lymphocytes.

Takayasu (necrotizing) aortitis usually presents as pan-aortitis with granulomatous inflammation and stenosis of the aortic arch and its major branches.[36] Initially, the inflammation is around the *vasa vasorum* and at the medio-adventitial site and advances into the intima. Rapid and severe inflammation can lead to the loss of smooth muscle cells, and may advance to produce aortic arch syndrome, segmental stenosis, occlusion, and/or aneurysms. Disintegration of elastic fibers is prominent as are reactive fibrosis and increased ground substance within the intima. The histologic hallmark of Takayasu aortitis is multifocal medial laminar necrosis rimmed by macrophages and occasional giant cells. Quiescent, or "burnt out" Takayasu's disease is characterized by dense adventitial fibrous thickening and marked medial fibrosis with loss of the normal lamellar structure.

Giant cell arteritis is a systemic vasculitis characterized by focal, transmural granulomatous inflammation with giant cells, intimal thickening, as well as infiltrates of mononuclear cells, neutrophils and eosinophils.[36] This manifestation is called granulomatous arteritis. The key characteristic of granulomatous arteritis is the segmental spread of inflammatory infiltrates, made up of T cells and histiocytes, that result in "skip lesions". Both Takayasu and Giant cell arteritis are large cell vasculitides that appear to the target of new

medical managements that include the use of targeted biologics.[37]

C. Penetrating Aortic Ulcer, Intramural Hematoma and Dissection

Penetrating aortic ulcer (PAU) and intramural hematoma (IMH) are a complex spectrum of aortic disease that are each somewhat unique, but are often an intertwined set of pathologies. This document is not intended to provide a review of aortic dissection as it will be reviewed in separate SVS guidelines.

Briefly, an atherosclerotic plaque can ulcerate and result in a limited dissection or PAU.[38] The ulceration penetrates the internal elastic lamina resulting in hematoma formation within the media. The plaque may precipitate a localized intramedial dissection associated with a variable amount of IMH within the aortic wall, which can spread into the adventitia, forming a pseudoaneurysm or causing rupture. PAU's are typically not aneurysmal, but can occur concurrently or in the absence of an aortic aneurysm, dissection or IMH.

IMH can also develop in apparent isolation in patients with mild or no atherosclerosis. Aortic IMH may represent a variant of dissection, the so-call "dissection in evolution", and is characterized by the absence of an intimal flap, re-entrant tear or double channel with false lumen. It is speculated that the *vasa vasorum* is responsible for IMH, with elevated pressures in the *vasa vasorum* leading to rupture within the aortic wall. Subsequently, progression and eventual rupture into the intima might occur, leading to typical AD. Recent studies examining the *vasa vasorum* have also suggested that hyperplasia leading to chronic, occlusive disease within the aortic wall can lead to chronic medial ischemia and degeneration. The complex pathologies of PAUs and IMH have been well described[39-41] and management decisions can often be complex depending on the clinical

presentation and anatomic location, among other important factors.

D. Mycotic Aneurysms, Aortoesophageal and Aortobronchial Fistulae

A mycotic (or infected) aneurysm is defined as an infectious break in the wall of an artery with formation of a blind, often saccular outpouching that is contiguous with the arterial lumen. Controversy has existed as to the exact mechanism(s) by which primary mycotic TAAs occur, as they may occur due to hematogenous dissemination of microorganisms, direct involvement of the intima or extension from a nearby septic focus. An intimal disruption, such as in atherosclerotic plaque, may be a site of bacterial lodgement, and histological specimens have often demonstrated neutrophilic infiltration and atherosclerotic change in the same aortic wall. Preexisting trauma or aneurysm may also facilitate the onset of the infectious process. Histopathological findings consist of variable elastic fibers degeneration, partial or complete lumen obliteration, compensatory fibrosis with increased thickness of the aortic wall and perivascular chronic infiltrate. It is important to exclude infection in all saccular TAA, as ~93% of mycotic aneurysms have this appearance on CTA.[42]

Aortoesophageal fistula (AEF) is a rare and potentially fatal disorder that often presents after rupture of an aneurysm into the esophagus. The main etiological factor contributing to AEF is aortic disease with over half of cases being secondary to rupture of an aneurysm of the DTA into the esophagus. Aortobronchial fistula (ABF)[43] is also a rare, but potentially life-threatening cause of hemoptysis if not adequately treated. In younger patients, ABF is more frequently seen secondary to surgical repair of congenital heart defects, and aortic coarctation repair. However, most ABFs originate from a descending atherosclerotic

aneurysm or pseudoaneurysm, which causes an erosion of the lung parenchyma or tracheobronchial tree.

F. Coarctation

Aneurysm formation can also develop in patients late after surgical repair after aortic coarctation as an infant and has been reported in numerous patients, with as many as 7% of patients developing "local" aneurysms.[44] These aneurysms may present as false, true or dissecting.[45] Cystic medial necrosis is a common histopathological feature observed in coarctation specimens from surgery or autopsy. This provides a pathologic basis for the formation of aneurysms observed in these patients after balloon angioplasty or repair.

G. Kommerell Diverticulum

This is a bulbous aortic dilatation that is an embryologic remnant of incomplete regression of an embryological aortic arch and is usually located at or near the origin of an aberrant subclavian artery (ASA).[46] Aberrant right and left subclavian arteries (in a right-sided aortic arch) are typically associated with a Kommerell diverticulum. The right ASA can arise distal to the LSA and crosses through the posterior mediastinum behind the esophagus on its way to the right upper extremity. The aberrant vessel has the potential to cause a vascular ring around the trachea and esophagus causing dysphagia and palsy of the recurrent laryngeal nerve due to anatomical position. Aneurysms rarely involve ASA, but they are associated with a high mortality rate if they rupture. The risk for rupture or dissection is variable and ranges from 19 to 53% in some of the case report series.[47] Surgical intervention should be considered when the diameter of the diverticulum exceeds 30 mm, and/or the diameter of the descending aorta adjacent to the diverticulum exceeds 50 mm.[48-50] Recent histological studies demonstrated the presence of cystic medial necrosis in the diverticulum wall, which would

explain the reported high rates of aortic dissection and rupture associated with these diverticuli.

H. Tumors

Primary malignant tumors of the aorta are extremely rare and exhibit enormous histologic heterogeneity.[51] They have been described as three distinctive morphologic types: intraluminal, intimal and adventitial. Most of the cases are sarcomas followed by malignant fibrous histiocytomas. Although intra-aortic biopsy is possible, these tumors are rarely expected or diagnosed before surgical exploration.

Practice Statement: There is a relative lack of high quality, long-term evidence on the use of TEVAR in the setting of arteritis,[52] aortoesophageal[53] and aortobronchial[43] fistulae, coarctation,[45] Kommerell Diverticulum,[54] and tumors.[55] Therefore, no strong recommendations can be made. However, it is recognized that there are numerous institutional and database reports documenting the use of TEVAR in these settings. Likely, especially in the setting of a ruptured thoracic aorta in association with these various pathologies, TEVAR can play a lifesaving role. Finally, there is also likely an advantage to TEVAR in the above-described pathologies in the non-infectious setting over the infectious ones. (**Ungraded good practice statement**)

7. DIAGNOSTIC EVALUATION OF THORACIC AORTIC DISEASE

Thoracic aortic pathology is increasingly found as an incidental finding on studies done for other indications due to the increasing use of cross-sectional imaging. Unlike abdominal ultrasound for screening for abdominal aortic aneurysms, there is no low-cost modality that can be used to image descending thoracic aortic pathology. Thus, there is more reliance on the patient's history, including familial history, as well as physical examination to guide the ordering of radiographic tests to screen for thoracic aortic disease. Genetic testing lends further support for diagnostic imaging. This section will be dedicated to the diagnostic evaluation of a patient with descending thoracic aortic pathology and also will discuss specifics of the history and physical examination, as well as the preoperative workup for patients prior to undergoing TEVAR.

Values and preferences:

The Committee acknowledges the lack of high-quality evidence supporting specific screening strategies: particularly as it pertains to screening intervals. The Committee placed high value on preventing catastrophic vascular events and lower value on screening burdens (including psychological burdens) and costs.

A. History and Physical Examination in the Evaluation of Thoracic Aortic Disease

History of the Patient's Illness

The clinical history should be directed towards determining if the patient is at elevated risk for TAA and should receive further diagnostic evaluation. Most patients are older, with uncontrolled hypertension as a primary risk factor. In younger patients, the clinical history should lead to an evaluation for secondary causes of severe hypertension,

including the use of legal and illicit sympathomimetic drugs, especially in patients with syndromic and non-syndromic genetic defects predisposing to aortic disease. Patients with an inflammatory vasculitis, such as Takayasu disease, giant cell arteritis and Behcet arteritis, should also be considered high risk for developing TAA. The history should also focus on history of previous aortic coarctation repair or a history of significant blunt trauma to the chest (especially those with a rapid deceleration injury). A detailed family history should be taken to elicit a history of familial TAAD. The past surgical history is carefully reviewed with specific attention to prior procedures, including internal mammary artery to coronary artery transposition, upper extremity arterial procedures, and hemodialysis access procedures. The history should also focus on history of aortic valve disease, recent catheterization of the aorta and known TAA, especially in the ascending aorta and aortic arch. Patients may also have symptomatology attributable to compression of adjacent structures in the thorax, such as dysphagia, shortness of breath or hoarseness related to stretching of the recurrent laryngeal nerves, especially in the setting of a large or saccular proximal DTA aneurysm.

Physical Examination

All patients should undergo a detailed physical examination designed to first detect the presence of a genetic syndrome associated with AD or TAA (e.g. Marfan, Loeys-Dietz, Ehlers-Danlos or Turner Syndrome). It is well-known that these patients with genetic syndromes have aneurysms in other anatomic locations, and thus palpation of the abdomen and popliteal fossa for aneurysms should be a routine part of the physical examination.

The history and physical examination should also be focused on identifying other factors, such as angina or COPD,that might preclude the patient from undergoing TEVAR

especially in the setting of general anesthesia. Physical examination should also include a pulse evaluation paying special attention to the presence of palpable femoral pulses for potential access sites to deliver the TEVAR.

Diagnostic Studies and Imaging in Symptomatic Patients

Recommendation 1: In patients considered at low or intermediate risk for a thoracic aortic aneurysm based on their history and physical examination, we suggest chest X-ray as the first radiographic test, as it may identify an alternate diagnosis for symptoms and may obviate the need for additional aortic imaging. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 2: In patients considered at high risk for symptomatic TAA or acute aortic syndrome, we recommend urgent imaging, usually Computed Tomography Angiography (CTA) due to its speed and ease of use for pre-operative planning. Magnetic resonance angiography (MRA) and transesophageal echocardiography (TEE) are also adequate for screening to identify thoracic aortic pathology, but have limited applicability in certain scenarios (discussed further below). **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Implementation remark: The choice of a screening diagnostic study should be based on what is immediately available at that institution.

Practice Statement: If there is a high clinical suspicion for an acute aortic process and the initial study was negative, a second imaging study may be considered while alternative diagnoses are further explored. **(Ungraded good practice recommendation)**

C. Preoperative Workup in Patients Undergoing Open Surgical and Endovascular Repair

The pre-operative cardiac assessments should follow the general recommendation of the ACC/ AHA guidelines.[56]

Emergent or Urgent Repair

In the presence of thoracic aortic disease with rupture, preoperative imaging should be adequate to evaluate whether the patient's anatomy is amenable to endovascular repair or not. This typically consists of CTA of the chest, abdomen and pelvis (from above the clavicles to the femoral heads) to evaluate the proximal and distal seal zones and evaluate for vascular access options. If coverage of the left subclavian artery is planned, CTA through the head and neck is useful to determine the anatomy of the vertebral arteries. In addition, identification of blood or effusions in the thoracic cavity may suggest that the lesion to be treated is acute in nature. CTA may also be useful in the setting of an AEF or ABF in order to determine the best way to approach the patient and determine additional interventions (i.e. esophagectomy, lung resection) that may be needed.

Elective Repair

Preoperative evaluation in the elective setting consists of cardiac risk stratification and includes weighing of the patient's inherent clinical risk with the risk of surgery. This algorithm is well detailed in the 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Non-Cardiac Surgery.[56]

Assessment of Left Ventricular Function

Recommendation 3: For elective TEVAR cases, we suggest assessment of left ventricular function by transthoracic echocardiogram in a patient with dyspnea of unknown origin or in a patient with known congestive heart failure with worsening dyspnea. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Additional Testing

Practice Statement: While there is little supporting data, when trying to determine whether a patient is an open TAA repair or TEVAR candidate in patients with severe COPD, the Committee recommends considering pulmonary function testing (PFT) preoperatively in an attempt to determine baseline pulmonary function, especially if general endotracheal anesthesia is being considered, to determine risk of ventilator dependency postoperatively, and to ultimately guide the choice of anesthesia (general vs local anesthesia). **(Ungraded good practice statement)**

8. **RECOMMENDATIONS FOR IMAGING THE DESCENDING THORACIC AORTA PRIOR TO TEVAR**

The goal of this section is to review commonly available aortic imaging modalities and their respective benefits. The most critical findings and clinical suggestions for optimizing image evaluation will be presented.

A. Chest Radiography

Chest radiographs are particularly prone to observational and interpretive errors. A study analyzing common diagnostic errors, including aortic pathology, in radiology found that 44% of errors occurred when interpreting plain film radiographs, with 49% of these involving chest radiographs.[57,58]

A large aneurysm alters the normal transverse dimension of the mediastinum, and blunts the normal interfaces. Proposed radiographic criteria for a widened mediastinum include a mediastinal width greater than 8 cm or a mediastinal to thoracic width ratio of 0.25 or greater. Other findings include a left apical “cap”, fluid in the left hemothorax from a ruptured aneurysm, widening of the left or right paraspinal line or right paratracheal stripe, an effaced aortic contour, anteroposterior window opacification, tracheal deviation, left mainstem bronchus depression, and deviation of a nasogastric tube to the right of the T4 spinous process[57-59]

TAA are typically located in the posterior mediastinum and associated with the cervicothoracic sign. This sign is based on the fact that the anterior mediastinum does not extend above the clavicles. Therefore, any mediastinal mass extending above the level of the

clavicle with sharply defined borders delineated by an air-soft tissue interface is located in the middle or posterior mediastinum.[60]

Practice Statement: The primary role of chest radiographs in the workup of acute aortic syndromes is the exclusion of other diagnoses. A chest radiograph may be completely normal despite the presence of PAU or IMH. (**ungraded good practice statement**)

B. Computed Tomography Angiography

CTA is the most widely utilized modality for definitive diagnosis of aortic pathologies and has become essential for planning aortic interventions, especially when used in conjunction with post-acquisition image processing and 3-dimensional reconstruction software. This limits radiation exposure and intravenous contrast use since thoracic aortic pathology. The CTA should also include the femoral and iliac arteries, as well as the abdominal aorta in addition to the neck and chest.[61] Advances in imaging techniques, including ECG gated CTA, have been demonstrated to decrease the risk of motion artifact in the thoracic aorta.[61A]

Recommendation 4: If TEVAR is being considered, we recommend fine cut (less than or equal to 0.25 mm) CTA of the entire aorta, as well as the iliac and femoral arteries. CTA of the head/neck is also needed to determine the anatomy of the vertebral arteries.[62,63] **Level of recommendation: Grade 1 (Strong), Quality of Evidence: A (High)**

Pixel spacing for modern CTA is submillimeter (0.5–0.75 mm) with a typically used slice thickness of around 1 mm depending on scanner type and manufacturer. Routine CT

scans are often performed in 3-5mm cuts, but 3D planning for endovascular intervention is best done with ≤2mm cuts.[62] Given the acquisition method on most modern CTA equipment, images can often be reformatted to thinner cuts if the original data set is still available to do so.

Ideally, CTA should provide aortic opacification ≥250 Hounsfield Unit (HU) range at minimum, ≥300 HU uniformly being ideal. There is tremendous institutional variation in how this is achieved. There is further variation based on the patient's body habitus, cardiac output and whether a test dose of contrast versus bolus-tracking software is utilized. In general, fast injection rates and high concentrations of iodine are the general principles that allow for high-quality imaging. A reasonable estimate is that a total of 60 to 140 mL of nonionic iodinated contrast can be injected at a rate of 4 to 6 mL/second. This high injection rate necessitates a power injector, preferably with an 18-20-gauge intravenous line, usually in the antecubital fossa. Central lines are not desirable as they result in artifacts and make timing of the contrast bolus in the thoracic aorta challenging.[64,65]

Multi-planar reconstructions (MPR) allow the aorta to be simultaneously visualized in coronal, sagittal and axial planes. This allows for a more nuanced understanding of the location of branches, aortic curvature and precise identification of seal zones. Centerline reconstructions are utilized to determine exact distances between branch arteries and the length of the thoracic aorta can be measured as well. The diameter of the aorta can be precisely determined with centerline measurements as errors of parallax caused by curvature are virtually eliminated.[65-68]

Recommendation 5: We recommend routine use of three-dimensional centerline reconstruction software for accurate case planning and execution in TEVAR. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

C. Magnetic Resonance Angiography

MRA is not utilized for routine management of thoracic aortic pathology primarily due to the speed and availability of CTA, as well as ease of interpretation. However, MRA can provide morphologic and blood flow information without utilization of iodinated contrast or radiation exposure and therefore can play an important role in the management of the thoracic aorta.

Traditional methods for non-contrast MRA, such as time-of-flight sequences, are being replaced by newer techniques, such as spin-echo and steady state free precession (SSFP) sequences.[69] These provide high spatial resolution, but are limited in their characterization of the aortic wall. Artifact can be present from embolization coils or from certain stent graft metallic components.

Contrast enhanced-MRA is typically performed with the administration of gadolinium, which is administrated intravenously using a power injector, with a dose of 0.1 mmol gadolinium/kg body weight. Images are acquired with a T1-weighted 3-D spoiled gradient-recalled echo (SPGR) sequence, usually during breath-hold. As with CTA, the relationship between contrast administration and image acquisition is crucial. The source images can be reformatted in multiple planes with maximum intensity projections (MIPs) and

volume rendering, and a 3D centerline reconstruction can be generated using the MRA dataset.[70,71]

Recommendation 6: We suggest contrast enhanced MRA for pre-operative planning for patients with severe iodinated contrast allergy. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

D. **Intravascular Ultrasound (IVUS)**

IVUS has become an important adjunct in the endovascular treatment of the thoracic aorta. The presence of thrombus, calcifications and poor aortic wall integrity can also be seen in the setting of PAUs. IVUS adds significant value when treating TAA by reducing intraoperative contrast and radiation use. It also allows for precise intraoperative measurement of distances and diameters of the aorta, adding to the preoperative CTA measurements, especially in angulated aortas.[72,73]

Recommendation 7: We recommend IVUS use in TEVAR for TAA to assess landing zones when cross-sectional imaging is of poor quality, a more detailed evaluation of landing zones or branch vessel origins are needed, or if a decrease in contrast use is desired. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

9. **PERIOPERATIVE MANAGEMENT AND OPERATIVE DECISION MAKING**

A. Perioperative Medical Management

Medical management of patients with thoracic aortic disease has been thoroughly described.[1] This includes control of hypertension, statin therapy/lipid optimization, and smoking cessation. Medical therapy using antihypertensive agents is widely used as a first line treatment in patients with aortic pathology.[74] Blood pressure control is based on anti-impulse therapy to limit the ventricular ejection force and the aortic wall stress, and is especially important in cases of symptomatic aneurysms or acute aortic syndromes. The goal of therapy is to reduce the systolic blood pressure to less than 120 mm Hg and the heart rate to less than 60 beats/min when possible before, during and after TEVAR (see exceptions below in Recommendations for Spinal Cord Protection). This is usually achieved with intravenous beta-blockers (or alpha/beta blockers) as first line therapy. For patients who do not respond to or are intolerant of beta blockers, calcium channel blockers, and/or angiotensin converting enzyme inhibitors/blockers can be used as alternatives or complementaries.[75]

For patients with dyslipidemia, treatment with a statin to achieve a target LDL cholesterol of less than 70 mg/dL is reasonable and may be helpful in controlling the progression of aneurysms.[76] Counseling for smoking cessation, reduction of environmental tobacco exposure, referral to special programs for cognitive behavioral therapy, initiation of pharmacotherapy or, preferably, multimodal management to achieve complete tobacco abstinence, is recommended for patients who have active tobacco use or exposure.[77,78]

Recommendation 8: As hypertension is a modifiable risk factor for the development of aortic aneurysms and is associated with accelerated aortic growth and rupture, we

recommend that blood pressure be managed to the adherence of the ACC/ AHA guidelines.[56]

Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)

Recommendation 9: We recommend interventions for smoking cessation in patients with thoracic aortic pathology, as even passive exposure may increase the risk of aortic rupture.

Level of recommendation: Grade 1 (Strong), Quality of Evidence: A (High)

B. Open Repair *versus* TEVAR for Thoracic Aortic Aneurysm

Until recently, surgical management for elective TAA required major open surgery with a significant risk for perioperative morbidity and mortality. Centers of excellence report impressively low mortality and spinal cord ischemia rates in elective cases of 4.8% and 4.6%, respectively.[79] In tandem, the mortality after open surgical treatment of *ruptured* TAA in highly specialized practices has been reported to be close to 26%.[80] In contrast, the overall mortality rates in the U.S. for elective, open repair of TAA is approximately 22%[81], highlighting the effect that surgeon/center experience has on overall outcomes of these patients. However, data have consistently demonstrated that TEVAR of isolated TAA is a safe alternative to open surgery and is associated with a substantially lower morbidity and mortality, and a shorter hospitalization.[82,83] It is important to recognize that large studies designed to evaluate the long-term (greater than 5 years) outcomes are only recently becoming available.[84] Only one small series of thoracic PAU showed a potential benefit to TEVAR due to a similar long-term survival (~50% at ten years in both groups), with lower morbidity in the TEVAR group, despite being done in patients with a higher number of

preoperative comorbidities.[85] In addition, only recently has there been an attempt to establish a risk scoring system specifically developed to predict mortality in patients undergoing TEVAR.[86,87]

A Cochrane Review compared thoracic stent grafting to open surgery for TAA and concluded "though stent grafting of the thoracic aorta is technically feasible and non-randomized studies suggest reduction of early adverse outcomes, such as paraplegia, mortality and hospital stay, high quality randomized controlled trials assessing clinically relevant outcomes including open conversion, aneurysm exclusion, endoleaks and late mortality are needed."[88,89] In addition, while there are no randomized, controlled prospective trials comparing open and endovascular TAA repair and likely never will be, industry-sponsored trials and registry data (**Table 1**) suggest clinical equipoise in centers experienced in both techniques.[80-83, 86, 90-99]

Recommendation 10: In patients who could undergo either technique (within the criteria of the device's IFU), we recommend TEVAR as the preferred approach to treat elective DTA aneurysms given its reduced morbidity and length of stay, as well as short term mortality.
Level of recommendation: Grade 1 (Strong), Quality of Evidence: A (High)

C. **Indications for Repair**

TEVAR for TAA

Untreated 6.0 cm TAA have a 5-year survival of 54%, yielding a 3.7% per year risk for rupture, and a risk of dying of ~12% per year.[100-101] A prospective database of more than

1600 TAA and AD found that an aneurysmal thoracic aorta grows an average of 0.10 cm per year (0.07 cm for the ascending aorta, and 0.19 cm for the DTA).[25, 100] In saccular aneurysms which may have a higher risk of rupture, TEVAR may be justified at a diameter less than 6.0 cm even though high quality data is not readily available. Data suggesting that lower thresholds for repair in DTA in females is also not readily available as aneurysm disease in the thoracic aorta is rarer than in the abdominal aorta. When making treatment recommendations, the patient's overall medical condition and risk profile should be considered. For patient's at higher risk for elective repair, a larger aortic diameter threshold may be more appropriate when considering their expected surgical complication rate. In addition, data are lacking regarding rapid aneurysm expansion and what size threshold over time is considered accelerated growth. Therefore, TEVAR based on "rapid expansion" should be individualized and should take into account the co-morbidities of the patients, their expected longevity, as well as risk factors for a poor outcome following TEVAR.

Recommendation 11: We recommend TEVAR in asymptomatic patients with a descending TAA when the maximum aneurysm diameter exceeds 5.5 cm in "low risk" patients with favorable aortic anatomy. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 12: We suggest using higher aortic diameter thresholds for TEVAR in patients deemed to have a particularly high-risk of death, renal failure or paraplegia from the procedure, where the benefit of treatment is lower than the risk posed by the natural history of the TAA. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

TEVAR for IMH and PAU

As mentioned previously IMH, PAU and aortic dissection may be similar disease processes along a spectrum of aortic pathologies, or may occur in isolation and therefore a discussion of the use of TEVAR for dissection will be undertaken in another SVS document. Patients with asymptomatic, acute IMH may often be managed conservatively with optimal medical therapy in an intensive care setting. According to a contemporary systematic review of 925 patients with IMH, the predictors of complications include persistent pain, hemodynamic instability, maximum aortic diameter >45 mm, IMH wall thickness >10 mm, presence of ulcer like projections, pleural effusion or hemomediastinum and periaortic hemorrhage.[102] The 3-year aortic related mortality was 5.4% with medical treatment, 23% with open surgery and 7.1% with endovascular therapy.[102] Due to the dynamic nature of IMH and its association with AD ("aortic dissection in evolution"), close observation and hypertension control with follow-up imaging is warranted.

Recommendation 13: Due to the dynamic nature of isolated IMH and its known association with AD, we recommend close observation and hypertension control with follow-up imaging as the initial management of patients with asymptomatic IMH. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 14: We recommend TEVAR in patients with IMH and/or PAU who have persistent symptoms, complications or show evidence of disease progression on follow-up

imaging following a period of hypertension control. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

The natural history and indications for repair in patients with PAU are controversial, but have been found in one series to grow 2 mm/year in their maximal aortic size and length, while only growing an average of 1.2mm/year in depth.[103] The presence of symptoms, an associated IMH or an increase in pleural effusion appear to be risk factors for complications.[85, 104] Treatment with TEVAR is indicated for patients who are symptomatic despite best medical therapy or have an increase in pleural effusion. The threshold for intervention for asymptomatic patients is also controversial. According to one study, PAU depth >10 mm and diameter >20 mm are risk factors for progressive disease.[104]

Recommendation 15: We suggest TEVAR in selected cases of asymptomatic PAU who have at-risk characteristics for growth or rupture. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**

Practice Statement: In the absence of clear and widely accepted parameters, the decision to intervene in asymptomatic patients with IMH and PAUs should be individualized. Asymptomatic patients treated for PAUs in the setting of a maximal aortic diameter less than 5.5 cm or with PAUs less than 10 mm deep/ and or < 20 mm in diameter needs further study. (**Ungraded good practice statement)**

TEVAR for Infected Thoracic Aortic Aneurysms

While the use of TEVAR to treat infected aortic pathologies has often been reported in single or small case series, there is no convincing long-term data to fully support it as a definitive therapy. Although TEVAR can be very effective when used to temporize ruptured infected TAA or life-threatening fistula with a hollow organ (ie aorto-esophageal and aorto-bronchial fistula), patients with this clinical presentation have a high morbidity and mortality regardless of the subsequent management strategy.[105-108] TEVAR may offer a more durable repair if the endograft is pre-treated with antibiotics, such as rifampin but there are very limited data in widely disparate clinical scenarios.[105,109,110]

Recommendation 16: We suggest TEVAR for symptomatic mycotic/infected TAA as a temporizing measure, but data are lacking demonstrating long-term benefit. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

D. Choice of Anesthetic and Monitoring Techniques

Anesthesia

It is technically feasible to perform TEVAR procedures percutaneously under monitored anesthesia care with local anesthesia.[111] Among other benefits of avoiding general anesthesia local anesthesia may theoretically allow for neurological evaluation of the patient's lower extremities.[112]

Arterial lines, large bore venous access, and cerebrospinal fluid (CSF) drains are placed prior to TEVAR. Choice of the necessity for each of these depends on the complexity of the repair, risk of spinal cord ischemia, the planned duration of the procedure and the likelihood of significant blood loss. Other adjunctive techniques performed during TEVAR, such as somatosensory and motor evoked potential monitoring (SSEP and MEP, respectively), rapid arterial pacing or pharmacologically-induced hypotension may be utilized as well.

Practice Statement: Comparative, high quality data regarding the use of local anesthesia versus general anesthesia during the performance of TEVAR is lacking and typically is physician/ hospital dependent. **(Ungraded good practice statement)**

Spinal Cord Protection

SCI can be a devastating complication that profoundly impacts the benefit of the procedure given the higher risk of mortality if it occurs. Although up to 70% of patients will have some functional improvement after suffering SCI, only 38% are reported to return to normal function.[113] Those patients who do not have functional improvement have an abysmal prognosis, with as high as 75% mortality at one year.[113,114]

Given these poor results after SCI, a number of prevention strategies have been employed to mitigate risk, including maintenance of LSA and hypogastric patency[115], staging

strategies for long segment aortic coverage[116], prophylactic CSF drainage, anemia prevention, permissive hypertension, steroid and naloxone therapy[117], burst suppression, permissive hypothermia and hyperoxygenation therapy. Most successful centers employ a multimodal and systematic approach to SCI prevention with detailed protocols on management of spinal drains, multidisciplinary coordination, and rescue procedures for those presenting with delayed SCI.[118]

Techniques for spinal cord protection after thoracic aortic surgery have evolved significantly over the last four decades.[100] Paraplegia after TEVAR limited to the DTA is uncommon (<5%) when compared to open aneurysm repair, despite the observation that TEVAR invariably covers intercostal branches. This highlights the fact that the etiology of spinal cord injury (SCI) after open and endovascular repair is multifactorial and not simply related to cessation of intercostal artery perfusion. However, there are data demonstrating that increased aortic coverage leads to a higher risk of spinal cord injury, supporting the notion that the intercostal arteries are in fact an important source of spinal cord perfusion.[119] Of note, protocols are published describing the complex interaction between mean arterial pressure and spinal cord pressure.[120]

SSEP and MEP permit continuous monitoring of the spinal cord's function, assist in the early detection of SCI and are popular techniques used in high-risk cases during open TAA repair or when patients are undergoing branched/ fenestrated endovascular aneurysm repair[121-122] They are rarely used in the setting of simple TEVAR.

Indications for prophylactic CSF drainage catheter placement during TEVAR are controversial, and CSF drains should be used as only one part of a multi-modal protocol to

reduce the risk of SCI. Some authors recommend selective CSF drain placement for only high-risk patients, while others perform CSF drain placement preoperatively routinely.[122-125] Risk factors for SCI after TEVAR include length of aortic coverage (especially when in excess of 15 cm of the DTA) and the existence of infrarenal aortic pathology.[126] In addition to these anatomical risk factors, chronic renal failure may also be an important risk factor.[127] According to one systematic review, the incidence of SCI after TEVAR with and without prophylactic CSF drain placement was 3.2% and 3.5%, respectively.[128] In contrast, a 2016 systematic review of the use of lumbar drains in open and TEVAR (including 3 randomized trials) concluded that spinal drains prevent early SCI with OR 0.48 (95% CI 0.30-0.76; P=0.002), an absolute risk reduction of 4.5% and number needed to treat of 23 in favor of CSF drainage.[129]

There are many differences in institutional protocols for CSF drain management, varying widely from where to level the drain (earlobe or spinal exit site), draining to a target pressure versus to a target volume, what the baseline pressure should be and the units (centimeters of water or millimeters of mercury) and the maximum amount of fluid that should be drained (per hour, per 4 hours or per day) to avoid intracranial bleeding or herniation.

Other adjunctive methods of SCI risk reduction include the use of routine Narcan and steroids, avoidance of long-acting narcotics, and hemoglobin management strategies, which vary across centers.[123,130] An often-utilized hemoglobin target is >10mg/dL, especially for patients who have developed SCI symptoms. Rescue protocols also exist, which include a further increase in systemic blood pressure to >100mm Hg, a drop in the CSF drain pressure

(often 5mm or 7mm of Hg), transfusion to a target hemoglobin of >10mg/dL,and the use of steroids.[131]

Recommendation 17: We recommend increasing perfusion pressure via controlled hypertension (mean arterial pressure of greater than 90) as a component of a spinal cord protection protocol in patients at high risk of SCI due to extensive coverage length (>15cm), poor hypogastric perfusion (occluded or significantly stenosed hypogastric arteries), coverage of important collaterals (subclavian/hypogastric arteries). **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 18: We recommend prophylactic CSF drainage for SCI protection in TEVAR cases that are deemed high-risk (covering extensive length of descending aorta, previous aortic coverage, including EVAR or open AAA repair, compromised pelvic perfusion with diseased or occluded common or internal iliac arteries, disease or occluded vertebral arteries, planned left subclavian artery coverage, or deemed high risk by the operating surgeon). **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

E. Management of the Left Subclavian Artery, and Vertebrobasilar System

An adequate proximal landing zone requires coverage of the LSA in 26% to 40% of patients undergoing TEVAR.[114,132] In the first U.S. TEVAR regulatory trial[133], all patients underwent prophylactic LSA revascularization prior to TEVAR if the operative plan called

for LSA coverage. Guidelines on LSA revascularization were published in 2009 by the SVS, yet there remains variability in this practice with continued debate on the indications for revascularization.[134] Some surgeons perform revascularization routinely, some selectively and some only perform LSA revascularization if symptoms occur after TEVAR.[134,135] There are four major concerns with coverage of the LSA: SCI, stroke, arm ischemia and vertebrobasilar ischemia.

1. Spinal Cord Ischemia

Understanding the anatomy of the LSA branches and the critical anterior spinal artery is important as the former provides inflow into the latter through multiple pathways. There is general consensus that patients with focal pathologies and treatment with shorter (≤15 cm) stent grafts.[119,132] Data from the EUROSTAR registry, one of the largest series with specific attention to TEVAR and anatomy, demonstrated rates of SCI and stroke as high as 8.4% when there was LSA coverage without revascularization compared to 0% in those patients who underwent prophylactic LSA revascularization (p=0.049).[114]

After reports of lower SCI rates in experimental, sequential and progressive embolization of spinal vessels in animal models[136], many have advocated for staging the coverage of large segments of the aorta to allow for preconditioning, or even purposeful spinal artery embolization prior to extensive TEVAR.[137]

2. Stroke

The incidence of stroke during and identified after TEVAR for TAA generally ranges from 3.2% to 6.2%[138], and may be lethal in one third of these cases.[139] However, this range may vary according to the indication for TEVAR. A recent meta-analysis of the Cook-sponsored multi-center trials demonstrated even lower rates in certain populations of patients with a 30-day stroke rate of 0% in the 56 patients treated for PAU. It was also only 2.4% in the 329 patients treated for TAA.[140]

There is published consensus that coverage of the LSA is associated with higher risk of stroke with TEVAR, despite the fact that the stroke may not always be in the posterior circulation. A series of 285 TEVAR patients showed that coverage of LSA was associated with an 11% stroke rate compared to 3% when it was not covered.[141] The current debate centers on what interventions may reduce this risk. Approaches to prevent stroke include careful manipulation of wires and catheters near the carotid vessels, denitrogenation devices, accurate imaging and positioning of devices, routine LSA revascularization and a thorough understanding of each patient's arch and cerebral anatomy. A systematic review of 27 studies, found a stroke rate of 5.6% associated with LSA coverage and a reduction to 3.1% with LSA revascularization (not statistically significant).[142] In the MOTHER registry of 1,010 TEVAR patients, stroke was 2.2% without coverage of LSA, 9.1% with coverage and no revascularization and 5.1% with LSA coverage and revascularization, supporting routine LSA revascularization.[143] The largest systematic review and meta-analysis supports these findings. A review published in 2017 evaluated the incidence of stroke in 2,594 patients treated with TEVAR and found the incidence in patients where the LSA was uncovered was 3.2% (95% CI 1.0–6.5). When the LSA was covered, but revascularized, the stroke rate was 5.3% (95% CI 2.6–8.6) compared to 8.0% (95% CI 4.1–12.9) when the vessel was covered

without revascularization.[144] Despite these data, selective LSA revascularization strategies are not embraced by some due to concerns for prolonging the procedure, complications of revascularization operations and a perception that patients at elevated risk for subclavian artery ischemia can be identified ahead of time.[145]

There are two scenarios where LSA revascularization should always be considered to reduce perioperative stroke, even in "selective" approaches: most concerning, when a non-revascularized vertebral artery ends in the posterior inferior cerebellar artery, which would risk causing inadequate flow through the Circle of Willis into the posterior cerebral circulation.[146] Additionally, when a dominant left vertebral artery is present (66-75% of patients) in the presence of an absent, atretic or diseased right vertebral artery, non-revascularization of the LSA increases the risk for posterior cerebral ischemia.

3. Arm Ischemia and Vertebrobasilar Insufficiency

Left arm ischemic symptoms may range from none to a frankly threatened limb. Special consideration should be given to LSA revascularization and left arm perfusion in patients at risk of coronary ischemia due to a prior left internal mammary (LIMA) to left anterior descending artery coronary bypass graft, as well as those with existing arteriovenous fistulae in the left arm. Although flow reversal in the vertebral artery is common after LSA coverage, most patients are asymptomatic from this hemodynamic perturbation. However, some may suffer from subclavian steal syndrome and symptomatic vertebrobasilar insufficiency manifested as syncope, diplopia or vertigo. In a recent series, upper extremity ischemia occurred 12-20% of the time after LSA coverage, although less than 40% of patients with symptoms of arm ischemia underwent delayed LSA revascularization.[147,148] Because

presentation of ischemic symptoms of the arm is often delayed, with time to presentation ranging from 2 days to 26 months, revascularization can typically be addressed on a less urgent basis.

Additional Considerations

The Knowledge and Encounter Research Unit performed a systematic literature review and meta-analysis relating to the effect of LSA coverage on the morbidity and mortality of patients undergoing TEVAR.[149] This analysis found that coverage of the LSA without revascularization compared to with revascularization was associated with trends toward increased risk of SCI (OR 2.69; 95% CI 0.75-9.68), anterior circulation stroke (OR 2.58; 95% CI 0.82-8.09), arm ischemia (OR 47.7; 95% CI 9.9-229.3), and vertebrobasilar ischemia (OR 10.8; 95% CI 3.17-36.7). More data has been published since 2009, such as a large single center series where the combined stroke, paraplegia and death rate comparing LSA revascularization to coverage alone is a striking 0% *vs.* 27.9%, $p<0.001$.[150] Additional findings from a 2017 report revealed a higher 30-day stroke rate in cases where the left subclavian was covered, when compared to when it was revascularized (14.3% vs. 1.9%, respectively; P=0.02).[144] The consistent nature of these findings (including another recent meta-analysis mentioned above[151]) all support elective LSA revascularization to lower the risk of stroke and/or paraplegia. Certain limitations persist in the observational nature of these data, and include heterogeneous patients, infrequent and inconsistently defined outcomes of interest and underpowered studies. Large databases often exclude specific

populations, such as trauma patients, or do not capture anatomic variables or staged LSA revascularization.[152]

LSA surgical revascularization is typically performed with a left carotid-subclavian bypass, subclavian to carotid transposition, or carotid-axillary bypass, with similar patency (84%-96% at 5 years) [135,153, 154] for each technique. Occasionally, when the left vertebral artery arises directly from the arch or is very proximal on the LSA, a separate vertebral transposition or bypass is necessary. A transposition is relatively contraindicated when there is coronary artery bypass from the LIMA, as this would cause myocardial ischemia during subclavian artery clamping and, potentially, difficulties mobilizing the LSA cephalad if it is tethered by the LIMA graft.

Complications of LSA revascularization, specifically in the setting of TEVAR, have been studied. From the recent systematic review, the overall incidence of phrenic nerve injury was low at 4.4% (95% CI, 1.6% - 12.20%).[149] Woo et al. examined 42 patients requiring LSA revascularization (5 transpositions, 37 bypasses), and only one patient (2.4%) developed a phrenic nerve palsy.[148] Zamor et al. reported 23 patients who underwent LSA revascularization (21 transpositions, 2 bypasses) prior to TEVAR, and had 2 (8.7%) occurrences of vocal cord paralysis, one of which resolved spontaneously.[135] Wound complications, such as hematoma, lymphatic leak and dissection, have also been reported.[155] A series of 101 LSA revascularizations had a relatively high rate of permanent nerve injuries (9%), along with a 6% lymph leak rate, requiring dietary modification alone.[156] Despite these complications, the series reported only a 2% ischemic stroke rate and 0% SCI after TEVAR.

Despite a net benefit of reduction in SCI and stroke,[157] these complications certainly compromise the effectiveness of TEVAR. Off label and emerging technologies offer the potential to reduce the complications of LSA surgical revascularization. For well over a decade, various techniques have been described for retrograde *in situ* graft fenestration and stenting where the TEVAR graft is punctured with a needle or laser, dilated and a covered stent is inserted to bridge from the fenestration to the LSA[158-160] although the impact of these techniques on the durability of the graft is unknown. Chimney or double barrel stents have also been described, which involve deploying a covered stent in the LSA concomitantly with a thoracic stent graft, preserving flow into the LSA.[161-162] More recently, TEVAR grafts with a branch for the LSA have been developed and are actively being evaluated in clinical trials.[163,164] Industry sponsored trials of fenestrated and branched distal aortic arch endografts, as well as multiple case reports of homemade or physician modified endografts, will likely change the approach to revascularization of the LSA in the future.

Recommendation 19: For elective TEVAR for a TAA where coverage of the LSA is necessary for adequate stent graft seal, we suggest preoperative or concomitant LSA revascularization. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 20: For patients where the anatomy to be treated compromises perfusion to vital structures (see below), we recommend LSA revascularization. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Examples of these circumstances include:

- Presence of a patent LIMA to coronary artery bypass graft
- Termination of the left vertebral artery into the posterior inferior cerebellar artery
- Absent, atretic or occluded right vertebral artery
- Patent left arm arteriovenous shunt for dialysis
- Prior infrarenal aortic operation or EVAR with previously ligated or covered lumbar and middle sacral arteries.
- Planned extensive coverage (≥15cm) of the descending thoracic aorta
- Hypogastric artery occlusion or significant occlusive disease
- Presence of aneurysm disease in the young patient, where future therapy involving the distal thoracic aorta may be necessary

Recommendation 21: For patients with acute thoracic emergencies, where TEVAR is required urgently and coverage of the LSA is necessary, it is suggested that revascularization should be individualized and addressed based on the patient's anatomy and urgency of the procedure. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**

F. Renal Protection Strategies for TEVAR

Acute kidney injury (AKI) occurring during hospitalization or after surgery has one of the highest risks of predicting mortality, especially if the AKI progresses to dialysis. When AKI occurs after TEVAR (~10-15%), it increases the odds ratio of death to almost 10 even

without a need for dialysis.[165,166] Many risk factors for AKI are associated with patients undergoing TEVAR (advanced age, chronic renal failure, diabetes, congestive heart failure, exposure to injectable contrast dye, blood loss, major surgery) and possibly embolic injury from endovascular manipulation within the aorta.

Importantly, contrast-induced nephropathy (CIN) is the third leading cause of AKI in hospitalized patients. While plagued with inconsistent definitions in the literature, its incidence varies between 5%-25%. Factors consistently shown to increase CIN risk include age, diabetes, previous renal disease and escalating doses of contrast. [166-168]

Strategies reported to prevent CIN are also marred by inconsistent reporting standards and patient risk factors[167-168] include: use of IVUS, minimizing the amount of contrast utilized during the operation, pre-hydration with normal saline (effectively increasing the volume of distribution of intravascular contrast), and the use of non-ionic, iso-osmolar contrast agents.[169,170] Research on pretreatment with statins is evolving.[171]

Recommendation 22: We recommend pre-procedural TEVAR planning to include sizing and landing sites before the case to minimize procedural contrast use. If available, intraoperative CTA overlay technology and IVUS should be used to minimize use of contrast. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 23: We recommend non-ionic, hypo-osmolar contrast with attempts at minimizing intra-arterial contrast use, especially in patients at high risk for CIN. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 24: Depending on the patient's corporal density and the capacity of the x-ray equipment available, we suggest diluting contrast in the power injector when possible (typically to 50% or 70%). Adjustments in injection volume and time (faster injection of smaller doses) can usually compensate when additional visibility is required. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)**

Recommendation 25: We suggest the use of on-table mapping software options on fixed-imaging X-ray systems, such as roadmapping, CT fusion or overlay reference to aid in locating target landing sites and minimize need for repeated injections. If available, CT overlay capability is extremely useful especially in cases where location and cannulation of branches will be needed. **Level of recommendation: Grade 2 (Weak), Quality of Evidence: B (Moderate)**

Implementation remark:

In high-risk patients, placing and leaving wires, catheters or sheaths in aortic branches can mark the location of target branches and minimize the need for repeated contrast angiograms. Using a marker catheter inserted through a small diameter left brachial artery sheath, for example, to mark the location of the LSA, or placing a wire or catheter in the celiac artery to mark its location regardless of aortic or thoracic motion may be performed. This strategy can also allow for bail out techniques in case of branch coverage.

Recommendation 26: To decrease the risk of atheroembolization, we recommend minimizing intraortic wire, catheter and endograft manipulation in the aortic arch and at or

above the visceral/renal arteries, especially in patients with significant aortic atheromatous disease or thrombus. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 27: We recommend minimizing the dwelling time of large or occlusive femoral artery sheaths to decrease the risk of spinal cord ischemia and lower extremity ischemia that can lead to postoperative compartment syndrome or rhabdomyolysis. In cases where a large sheath must be left in place for a prolonged period of time, it can be withdrawn into the external iliac artery to allow antegrade flow into the ipsilateral internal iliac artery. Meticulous postoperative vigilance to detect inadequate lower extremity perfusion and/or compartment syndrome should be routine. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

G. Recommendation for Coverage or Occlusion of the Celiac Artery during TEVAR

TAA treated by TEVAR may require covering the celiac artery (CA) in about 4%-6% of cases.[172,173] This can add 1 to 2.5 cm or more of aorta to obtain a distal seal. In addition, the CA is stenotic in approximately 20% of patients, most of these being asymptomatic, presumably due to collateral mesenteric flow.[174-176] Collaterals generally arise from the superior mesenteric artery (SMA), and can be evaluated via selective SMA arteriography. Collateral pathways can also be identified using high-resolution CTA reconstructions (ideally 1 mm cuts or smaller, 16-slice or greater), and the anatomic correlations have been well described.[177] In 94 cases of celiac stenosis (13 with aberrant hepatic artery origins), 95% had

collateral flow from the pancreaticoduodenal (PDA) and 75% from the dorsal pancreatic arteries. These were similar in cases where the hepatic arteries originated from the SMA (92% and 77%, respectively). In addition, flow from the left and right gastric arteries to the hepatic arteries has been documented.[177] Examples of CTA relevant findings that are important to note if CA coverage is planned include significant stenosis of the SMA, an occluded inferior mesenteric artery, a large post-stenotic dilatation of the CA, inability to visualize the PDA or dorsal pancreatic branches. CTA alone may predict ischemia after CA coverage and the need for CA revascularization via open (traditional open surgical management) or endovascular interventions (such as parallel ["snorkel"] stents or fenestrations given appropriate IDE and local experience). However, CTA does not demonstrate dynamic flow and has proven to be incorrect as a single imaging modality in predicting safe coverage of the CA after TEVAR by some authors.[178,179]

If CA coverage occurs without revascularization, a high degree of suspicion for ischemic complications should be maintained post-operatively. Further, ischemia symptoms can range from mild reversible abdominal pain to mild liver enzyme elevation to lethal ischemic injury of the foregut, spleen and/or liver. Balloon occlusion has been reported by some in a very small number of cases (N= 5 each) to determine suitability for CA coverage with unclear sensitivity and specificity.[180] Thus, while reasonable in cases where the results from mesenteric angiography are equivocal, no strong recommendation can be made.

The largest series of CA coverage included only 31 cases. Their protocol was to evaluate CTA for collaterals and if absent, perform an SMA angiogram to evaluate for retrograde flow into the celiac branches. If absent, they occluded the CA with a balloon and repeated the imaging. Notably, they aggressively and preemptively treated SMA stenosis or

cases in which partial SMA coverage occurred during TEVAR (39% of cases) with balloon expandable stents. They documented one case of lethal hepatic ischemia (despite subsequent open bypass), one case of acalculous cholecystitis, and one case of sigmoid colon ischemia thought to be embolic.[181] Another study evaluated 18 TEVAR cases using only angiography (no balloon occlusion) prior to CA coverage. Two patients had documented mesenteric ischemia after CA coverage. One patient had self-limited abdominal pain and two others had elevated white blood cell counts, also self-limited. No elevation in their liver or pancreatic enzymes occurred after TEVAR.[182,183] In another series, CA coverage led to a delayed presentation of iatrogenic chronic mesenteric ischemia despite only "encroaching" the CA and a widely-patent SMA.[184]

If the seal zone includes the CA orifice, then an appropriately sized endograft alone should occlude the origin of the CA, obviating the need for embolization. If absolutely needed, CA embolization should be done carefully and sparingly to avoid inadvertent extension of the embolic material into the common CA trunk and risking foregut ischemia. In cases of TEVAR covering the CA, vigilant postoperative clinical examination and serial laboratory studies should follow the early post-TEVAR period to detect and address foregut and hepatic ischemia as early as possible, to avoid morbid and lethal complications.

Practice Statement: While there is little high quality data, we suggest dedicated SMA angiography via the SMA and/or CA with adequate imaging of the entire SMA/CA mesenteric collateral system to precede TEVAR with intended or high risk for CA coverage. **(Ungraded good policy statement)**

Recommendation 28: We recommend preemptive SMA stenting with a balloon-expandable stent, in cases of >50% stenosis of the SMA in the following conditions: prior to or after CA coverage or encroachment, TEVAR that is encroaching the SMA origin, or in any patient otherwise considered as high risk for post-TEVAR mesenteric ischemia. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 29: In anticipation of high risk for CA-territory ischemia (non-visualization of CA collateral branches by CTA or dedicated SMA angiography), we recommend open or endovascular revascularization of the CA before TEVAR. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Practice Statement: Maintain meticulous vigilance for signs and symptoms of mesenteric and hepatic ischemia early after CA coverage. **(Ungraded good practice statement)**

H. Recommendation for Access during TEVAR.

Importantly, access-related issues remain a common source of morbidity after TEVAR, although these complications are certainly decreasing with the increasing lubricity and decreasing diameter of device delivery systems. In several early multicenter, industry sponsored trials, procedural failures ranged from 0.5 to 2% and were almost all secondary to the inability to advance the device through inadequate iliac arterial systems.[133,185,186] There has been an effort recently to decrease the size of the sheaths and improve the trackability of

TEVAR delivery systems. Nonetheless, a recent study of a lower profile device (sheath sizes 16-20 French) still had a 2% failure to implant secondary to access issues.[187]

Depending upon the size of the graft to be implanted, the outer diameter of delivery systems can be larger than 24 French with some devices. One French is approximately 1/3mm, thus a 24F outer diameter sheath is 8mm in diameter. In the setting of normal vessels with little tortuosity, the vessel may stretch and allow delivery of a sheath that is larger than the actual inner diameter of the vessel. However, increasing tortuosity and/or calcification can reduce the effective inner diameter of the delivery vessel, leading to the need for adjunctive methods of device delivery.

There are several adjunctive measures that can be used to facilitate access in patients with small iliac vessels, including the use of more proximal arteries, as well as open or endovascular conduits.

Femoral

Traditional open femoral exposure during TEVAR involves exposing the common femoral artery (CFA) at the level of the inguinal ligament and establishing sites for proximal and distal control. Unlike endovascular AAA repair, where medium-large diameter sheaths are placed in both groins, TEVAR can usually be accomplished through one femoral artery exposure with the other reserved for diagnostic imaging through a 5 or 6 French percutaneously placed sheath, if needed. When open femoral artery exposure is performed, a transverse or oblique skin incision is favored over the vertical approach in the groin as it is associated with fewer wound complications. Wound complication rates (excluding

hematomas) after endovascular repair with a vertical incision are as high as 18%,[188] while several studies with oblique incisions have reported virtually no infectious wound complications.[189,190]

Percutaneous access of the CFA for TEVAR is also a common approach to access, and is increasing in frequency as surgeons become more comfortable with it.[191] A discussion of the pitfalls and merits of individual closure devices is beyond the scope of this document. However, there have been several techniques described for identification of the femoral artery including access through a small transverse incision[192,193] and ultrasound guidance[194,195] with reported success rates ranging from 92-96%. Ultrasound guidance has become a standard component of percutaneous endovascular access at most institutions as it helps the operator identify and avoid anatomic factors that could lead to failure of closure such as coursing through the inguinal ligament or calcium on the anterior wall of the artery. A recent study reported that the use of ultrasound led to a ten-fold increase in successful percutaneous EVAR procedures compared to those performed without ultrasound ($p=0.03$).[196]

A meta-analysis performed of 3,606 percutaneous arterial access attempts for endovascular aortic repair included 469 percutaneous TEVAR procedures. The overall technical success rate was 94% per arterial access and the groin complication rate was 3.6% with only 1.6% of patients requiring open repair of the groin.[197] The most common complication was groin hematoma (1.8%) followed by pseudoaneurysm (0.7%). Factors that improved successful percutaneous access included ultrasound guidance (96.4% with US vs. 93.5% without, $p=0.02$) and a sheath size <20 French (94.2% <20 French vs. 88.7% $\geq$20 French, $p<0.001$).[197] Other anatomic factors that have been associated with improved success

with a percutaneous approach include a >1 cm segment of mid common femoral artery without anterior calcification, absence of severe scarring in the groin, native arterial access (as opposed to access in graft material) and access vessel diameter >5 mm.[192,194,196,197] Percutaneous femoral access has a safety profile that is comparable to open femoral access in anatomically appropriate patients and both approaches are appropriate for TEVAR, even in the obese.[198]

Recommendation 30: If an open approach is used, we recommend using transverse or oblique incisions when performing open femoral access for TEVAR. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 31: We recommend using ultrasound guidance for percutaneous access to improve procedural success and decrease the rate of major complications. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Recommendation 32: We recommend that percutaneous access for TEVAR is safe and an acceptable alternative to open common femoral artery exposure if certain anatomic criteria are met (i.e. diameter of common femoral artery, lack of front wall calcium, etc.). **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Iliac/ Aortic Access

Multiple industry-sponsored trials of TEVAR have shown that the sizes of the common and external iliac arteries remain a barrier to device delivery in some patients.

Atherosclerotic occlusive disease can be treated with balloon angioplasty and/or using the Dotter technique with serially larger balloons/dilators to facilitate transfemoral delivery of a device, but should be performed carefully with low pressure inflations starting with a small balloon to avoid iliac rupture.

Data from the early FDA and prospective company-sponsored IDE trials showed that iliac conduits were used in 15-21% of patients.[133,185,199] Improvements in the profile and size of delivery systems has decreased this number significantly. A recent industry sponsored trial of a TEVAR device with delivery systems that range from 16-20 French required an iliac conduit in only one (0.9%) patient.[187] This low number was aided by strict exclusion criteria that included iliac tortuosity, calcification, occlusive disease or an inner wall diameter that was not adequate for the required sheath diameter.[187]

A review of the NSQIP database showed that conduits were more likely to be performed in women (15.7% female vs. 5.8% male, $p<0.001$), patients who are current smokers and patients with a previous coronary intervention.[200] The decision to use an iliac conduit should be made during the planning phase of the case as attempts to deliver a large device through clearly inadequate iliac vessels can lead to prolonged operative times and an increase the risk of hemorrhage and death secondary to iliac disruption. The anatomic factors that increase the need for conduits include tortuous iliac arteries, heavy calcification and small vessel size relative to the chosen device.

An open surgical iliac conduit is usually performed with a retroperitoneal exposure of the common iliac artery or distal aorta through an oblique incision in the lower quadrant of the abdomen. The choice of common iliac artery versus the aorta should be made based upon

CTA findings, such as calcification and artery size. A 10 mm prosthetic conduit is best used because it will facilitate delivery of all currently available stent graft systems. The anastomosis can be performed in an end to side or end to end fashion. The conduit can be tunneled to the groin or brought subcutaneously through the abdomen so that it creates an angle that allows for straight delivery. At the completion of the procedure, the conduit can be oversewn near the anastomosis. Alternatively, the distal end can be anastomosed to the common femoral artery to bypass an occluded or injured external iliac artery, while also providing an easy conduit in the future if further interventions are necessary.[201]

Direct puncture of the iliac artery and the aorta has also been described with avoidance of the need for a conduit. Most often these arteriotomies are closed primarily especially in the absence of extensive atherosclerotic occlusive disease.[202]

Recommendation 33: We recommend the use of iliac conduits or direct iliac/ aortic punctures for TEVAR delivery to facilitate access in patients with small (relative to the chosen device), tortuous or calcified iliac vessels. The decision to perform a conduit should be made in the pre-operative setting, when possible. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Endoconduit

In an effort to avoid the potential increased morbidity and operative time associated with a retroperitoneal exposure of the common iliac vessels or distal aorta, the use of

angioplasty and stenting as an endoconduit has been reported.[202] Generally, a 10 mm self-expanding covered stent graft is placed, but others have advocated placing an EVAR limb with at least a 12 mm distal diameter as an endoconduit. This can then be dilated with balloon angioplasty to an appropriate size. Some authors have advocated for intentional rupture of the iliac vessel within the stent-grafted portion given that the vessel wall/atherosclerosis can continue to impede device delivery even after endoconduit placement, especially when there is bulky calcific disease.[203]

In a retrospective series comparing open iliac conduit to endoconduit including 39 patients (23 open conduits, 16 endoconduits), the iliofemoral complication rate was 20% for the entire cohort, but was lower in the endoconduit group when compared to the open conduit (12.5% *vs.* 26.1%). This was not statistically different secondary to small numbers of patients.[204] Other published experiences with this technique include small cohorts of patients.[205-207]

Recommendation 34: We suggest that endoconduits to facilitate access for TEVAR are an acceptable alternative in some cases to an open iliac conduit, but little data comparing them with an iliac conduit or long-term data describing their outcomes over time are available.
Level of recommendation: Grade 2 (Weak), Quality of Evidence: C (Low)

<u>Carotid/Axillary Access</u>

Use of the carotid and axillary arteries to deliver and deploy thoracic stent grafts has been described,[208] but these cases have been reserved for extreme situations where access cannot be obtained from the lower extremities due to iliac or distal aortic occlusion. An approach to the DTA from the arch vessels means that the stent graft will be deployed in an inverted fashion (unless pre-deployed and reinserted into the sheath, which would be an off-label use of the device) and may be associated with an increased risk of neurologic complications due to the wires/sheaths crossing the arch of the aorta.

More commonly, the brachial or axillary arteries are used to facilitate access from below using the so-called "body floss" technique with a brachio-femoral or axillo-femoral wire, in which a wire is passed from the right brachial or axillary artery and brought out the ipsilateral groin, typically by snaring the wire. With tension on both ends, this technique can allow delivery of a stiff device through a very tortuous and otherwise impassable aorta. Care should be taken not to injure the origins of the brachiocephalic vessels with the stiff wire passing through them. A long sheath (typically 5 or 6F) should be used to protect these vessels and can be used to cover the tip of the delivery system on the stentgraft and facilitate delivery using a "push/pull" technique.

Practice Statement: Brachiocephalic access for TEVAR device delivery may be acceptable in situations where trans-femoral/iliac access is not available. However, more data are required to determine whether carotid-axillary artery access for delivery of a thoracic endograft is associated with increased complications. **(Ungraded good practice statements)**

I. Recommendations for Treatment of Symptomatic and Ruptured Thoracic Aortic Aneurysms

Early mortality after open repair of ruptured DTA is high as evidenced by a Swedish study from the pre-endovascular era that reported an in-hospital mortality that approached 100%.[209] The results with TEVAR have been much more promising. A multicenter trial of acute aortic catastrophes showed a mortality of 15% in the ruptured arm.[210] This compared favorably to the results of open repair from the NIS database which had an early mortality of 45%.[211] Indeed, a review of the Medicare database from 2004-2007 showed that the percentage of ruptured DTA patients who were treated with TEVAR increased from 17% in 2004 to 49% in 2007 (a total of 1033 patients treated) with a significant decrease in mortality from 45% in open repair to 24% with TEVAR ($p<0.001$).[212] It is likely that there is an early survival advantage to treating ruptured DTA with TEVAR over open repair.

There appear to be advantages to TEVAR over open repair for DTA beyond survival. A meta-analysis comparing 224 patients from 28 articles showed a significantly lower incidence of perioperative myocardial infarction (11% vs. 3.1%, <0.05) when compared to open repair.[213] In addition, a comparison of 161 patients from 7 hospitals over a 15 year period showed a lower incidence of the composite endpoint of stroke, paraplegia and death in the TEVAR cohort compared to open repair (36.2% vs. 21.7%, $p<0.05$), but no difference was seen in the individual outcomes due to small numbers.[214] Long-term outcomes have been reasonable after TEVAR for ruptured DTA. A review of 21 patients treated with TEVAR

with a median follow-up of over 5 years reported a late mortality of 52% with only one known aortic related death.[215]

Most of the large series evaluating TEVAR for ruptured DTA are from administrative databases, such as Medicare and the National Inpatient Sample, and lack the anatomic granularity that would allow for meaningful comparison of the cohort of patients undergoing each procedure (open TAA repair vs TEVAR). In addition, it is difficult to determine the state of the patient at the time of presentation as it is possible that one approach is favored in stable patients and another is used when a patient presents in extremis. Within these limitations, it appears that TEVAR for ruptured DTA is associated with improved survival and lower morbidity when compared to open repair.[5]

Recommendation 35: We recommend TEVAR over open repair for the treatment of ruptured DTA when anatomically feasible. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

10. SURVEILLANCE FOLLOWING TEVAR

Surveillance after TEVAR is critical to identify endoleaks after initial placement and to evaluate if long-term complications appear, such as migration, aneurysm expansion despite no evidence of endoleak (Type V endoleak, so-called endotension), new endoleaks, device failure (fracture, migration, component separation), stenosis or occlusion. In addition, long-term evaluation may detect signs of graft infection. The most often reported protocol

after TEVAR for aneurysm surveillance is clinical examination and computed tomography scans at 1 month, 6 months and yearly thereafter.[216,217] When TEVAR is placed for emergent indications, earlier evaluation either during hospitalization or within one week of placement may be warranted.[218]

Difficulties in establishing surveillance protocols include variability in reporting of institutional protocols, as well as reported rates of re-intervention versus reporting of new findings in the surveillance protocols. Low re-intervention rates could imply the absence of significant findings on surveillance imaging or a lack of intervention despite the presence of new findings. Conversely, high reported re-intervention rates could reflect either a high rate of significant findings or simply a more aggressive approach to the findings treated conservatively at other institutions. Recent evidence also shows that TEVAR surveillance may be best tailored to the indication for the TEVAR as certain pathologies may warrant more frequent surveillance. A recent publication by Meena et al. evaluated 203 patients treated with TEVAR with follow-up CT scans and demonstrated aortic-related complications in 35% of patients, with sac expansion accounting for 77% of these.[219]

While long-term outcomes are beginning to be reported, patients undergoing TEVAR for DTA aneurysm with straightforward anatomy and who fit within the device IFU criteria rarely require late re-intervention. In a series of 82 patients treated for TAA, only 11% required re-intervention at 60 months follow-up.[216] Indications for re-intervention were Type I endoleaks in about 7%, infection and Type III endoleaks in 1% each. No secondary intervention was performed for aneurysm expansion or endograft collapse.[216]

In contrast, 63 consecutive patients treated in Essen, Germany with TEVAR for PAU were followed for a mean of 46 months. In this experience, 19% required re-intervention due to late endoleaks (6.3%) with the remainder requiring re-intervention secondary to disease progression.[220] A review of the outcomes captured in the Hospital Episode Statistics database in England revealed that 6% of patients treated for intact aortic aneurysms required re-intervention within 30 days following TEVAR.[221] The average time to any re-intervention was 28 months. In contrast to those treated for intact TAA, 33% of patients treated with TEVAR for ruptured aneurysms will require additional intervention at three years.[221]

Concern for long-term, cumulative radiation exposure has been growing, especially when TEVAR is performed in younger patients. Patients treated with TEVAR for intact aneurysms with favorable imaging findings by CTA at 1 and 6 months are unlikely to have any complication in their lifetime that will need re-intervention.[222] Given the good outcomes exemplified in the two scenarios above, it is not surprising that delayed follow-up imaging (>1.5 years) has been shown to be relatively safe in mid-term studies.[223] However, there is an absence of long-term data supporting this approach. Additionally, late stent graft collapse, infection and endograft disruption can occur[224] and late conversion to open repair occurs at an average of 5 years and up to 98 months after initial implantation, suggesting that patients undergoing TEVAR should be followed for life.[106, 225]

Recommendation 36: We recommend contrast-enhanced CT scanning at one and twelve months after TEVAR, and then yearly for life, with consideration of more frequent imaging

if an endoleak or other abnormality of concern is detected at one month. **Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)**

Implementation remarks about surveillance:

1. In cases where the 1-month CT demonstrates morphologic endograft concerns (e.g. "bird beaking", in-folding of endograft, etc.), endoleaks, evidence of sac growth or in high-risk patients (e.g. those treated for PAU or ruptured aortic aneurysms), a repeat CTA with arterial and delayed phase imaging is recommended within 6 months.
2. In cases at low risk for expansion, such as those with a shrinking aneurysm sac and over 3 years of stability, non-contrast CT of the chest may be used to follow aneurysm sac size and component stability.
3. We can neither recommend eliminating TEVAR surveillance, nor can we recommend extending it further than annually given the lack of long-term evidence of safety and due to evidence of aneurysm growth and new endoleaks reported, despite a previously-sealed aneurysm.

11. SPECIAL TAA CONSIDERATIONS

Guidelines for hospital privileges have been established for TEVAR by the SVS.[226] Calligaro and others[227] suggested that the requirements for TEVAR include full basic privileges with either: 1) 10 TEVARs within the last 2 years or 2) less than this minimum for surgeons with a robust EVAR experience, defined as 25 EVARs with 12 as the primary

operator. Trainees should also be able to manage complex aortic patients, as well as perform adjunctive procedures, including iliac conduits and carotid-subclavian bypass grafting.

The relationship between volume and outcomes has been explored for TEVAR,[228-230] and the data supporting or refuting such a relationship is poor, mainly because it is typically underpowered and the data is heterogenous including EVAR and TEVAR or uses TEVAR to treat multiple pathologies (i.e. aneurysm and dissection). One study using Medicare claims database from 1999 to 2007 documented a mortality rate for TEVAR in low volume centers of 9-10%, whereas mortality was 7% in high volume TEVAR centers. Despite these gross mortality differences, a multivariable model for mortality failed to show volume as a predictor (p=0.328).[228] A second study using MEDPAR data also found no association between TEVAR volume and mortality.[229] Finally, a study using a MEDPAR dataset in 10,000 patients undergoing TEVAR found no clear relationship between hospital volume effect and survival. However, these same practitioners suggested that using a mixed-effect Cox model demonstrated there was an "independent hospital effect" associated with certain hospitals with a death 50% of what occurred at other hospitals.[230] These data suggest at present, no clear conclusion can be drawn between hospital volume and outcomes following TEVAR. Importantly, even less data is available to examine the role of individual clinician TEVAR volume and outcomes.

12. CONCLUSIONS

Thoracic endovascular aortic repair (TEVAR) is used to treat a myriad of aortic pathologies. While there are no randomized, controlled trials comparing open and

endovascular DTA repair directly and likely never will be, consensus documents, large administrative datasets, and meta-analyses have strongly suggested that TEVAR for isolated descending thoracic aortic aneurysms should be the primary method of repair in both the elective and emergent setting based on improved short- and mid-term mortality, as well as morbidity.

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81. **"03**an JA, **%**imick JB, Henke **5@**, H-<er **)***, ***tan'e$** J", **;2**c**1**-rc**1** G . ***-r,**ica' treatment **0/** intact t**10**rac**0**a<.**0**mina' a**0**rtic ane-r**$**sms in t**1**e ;nite. ***tates**4 **10**s**2**ita' an. s-r,e**0**n **60'**-me**:** re'ate. **0**-tc**0**mes. J**0**-rna' **0/ 6**asc-'ar s-r,er**$**. 200373786941169:74.
82. **!** akar**0**-n **! ***, **%**i"a**60**- **&%**, @ee ***)**, ***icar. G, **"1**aik**0/ &**, Ba**6**aria J, et a'. **&n.06**asc-'ar treatment **0/** t**10**racic a**0**rtic ane-r**$**sms4 res-'ts **0/** t**1**e **21**ase ++ m-'ticenter tria' **0/** t**1**e G**B &)**AG t**10**racic en.**02**r**0**st**1**esis. J**0**-rna' **0/ 6**asc-'ar s-r,er**$**. 200574181941:9.
83. HeiAmen H, **%**e<'ier +G, **! 0"** FL, **%0**ssc**1**e @ **!** , **6**an .en Ber, J", **B6**ert**00**m **))**, et a'. **&n.06**asc-'ar stent:,ra**/**tin, **/0**r .escen.in, t**10**racic a**0**rtic ane-r**$**sms. **&**-r**02**ean A**0**-rna' **0/** car.i**0:** t**10**racic s-r,er**$**. 200272181945:9.
84. **"1**i- **5**, G**0'**.st**0**ne AB, ***c1**a**//**er J **!** , Lin,a'a B, **!** i"er **%"**, **!** itc**1**e" ***, **? 00 J**J, Fisc**1<**ein **! 5**, %ake **! %**.. **&n.06**asc-'ar **#**ers-s **B2**en e**2**air **0/** +ntact **%**escen.in, **)10**racic A**0**rtic Ane-r**$**sms. J Am **"0" "**ar.i**0'**. 20197738694643:651.

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86. **%a$ "**, B-cken**1**am **)**. **&n.06**asc-'ar re**2**air **0/** t**1**e t**10**racic a**0**rta4 **2**re.ict**0**rs **0/** 30:.a**$** m**0**rta'it**$** in **2**atients **0**n t**1**e =e**3 C**ea'an. **)10**racic A**0**rtic ***t**ent **%**ata<ase 8=**C)**A***9**. **&**-r**02**ean J**0**-rna' **0/ #**asc-'ar an. **&n.06**asc-'ar ***-r,**er**$**. 200973782941160:5.
87. **5**atters**0**n B**B**, **#i.a':%**iez A, H**0**'t **5**J, ***ca'i ***)**, Beck A**?** , **)10**m**2**s**0**n **! !** . **5**re.ictin, **!** i.:term A**":**ca-se **! 0**rta'it**$** in **5**atients ;n.er,**0**in, **&**'ecti**6**e **&n.06**asc-'ar e**2**air **0/** a **%**escen.in, **)10**racic A**0**rtic Ane-r**$**sm. Anna's **0/** s-r,er**$**. 2016 **%**ec7 264 8694 1162:67.
88. A<ra**1**a +, **0**ma,n**0**'i **", ! 0**nte.**0**ri A, **"**ir**0**cc**1**i . **)10**racic stent ,ra**/**t **6**ers-s s-r,er**$ /0**r t**10**racic ane-r**$**sm. **"0**c**1**rane **%**ata<ase ***$**st e**6**. 2016869**4"%**006796.
89. A<ra**1**a +, **0**ma,n**0'**i **", ! 0**nte.**0**ri A, **"**ir**0**cc**1**i . **)10**racic stent ,ra**/**t **6**ers-s s-r,er**$ /0**r t**10**racic ane-r**$**sm. **)1**e **"0**c**1**rane Li<rar**$**. 2009.
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91. **!** ats-m-ra J***, **"**am<ria **5**, **%**ake **! %**, **! 00**re **%**, ***6**enss**0**n LG, ***n$.**er ***, et a'. +nternati**0**na' c**0**ntr**0"**e. c'inica' tria' **0/** t**10**racic en.**06**asc-'ar ane-r**$**sm re**2**air **3**it**1** t**1**e **C**enit**1)Q**2 en.**06**asc-'ar ,ra**/**t4 1:**$**ear res-'ts. J**0**-rna' **0/ 6**asc-'ar s-r,er**$**. 200874782942**4**7:57. e3.
92. Fairman **!** , "ria.**0** F, Far<er **!** , @**30'**ek **", ! e1**ta **!** , **?1**ite , et a'. **5i60**ta' res-'ts **0/** t**1**e me.tr**0**nic **6**asc-'ar ta'ent t**10**racic stent ,ra**/**t s**$**stem4 t**1**e #AL**B** tria'. J**0**-rna' **0/ 6**asc-'ar s-r,er**$**. 200874883945**4**6:54.
93. **"0**se"i J***, Le **!** aire ***A, **! i"er "", 3r., *c1**mitt'in, C"**, @**0**ks**0$ "**, **5**a,an J, et a'. **! 0**rta'it**$** an. **2**ara**2'**e,ia a**/**ter t**10**rac**0**a<.**0**mina' a**0**rtic ane-r**$**sm re**2**air4 a risk **/**act**0**r ana'**$**sis. Ann **)10**rac ***-**r,. 200076982944**0**9:14.
94. **5**atters**0**n B**B**, **#i.a':%**iez A, H**0'**t **5**J, ***ca'i ***)**, Beck A**?** , **)10**m**2**s**0**n **! !** . **5**re.ictin, **!** i.:term A**":**ca-se **! 0**rta'it**$** in **5**atients ;n.er,**0**in, **&**'ecti**6**e **&n.06**asc-'ar e**2**air **0/** a **%**escen.in, **)10**racic A**0**rtic Ane-r**$**sm. Ann ***-**r,. 201672648694**1**162:7.

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101. %a6ies , G0'.stein LJ, "0a.$! A,)itt'e *L, izz0 JA, @02/ G*, et a'. Jear'$ r-2t-re 0r
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102. &6an,e'ista A, "zern$! , =iena<er ", *c1e2ens ! , 0-ssea- H, "a0 5, et a'. +nter.isci2'inar$
eL2ert c0nsens-s 0n mana,ement 0/ t$2e B intram-ra' 1aemat0ma an. 2enetratin, a0rtic -'cer.
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1ist0r$ an. 0-tc0mes /0r t10racic an. a<.0mina' 2enetratin, a0rtic -'cers. J #asc *-r,.
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104. Gana1a F, ! i"er %", *-,im0t0 @, %0 J*, ! inami,-c1i H, *ait0 H, et a'. 5r0,n0sis 0/ a0rtic
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ana'$sis. "irc-'ati0n. 2002710683943 42:8.
105. A.kiss0n "%, B'.en<-r, ? A, Be"i &#, Harris A*, ? a'ser & ! , Hakaim AG.)reatment 0/ a
m$c0tic .escen.in, t10racic a0rtic ane-r$sm -sin, en.06asc-'ar stent:,ra/t 2'acement an. ri/am2in
in/-si0n 3it1 20st02erati6e as2irati0n 0/ t1e ane-r$sm sac. #asc &n.06asc-'ar *-r,. 20117458894765:
8.
106. %a6is F ! , ! i"er %J, =e3t0n %, Ar$a *, &sc0<ar GA. *-ccess/-' treatment 0/ a m$c0tic
m-'ti/0ca' t10rac0a<.0mina' a0rtic ane-r$sm as a 'ate seG-e'ae 0/ intra6esica' <aci"-s "a'mette:
G-erin t1era2$4 case re20rt an. 'iterat-re re6ie3. Ann #asc *-r,. 20157298494840 e9:13.
107. ! ezzett0 L,)re22ie.i &, *c0rs0ne L, Giac02-zzi *, 5eran.ini *, ! acri ! , et a'.)10racic
A0rtic 5se-.0ane-r$sm a/ter &s021a,ea' 5er/0rati0n an. ! e.iastinitis "a-se. <$ Acci.enta'
+n,esti0n 0/ a ! -tt0n B0ne4 A "ase e20rt 0n *ta,e. &n.0sc02ic an. &n.06asc-'ar)reatments. Ann
#asc *-r,. 20167304307 e15:9.
108. *etacci ", .e %0nat0 G, *etacci F. &n.0,ra/ts /0r t1e treatment 0/ a0rtic in/ecti0n. *emin
#asc *-r,. 20117248494242:9.
109. &sc0<ar GA, &'ias0n JL, H-rie J, Ar$a *, ecten3a'. J&, "0'eman % ! . i/am2in s0akin,
.acr0n:<ase. en.0,ra/ts /0r im2'antati0n in in/ecte. a0rtic ane-r$sms::ne3 a22'icati0n 0/ a time:
teste. 2rinci2'e. Ann #asc *-r,. 20147288394744:8.

110. Hene,1an &, *in,1 =, *tarnes B?. *-ccess/-' emer,ent en.06asc-'ar re2air 0/ a r-2t-re. m$c0tic t10racic a0rtic ane-r$sm. Ann #asc *-r,. 201572984948 43 e1:6.
111. 6an %0r2 !, Gi'<ers !, La-3ers 5, #an *c1i' 5&, Hen.riks J !. L0ca' Anest1esia /0r 5erc-tane0-s)10racic &n.06asc-'ar A0rtic e2air. A0rta 8*tam/0r.9. 201674839478:82.
112. H0,en.00rn ?, *c1'0sser FJ, ! -1s B&, 502esc- ? !. *-r,ica' an. anest1etic c0nsi.erati0ns /0r t1e en.06asc-'ar treatment 0/ r-2t-re. .escen.in, t10racic a0rtic ane-r$sms. "-rr B2in Anaest1esi0'. 2014727819412:20.
113. %e*art @, *ca'i *), Feez0r J, H0n, !, Hess 5J, Jr., Bea6er) !, et a'. Fate 0/ 2atients 3it1 s2ina' c0r. isc1emia c0m2'icatin, t10racic en.06asc-'ar a0rtic re2air. J #asc *-r,. 20137588394635: 42 e2.
114. B-t1 J, Harris 5L, H0<0 , 6an &2s , "-$2ers 5, %-iAm L, et a'. =e-r0'0,ic c0m2'icati0ns ass0ciate. 3it1 en.06asc-'ar re2air 0/ t10racic a0rtic 2at10'0,$4+nci.ence an. risk /act0rs. a st-.$ /r0m t1e &-r02ean "0"a<0rat0rs 0n *tent(Gra/t)ec1niG-es /0r A0rtic Ane-r$sm e2air 8&; B*)A 9re,istr$. J #asc *-r,. 20077468694 1103:10.
115. &a,'et0n !J, *1a1 *, 5etk0se6ek %, ! astracci) !, Green<er, @. H$20,astric an. s-<c'a6ian arter$ 2atenc$ a//ects 0nset an. rec06er$ 0/ s2ina' c0r. isc1emia ass0ciate. 3it1 a0rtic en.0,ra/tin,. J #asc *-r,. 2014759819489:94.
116. B>"a"a,1an A, ! astracci) !, &a,'et0n !J. *ta,e. en.06asc-'ar re2air 0/ t10rac0a<.0mina' a0rtic ane-r$sms 'imits inci.ence an. se6erit$ 0/ s2ina' c0r. isc1emia. J #asc *-r,. 201576182 94347: 54.
117. Ac1er "?, ?$nn ! !, H0c1 J , 502ic 5, Arc1i<a'. J,)-rni2see. ?%. "0m<ine. -se 0/ cere<ra' s2ina' /'-i. .raina,e an. na'0L0ne re.-ces t1e risk 0/ 2ara2'e,ia in t10rac0a<.0mina' ane-r$sm re2air. J #asc *-r,. 19947198294236:46.
118. Ac1er ", Ac1er "?, ! arks &, ?$nn !. +ntra02erati6e ne-r02r0tecti6e inter6enti0ns 2re6ent s2ina' c0r. isc1emia an. inA-r$ in t10racic en.06asc-'ar a0rtic re2air. J #asc *-r,. 2016763869 41458: 65.

119 ! e'issan0 G, @a1'<er, A, Bert0,'i0 L, "1iesa . &n.06asc-'ar eLc'-si0n 0/ t10racic a0rtic ane-r$sms 3it1 t1e 1: an. 2:c0m20nent Cenit1)Q2 TAA en.06asc-'ar ,ra/ts4 ana'$sis 0/ 2:$ear .ata /r0m t1e)Q2 2i60ta' tria'. J &n.06asc)1er. 2011 J-n7188394338:49.

120. B0<a.i"a JL, ?$nn !,)e/era G, Ac1er "?. L03 inci.ence 0/ 2ara2'e,ia after t10racic en.06asc-'ar ane-r$sm re2air 3it1 2r0acti6e s2ina' c0r. 2r0tecti6e 2r0t0c0's. J #asc *-r,. 2013 J-n7578694 1537:42.

121. @e$1ani @, ! i"er "", 3r., &strera AL, ?e,r$n), *1ein<a-m , *a/i HJ. Ana'$sis 0/ m0t0r an. s0mat0sens0r$ e60ke. 20tentia's .-rin, t10racic an. t10rac0a<.0mina' a0rtic ane-r$sm re2air. J #asc *-r,. 2009749819436:41.
122. Ban,a 5#, B.eric1 G*, eis .e *0-za L, H0/er J, "azares G0nza'ez ! L, 5-'i.0 J=, et a'. =e-r0m0nit0rin,, "ere<r0s2ina' F'-i. %raina,e, an. *e'ecti6e ;se 0/ +'i0/em0ra' "0n.-its t0 ! inimize isk 0/ *2ina' "0r. +nA-r$ %-rin, "0m2'eL &n.06asc-'ar A0rtic e2air. J &n.06asc)1er. 2016723819 4139:49.
123. Ac1er ", Ac1er "?, ! arks &, ?$nn !. +ntra02erati6e ne-r02r0tecti6e inter6enti0ns 2re6ent s2ina' c0r. isc1emia an. inA-r$ in t10racic en.06asc-'ar a0rtic re2air. J #asc *-r,. 2016763869 41458: 65.
124. Arna0-takis %J, Arna0-takis GJ, Bea-'ie- J, A<-'arra,e "J, L-m J?, B'ack JH, 3r.. es-'ts 0/ a.A-ncti6e s2ina' .raina,e an.(0r 'e/t s-<c'a6ian arter$ <$2ass in t10racic en.06asc-'ar a0rtic re2air. Ann #asc *-r,. 2014728819465:73.

125. @eit1 "J, Jr., 5assman ! A, "ari,nan ! J, 5armar G ! , =a,re *B, 5atters0n ! A, et a'. 5r0t0c0' im2'ementati0n 0/ se'ecti6e 20st02erati6e '-m<ar s2ina' .raina,e a/ter t10racic a0rtic en.0,ra/t. J #asc *-r,. 201275581941:8.
126. Bis.as), 5an-cci0 G, *-,im0t0 ! ,)0rse"0 G, A-stermann ! . isk /act0rs /0r s2ina' c0r. isc1emia a/ter en.06asc-'ar re2air 0/ t10rac0a<.0mina' a0rtic ane-r$sms. J #asc *-r,. 201576186941408:16.
127. ;"er$ B?, "1e-n, A), Fairman ! , Jacks0n B ! , ?00 &J, Ba6aria J, et a'. isk /act0rs, 0-tc0mes, an. c'inica' mani/estati0ns 0/ s2ina' c0r. isc1emia /0"03in, t10racic en.06asc-'ar a0rtic re2air. J #asc *-r,. 20117548394677:84.
128. ?0n, "*, Hea'$ %, "annin, ", "0//e$ J", B0$'e J , ?a's1 * . A s$stematic re6ie3 0/ s2ina' c0r. inA-r$ an. cere<r0s2ina' /'-i. .raina,e a/ter t10racic a0rtic en.0,ra/tin,. J #asc *-r,. 201275685941438:47.
129. @1an = , *ma"e$ C, =es6ick "L, Lee *L, ! ic1ae' L ! , 2n..)1e -se 0/ '-m<ar .rains in 2re6entin, s2ina' c0r. inA-r$ /0"03in, t10rac0a<.0mina' a0rtic ane-r$sm re2air4 an -2.ate. s$stematic re6ie3 an. meta:ana'$sis. J =e-r0s-r, *2ine. 20167258394383:93.

130. *ca'i *), @im ! , @-<i'is 5, Feez0r J, Gi'es @A, ! i"er B, Fatima J, H-<er)*, Berce'i *A, Back ! , Beck A?. +m2'ementati0n 0/ a <-n.'e. 2r0t0c0' si,ni/icant'$ re.-ces risk 0/ s2ina' c0r. isc1emia a/ter <ranc1e. 0r /enestrate. en.06asc-'ar a0rtic re2air. J #asc *-r,. 2018 Fe<7678294409:423

131. &strera AL, *1ein<a-m , ! i"er "", Azizza.e1 A, ?a'kes J", Lee)J, et a'. "ere<r0s2ina' /'-i. .raina,e .-rin, t10racic a0rtic re2air4 sa/et$ an. c-rrent mana,ement. Ann)10rac *-r,. 200978881949:15.
132. Feez0r J, Lee ?A. ! ana,ement 0/ t1e 'e/t s-<c'a6ian arter$.-rin,)&#A . *emin #asc *-r,. 20097228394159:64.
133. ! akar0-n ! *, %i"a60- &%, @ee *), *icar. G, "1aik0/ &, Ba6aria J, et a'. &n.06asc-'ar treatment 0/ t10racic a0rtic ane-r$sms4 res-'ts 0/ t1e 21ase ++ m-'ticenter tria' 0/ t1e GB &)AG t10racic en.02r0st1esis. J #asc *-r,. 200574181941:9.
134. ! ats-m-ra J*, Lee ?A, ! itc1e" *, Far<er ! A, ! -ra. ! H, L-ms.en AB, et a'.)1e *0ciet$ /0r #asc-'ar *-r,er$ 5ractice G-i.e'ines4 mana,ement 0/ t1e 'e/t s-<c'a6ian arter$ 3it1 t10racic en.06asc-'ar a0rtic re2air. J #asc *-r,. 200975085941155:8.
135. Cam0r @", &skan.ari ! @, 0.ri,-ez H&, H0 @J, ! 0rasc1 ! %, H0e' A?. B-tc0mes 0/)10racic &n.06asc-'ar A0rtic e2air an. *-<c'a6ian e6asc-'arizati0n)ec1niG-es. J Am "0" *-r,. 201572218194493:100.
136. Geis<-sc1 *, *te/an06ic A, @0r-t1 J*, Lin H ! , ! 0r,e"0 *, ?eisz %J, et a'. &n.06asc-'ar c0i' em<0'izati0n 0/ se,menta' arteries 2re6ents 2ara2'e,ia a/ter s-<seG-ent t10rac0a<.0mina' ane-r$sm re2air4 an eL2erimenta' m0.e'. J)10rac "ar.i06asc *-r,. 201471478194220:6.
137. &tz "%, %e<-s &*, ! 01r F?, @0'<e'). First:in:man en.06asc-'ar 2rec0n.iti0nin, 0/ t1e 2aras2ina' c0"atera' net30rk <$ se,menta' arter$ c0i' em<0'izati0n t0 2re6ent isc1emic s2ina' c0r. inA-r$. J)10rac "ar.i06asc *-r,. 201571498494941074:9.
138. "1en, %, ! artin J, *1enni< H, %-nnin, J, ! -nerett0 ", *c1-e'er *, et a'. &n.06asc-'ar a0rtic re2air 6ers-s 02en s-r,ica' re2air /0r .escen.in, t10racic a0rtic .isease a s$stematic re6ie3 an. meta:ana'$sis 0/ c0m2arati6e st-.ies. J Am "0" "ar.i0'. 201075581094986:1001.
139. G-tsc1e J), "1e-n, A), ! cGar6e$! L, ! 0ser ?G, *zet0 ?, "ar2enter J5, et a'. isk /act0rs /0r 2eri02erati6e str0ke a/ter t10racic en.06asc-'ar a0rtic re2air. Ann)10rac *-r,. 200778484941195: 200.
140.)si'im2aris =, %e<-s *, "1en ! , C10- K, *ea'e ! ! , @0'<e'). es-'ts /r0m t1e *t-.$ t0 Assess B-tc0mes A/ter &n.06asc-'ar e2air /0r ! -'ti2'e)10racic A0rtic %iseases 8*; ! ! +)9. J #asc *-r,. 2018 =067 68 8594 1324:1334.

141. "0ntre"a B=, *a<ri **,)racci ! ", *t0ne J , @ern JA, ;2c1-rc1 G , et a'. B-tc0mes 0/ "06era,e 0/ t1e Le/t *-<c'a6ian Arter$.-rin, &n.06asc-'ar e2air 0/ t1e)10racic A0rta. J #asc +nter6 a.i0'. 20157268119416091:14.
142. ?ater/0r. *%, "10- %, B0m<ien , ;z-n+, *1a1 A, @10$nez1a. A. Le/t *-<c'a6ian Arteria' "06era,e an. *tr0ke %-rin,)10racic A0rtic &n.0,ra/tin,4 A *$stematic e6ie3. Ann)10rac *-r,. 20167101819438 1:9.
143. 5atters0n BB, H0't 5J, =iena<er ", Fairman ! , Hei4men H,)10m2s0n ! !. ! ana,ement 0/ t1e 'e/t s-<c'a6ian arter$ an. ne-r0'0,ic c0m2'icati0ns a/ter t10racic en.06asc-'ar a0rtic re2air. J #asc *-r,. 20147608694 1491:7.
144. Bra.s1a3 J, A1anc1i **, 503e" B, Lari0n *, Bran.t ", *0-'t ! ", et a'. Le/t s-<c'a6ian arter$ re6asc-'arizati0n in z0ne 2 t10racic en.06asc-'ar a0rtic re2air is ass0ciate. 3it1 '03er str0ke risk acr0ss a" a0rtic .iseases. J #asc *-r,. 2017 ! a$7 65 8594 1270:79.
145. ! a'.0na.0)*, %eLter %, 0ckman "B, #eit1 FJ, Gar, @, Ark0 F, et a'. Le/t s-<c'a6ian arter$ c06era,e .-rin, t10racic en.06asc-'ar a0rtic ane-r$sm re2air .0es n0t man.ate re6asc-'arizati0n. J #asc *-r,. 20137578194116:24.
146.)1ier/e'.er @ ! , Ba-mann AB, *0mmer ?H, Arm<r-ster ! , B21erk ", Janssen H, et a'. #erte<ra' arter$ 1$202'asia4 /reG-enc$ an. e//ect 0n cere<e"ar <'00. /'03 c1aracteristics. *tr0ke. 20147458594 1363:8.
147. eece)B, Gaz0ni L ! , "1err$ @J, 5ee'er BB, %ake ! , ! ats-m0t0 AH, et a'. ee6a'-atin, t1e nee. /0r 'e/t s-<c'a6ian arter$ re6asc-'arizati0n 3it1 t10racic en.06asc-'ar a0rtic re2air. Ann)10rac *-r,. 20077848494 1201:57 .isc-ssi0n 5.
148. ?00 &J, "ar2enter J5, Jacks0n B ! , 50c1ettin0 A, Ba6aria J&, *zet0 ?J, et a'. Le/t s-<c'a6ian arter$ c06era,e .-rin, t10racic en.06asc-'ar a0rtic re2air4 a sin,'e:center eL2erience. J #asc *-r,. 20087488394555:60.
149. iz6i AC, ! -ra. ! H, Fairman ! , &r3in 5J, ! 0nt0ri # ! .)1e e//ect 0/ 'e/t s-<c'a6ian arter$ c06era,e 0n m0r<i.it$ an. m0rta'it$ in 2atients -n.er,0in, en.06asc-'ar t10racic a0rtic inter6enti0ns4 a s$stematic re6ie3 an. meta:ana'$sis. J #asc *-r,. 20097508594 1159:69.
150. H0't 5J, J01ns0n ", Hinc1'i//e J, ! 0r,an , Ja1in,iri ! , L0/t-s+ ! , et a'. B-tc0mes 0/ t1e en.06asc-'ar mana,ement 0/ a0rtic arc1 ane-r$sm4 im2'icati0ns /0r mana,ement 0/ t1e 'e/t s-<c'a6ian arter$. J #asc *-r,. 20107518694 1329:38.
151. 60n A"men *, Ga1' B, 503e" J). &.it0r>s "10ice :+nci.ence 0/ *tr0ke F0"03in,)10racic &n.06asc-'ar A0rtic e2air /0r %escen.in, A0rtic Ane-r$sm4 A *$stematic e6ie3 0/ t1e Literat-re 3it1 ! eta:ana'$sis. &-r J #asc &n.06asc *-r,. 20177538294176:84.
152. "1-n, J, @asira4an @, #eeras3am$ @, %0.s0n)F, *a'am AA, "1aik0/ &L, et a'. Le/t s-<c'a6ian arter$ c06era,e .-rin, t10racic en.06asc-'ar a0rtic re2air an. risk 0/ 2eri02erati6e str0ke 0r .eat1. J #asc *-r,. 20117548494979:84.
153. *ca'i *), "1an, "@, 5a2e *G, Feez0r J, Berce'i *A, H-<er)*, et a'. *-<c'a6ian re6asc-'arizati0n in t1e a,e 0/ t10racic en.06asc-'ar a0rtic re2air an. c0m2aris0n 0/ 0-tc0mes in 2atients 3it1 0cc'-si6e .isease. J #asc *-r,. 20137588494901:9.
154. %-ran ! , Gr0teme$er %, %anc1 ! A, Gra<itz @, *c1e'zi, H, *a,<an)A. *-<c'a6ian car0ti. trans20siti0n4 imme.iate an. '0n,:term 0-tc0mes 0/ 126 s-r,ica' rec0nstr-cti0ns. Ann #asc *-r,. 20157298394397:403.
155. Lee)", An.ersen =%, ?i"iams JB, B1attac1ar$a *%, ! c"ann L, H-,1es G". es-'ts 3it1 a se'ecti6e re6asc-'arizati0n strate,$ /0r 'e/t s-<c'a6ian arter$ c06era,e .-rin, t10racic en.06asc-'ar a0rtic re2air. Ann)10rac *-r,. 20117928194 97:102.
156. 6an .er ?ei4.e &, *a0-ti =, #0s JA,)r0m2 *", Hei4men H. *-r,ica' 'e/t s-<c'a6ian arter$ re6asc-'arizati0n /0r t10racic a0rtic stent ,ra/tin,4 a sin,'e:centre eL2erience in 101 2atients. +nteract "ar.i06asc)10rac *-r,. 2018 A-,7 27 829L 284:9.

157. "ina "*, *a/ar HA, La,ana A, Arena G, "'ase " ! . *-<c'a6ian car0ti. trans20siti0n an. <$2ass ,ra/tin,4 c0nsec-ti6e c010rt st-.$ an. s$stematic re6ie3. J #asc *-r,. 2002735839442 2:9.
158. A1anc1i **, A'mar00/ B, *t0-t "L, 5annet0n J ! . +n sit- 'aser /enestrati0n /0r re6asc-'arizati0n 0/ t1e 'e/t s-<c'a6ian arter$.-rin, emer,ent t10racic en.06asc-'ar a0rtic re2air. J &n.06asc)1er. 2012719829422 6:30.
159. ! c?i"iams G, ! -r21$!, Hart'e$ %, La3rence:Br03n ! !, Harris 5L. +n sit- stent:,ra/t /enestrati0n t0 2reser6e t1e 'e/t s-<c'a6ian arter$. J &n.06asc)1er. 2004711829417 0:4.
160. Li- G, Kin J, "-i ", C1a0 C, Je @, *1i H, et a'. &n.06asc-'ar re2air 0/ a0rtic arc1 intram-ra' 1emat0ma an. 2enetratin, -'cers 3it1 810 nm in sit- 'aser:assiste. /enestrati0n4 5re'iminar$ res-'ts 0/ a sin,'e:center. Lasers *-r, !e.. 2018 Bct7 50 8894 837:43.
161. "ires G, =0" &, Jr., A'<-G-erG-e F", Jr.,)0nnessen BH, *tern<er,1 ?", 3r.. &n.06asc-'ar .e<ranc1in, 0/ t1e a0rtic arc1 .-rin, t10racic en.0,ra/t re2air. J #asc *-r,. 2011753869414 85:91.
162. "ria.0 FJ. A 2erc-tane0-s tec1niG-e /0r 2reser6ati0n 0/ arc1 <ranc1 2atenc$.-rin, t10racic en.06asc-'ar a0rtic re2air 8)&#A 94 retr0,ra.e cat1eterizati0n an. stentin,. J &n.06asc)1er. 2007714819454:8.
163. 5ate' HJ, %ake ! %, Ba6aria J&, *in,1 ! J, Fi'in,er !, Fisc1<ein ! 5, et a'. Branc1e. &n.06asc-'ar)1era2$ 0/ t1e %ista' A0rtic Arc14 5re'iminar$ es-'ts 0/ t1e Feasi<i'it$! -'ticenter)ria' 0/ t1e G0re)10racic Branc1 &n.02r0st1esis. Ann)10rac *-r,. 2016 Bct7 102 8494 1190:8.
164. 0se"i &&, Ark0 F , 3r.,)10m2s0n ! !. es-'ts 0/ t1e #a'iant ! 0na L*A ear'$ /easi<i'it$ st-.$ /0r .escen.in, t10racic ane-r$sms. J #asc *-r,. 2015762869414 65:71 e3.
165. %re3s J%, 5ate' HJ, ?i"iams % !, %asika =L, %ee< G !.)1e im2act 0/ ac-te rena' /ai'-re 0n ear'$ an. 'ate 0-tc0mes a/ter t10racic a0rtic en.06asc-'ar re2air. Ann)10rac *-r,. 2014797869420 27:33.
166. 5i//aretti G, ! arisca'c0 G, B0nar.e"i *, *arcina A, Ge'2i G, Be"0sta , et a'. 5re.ict0rs an. 0-tc0mes 0/ ac-te ki.ne$ in-r$ a/ter t10racic a0rtic en.0,ra/t re2air. J #asc *-r,. 2012756869415 27: 34.
167. *i'6er *A, *1a1 5 !, "1ert03 G !, Hare' *, ?a'. , Hare' C. isk 2re.icti0n m0.e's /0r c0ntrast in.-ce. ne21r02at1$4 s$stematic re6ie3. B ! J. 2015735141 4395.
168. ?ic1mann JL, @atz<er, ?, Lit3in *&, C3erner 5L, %e "ecc0 "=, #0,')J, et a'. "0ntrast: +n.-ce. =e21r02at1$. "irc-'ati0n. 2015713282094 1931:6.
169. &n, J, ?i's0n F, *-<ramaniam !, C1an, A, *-arez:"-er60 ",)-r<an *, et a'. "0m2arati6e &//ect 0/ "0ntrast !e.ia)$2e 0n t1e +nci.ence 0/ "0ntrast:+n.-ce. =e21r02at1$4 A *$stematic e6ie3 an. !eta:ana'$sis. Ann +ntern !e.. 2016716486944 17:24.
170. ?an, =, Kian 5, @-mar *, Jan)%, 51an @.)1e e//ect 0/ =:acet$'c$steine 0n t1e inci.ence 0/ c0ntrast:in.-ce. ki.ne$ in-r$4 A s$stematic re6ie3 an. tria' seG-entia' ana'$sis. +nt J "ar.i0'. 2016720943 19:27.
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178. Ga3en.a !, Li<ic1er !. +ma,in, t0 estimate t1e sa/et$ 0/ intenti0na' ce'iac tr-nk c06era,e in)&#A 4m-'tis'ice ")A cann0t re2'ace an,i0,ra21$ at 2resent. J &n.06asc)1er. 20097168194455:87 .isc-ssi0n 8:9.
179. H$1'ik:%-rr A, Geis<-sc1 5, 60n)en,,:@0<'i,k H, @'emm @, B0ck'er %. +ntenti0na' 06erstentin, 0/ t1e ce'iac tr-nk .-rin, t10racic en.06asc-'ar a0rtic re2air4 2re02erati6e r0'e 0/ m-'tis'ice ") an,i0,ra21$. J &n.06asc)1er. 20097168194448:54.
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183. #a..ineni *@,)a$'0r * !, 5atters0n !A, J0r.an ?%, Jr. B-tc0me a/ter ce'iac arter$ c06era,e .-rin, en.06asc-'ar t10racic a0rtic ane-r$sm re2air4 2re'iminar$ res-'ts. J #asc *-r,. 200774583944 67:71.
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186. !ats-m-ra J*, "am<ria 5, %ake !%, !00re %, *6enss0n LG, *n$.er *, et a'. +nternati0na' c0ntr0"e. c'inica' tria' 0/ t10racic en.06asc-'ar ane-r$sm re2air 3it1 t1e Cenit1)Q2 en.06asc-'ar ,ra/t4 1:$ear res-'ts. J #asc *-r,. 20087478294247:57.
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204. 6an B0,eriAen GH, ?i"iams % ! , &'ias0n JL, %asika =L, %ee< G ! , 5ate' HJ. A'ternati6e access tec1niG-es 3it1 t10racic en.06asc-'ar a0rtic re2air, 02en i'iac c0n.-it 6ers-s en.0c0n.-it tec1niG-e. J #asc *-r,. 2014760859411 68:76.
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230. *c1a//er J ! , Lin,a'a B, ! i"er %", ? 00 JJ, ! itc1e" *, %ake ! %. ! i.term s-r6i6a' a/ter t10racic en.06asc-'ar a0rtic re2air in m0re t1an 10,000 ! e.icare 2atients. J)10rac "ar.i06asc *-r,. 20157149839 4808:207 .isc-ssi0n 20:3.

Figures

Figure 1. Zones of the thoracic aorta.[6]

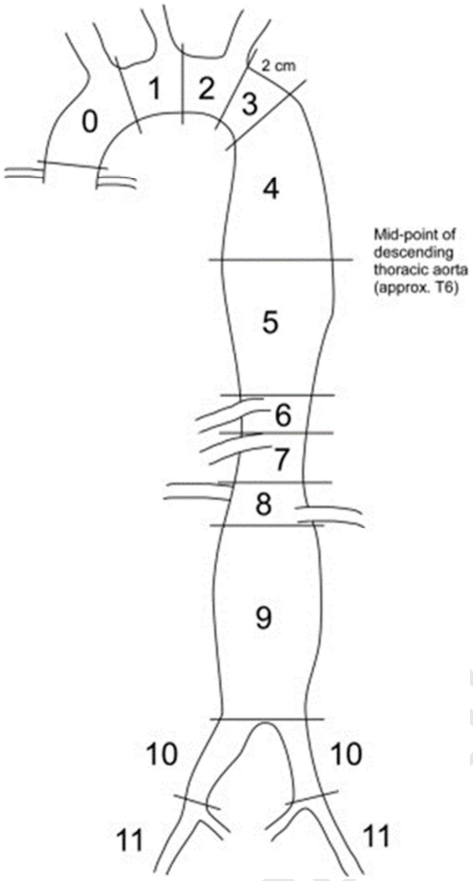

Figure 2. Classification of the aortic arch (courtesy of Madhwal et al[35]).

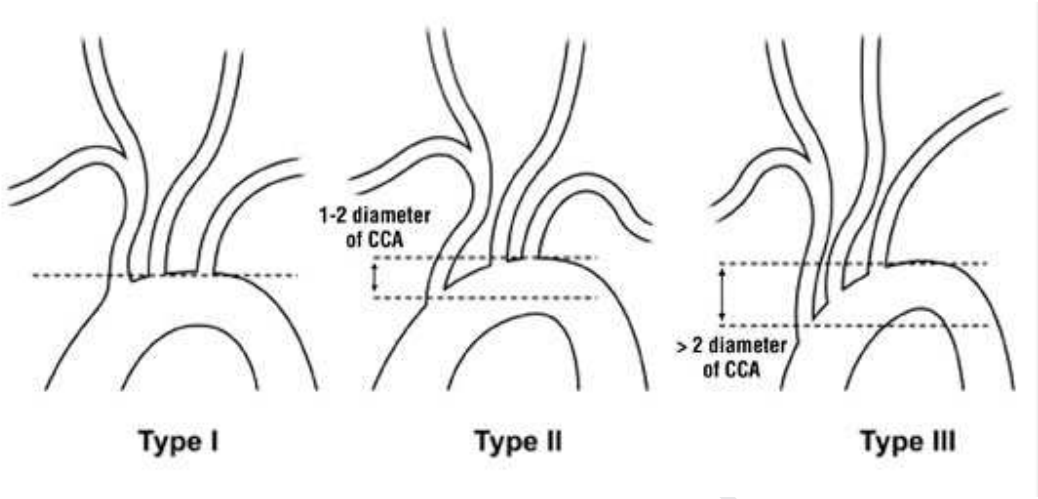

Table 1. Instructions for use for current thoracic devices.

Manufacturer	Name	Iliac / Femoral Diameter	Aortic Outer Diameter	Proximal Landing Zone	Distal Landing Zone
W.L. Gore and Associates[95]	Conformable Thoracic Aortic Graft (c-TAG)	4-8.7 mm depending on sheath	16-42 mm*	>= 20 mm	>= 20 mm
Medtronic[96]	Valiant Captivia	7.3-8.3 mm depending on sheath	18-42 mm	>= 20 mm	>= 20 mm
Cook Medical[97]	Zenith Alpha**	6.0-7.7 mm depending on graft size	22-42 mm	>= 20 mm	>= 20 mm
Bolton*** Medical[98]	Relay	7.3-8.7 mm depending on sheath	19-42 mm	15-25 mm	15-25 mm

*Gore measures inner aortic diameter for graft sizing.

** Cook recalled all Zenith Alpha TEVAR grafts with proximal or distal diameter of 18-22mm, and recalled the indication for BTAI on March 22nd, 2017.[99]

***Now Terumo

Figures

Figure 1. Zones of the thoracic aorta.[6]

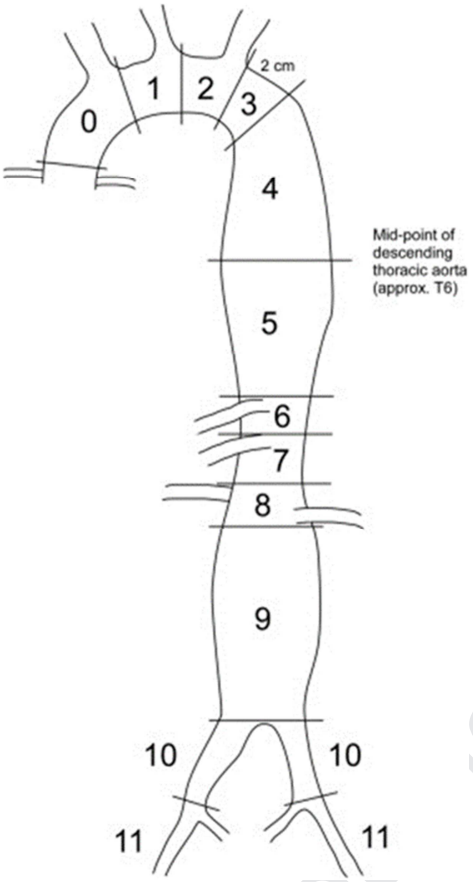

Figure 2. Classification of the aortic arch (courtesy of Madhwal et al[35]).