Review Article

Open and endovascular repair of thoracoabdominal aortic aneurysm—a narrative review

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Background and Objective: Thoracoabdominal aortic aneurysms (TAAAs) are associated with a high rate of morbidity and mortality when left untreated, yet their repair has been associated considerable risk as well. We aim to present a review of the epidemiology, risk factors, pathophysiology, natural history, management strategies, and current literature on open and endovascular repair of thoracoabdominal aortic aneurysms.

Methods: A narrative review using current search of the most recent literature on this topic.

Key Content and Findings: Indication for surgical repair includes symptomatic aneurysms and those at a size or growth threshold. Open repair is the gold standard for surgical management of TAAA and remains so, however, endovascular repair has an increasingly broad applicability, and technology has correspondingly improved in the two decades since its introduction to clinical practice. Reported contemporary operative mortality has ranged from less than 5% to over 20%. The primary adverse complication of open and endovascular repair is ischemic injury, including renal injury, with reported incidence ranging from 2.8–12.5% in contemporary series, and spinal cord injury, with reported incidence ranging from 2.5% to 7.3% in contemporary series. Cerebrospinal fluid drainage has been shown to be the best-proven mechanism to date to prevent spinal cord injury in both open and endovascular repair.

Conclusions: Endovascular repair is associated with a higher reintervention rate than open repair. Surgeon and center volume and expertise are related to clinical outcomes and should be taken into consideration when deciding on surgical repair.

Keywords: Thoracoabdominal aortic aneurysm (TAAA); endovascular thoracoabdominal aortic aneurysm repair; open thoracoabdominal aortic aneurysm repair

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Introduction

Thoracoabdominal aortic aneurysms (TAAAs) account for approximately 10% of all aortic aneurysms (1). Successful repair of TAAAs requires careful consideration of the individual patient's clinical and aortic anatomical characteristics. While open repair has traditionally been the standard approach, endovascular repair has evolved over the last two decades to handle complex patient anatomy. Ischemic injury is a known and feared complication of both open and endovascular TAAA repair, and numerous strategies for prevention of renal, visceral, and spinal cord ischemia have been developed and tested. Herein we present a comprehensive narrative review of the epidemiology, classification, and pathophysiology of TAAAs, approaches to repair, outcomes after surgical intervention, and common complications with their strategies for their prevention. We further discuss the most recent American Heart Association (AHA)/American College of Cardiology

Table 1 The search strategy summary

Items	Specification
Date of search	November 4th, 2022
Databases and other sources searched	PubMed
Search terms used	"thoracoabdominal aortic aneurysm", "endovascular thoracoabdominal aortic aneurysm repair", "open thoracoabdominal aortic aneurysm repair"
Timeframe	2007–2022
Inclusion criteria	Prioritized English-language and randomized trials, included observational studies
Selection process	Junior and senior author agreement
Additional considerations	Identification of historically relevant or related articles by agreement between junior and senior author

(ACC) Clinical Practice Guidelines for management of TAAA, changes in the current recommendations compared with prior iterations, and the evidence that forms the basis of these updated recommendations. We present this article in accordance with the Narrative Review reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-22-1880/rc).

Methods

We searched PubMed for references with the terms "thoracoabdominal aortic aneurysm", "endovascular thoracoabdominal aortic aneurysm repair", "open thoracoabdominal aortic aneurysm repair" or their combination in the title or abstract. We also identified relevant articles from the references lists of selected articles. We prioritized randomized trials and publications from the last 15 years but cited other references where historically relevant and necessary. The search strategy is summarized in *Table 1*.

Epidemiology and risk factors

TAAAs have an incidence of ten per 100,000 person-years (1) and account for approximately 10% of all aortic aneurysms, with an increasing incidence in the last twenty years attributable to incidental diagnoses secondary to the increased use of cross-sectional imaging (2). The preponderance (60–70%) of TAAAs occur in men (3,4). However, men with TAAAs are less likely than women to suffer a dissection or rupture, as demonstrated in a 2002 retrospective study of 721 TAAA patients (446 male, 275 female) by Davies *et al.* that found that male sex was associated with a lower risk of dissection or rupture [odds ratio (OR) 0.340, 95% CI: 1.14–0.819; P=0.0162] (3). The annual growth rate is also higher in women than in men (4). A 2017 retrospective study by Cheung *et al.* of 82 TAAA patients found that sex was significantly associated with greater aneurysm growth on multivariable analysis (4); a 2021 retrospective study of 907 TAAA patients (292 women) found a growth rate of 0.17 cm per year in men compared to 0.25 cm per year in women (P<0.001) (5).

Risk factors for TAAA include hypertension (present in the vast preponderance of patients with TAAAs), atherosclerosis, smoking, and chronic obstructive pulmonary disease (COPD) (6). Hereditary disorders, including syndromes such as Marfan, Loeys-Dietz, and Ehlers-Danlos, account for 15–20% of all TAAAs and put patients at higher risk of aortic dissection (7). Chronic aortic dissection is related either to hypertension or the aforementioned hereditary disorders, and 30–40% of patients with chronic dissection will require TAAA repair (6-8). Other disorders carrying an increased risk of TAAA include familial thoracic aortic disease (7), inflammatory aortitis, autoimmune disorders, traumatic injury, and congenital conditions (coarctation of the aorta, Turner syndrome) (9).

Pathophysiology, natural history, and classification

The normal diameter of the descending thoracic aorta ranges from 2.42–2.98 cm in men and 2.2–2.68 cm in women, with a decrease in size ranging from 0.2–0.5 cm as the aorta traverses the diaphragm and abdomen (10,11). The normal diameter varies by the segment of the aorta in

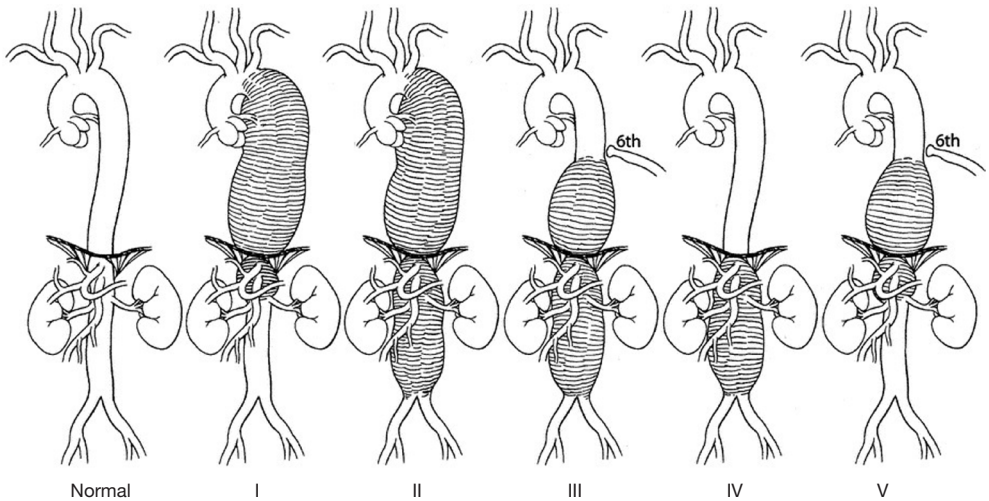

Figure 1 Classification of thoracoabdominal aortic aneurysms. Reproduced with permission from Safi *et al*.

question and by the anatomical characteristics (sex, height, body size) of the individual patient.

The aortic wall is comprised of three layers: intima, media, and adventitia. Degradation of structural proteins in the media leads to aneurysmal degeneration. In particular, loss of collagen and elastin in the medial layer and deposition of proteoglycans contributes to the loss of structural integrity. The vast majority, approximately 70%, of TAAAs are termed degenerative aneurysms, in which atherosclerotic disease is superimposed on, and contributes to, medial degeneration (12-14). A detailed description of the biochemical pathways and proteins contributing to degeneration of the aortic wall is not yet clearly delineated and is beyond the scope of this review.

Hemodynamic stress against the weakened wall of the aorta leads to dilatation of those areas and formation of the aneurysm. Hypertension increases the pressure against the aortic wall and accelerates the process of dilatation. A tear or disruption of the intima, often secondary to excessive hemodynamic force associated with hypertension, leads to the creation and eventual propagation of a dissection. The average growth rate of TAAAs has been reported between 0.10 cm per year to 0.42 cm per year (3,15,16). Extent of dilatation, or aortic diameter, has historically been the best predictor of rupture, dissection, and mortality (3,6,16,17), and forms the basis for current guideline recommendations for surgical intervention. Without surgical intervention, the 5-year survival of TAAA ranges from 10–20% (1,15).

TAAAs may present with compressive symptoms, such as intrascapular pain, chest pain radiating to the back, or abdominal pain. Depending upon the size and location of the aneurysm, other symptoms may include dysphagia secondary to compression of the esophagus, wheezing or coughing secondary to airway compression, or hoarseness secondary to stretching of the recurrent laryngeal nerve (18). In addition, embolization of thrombus associated with the aneurysm wall, or of atheromatous debris, may cause malperfusion of any of a number of end organ targets. Ischemic symptoms with end organ dysfunction, including mesenteric ischemia, renal ischemia, or peripheral limb ischemia, and symptoms of erosion of the aneurysm into abutting structures (i.e., aorto-enteric fistula presenting with gastrointestinal bleeding) should be urgently or emergently addressed (9). However, most TAAAs are asymptomatic until dissection or rupture (9).

TAAAs involve the descending thoracic aorta and the abdominal aorta, ranging from below the origin of the left subclavian artery and to the bifurcation of the abdominal aorta, and are classified based upon the predicted extent of an open operative repair using the Crawford classification system (13), depicted in *Figure 1*. Extent I repairs start distal to the origin of the left subclavian artery (proximal to the sixth rib) and extend to the renal arteries. Extent II repairs reach from the left subclavian (proximal to the sixth rib) and extend below the renal arteries, to the aortoiliac bifurcation. Extent III repairs cover from distal to the sixth rib, above the level of the diaphragm and inclusive of the distal thoracic aorta, to the abdominal aorta. Extent IV repairs begin below the level of the diaphragm and extend to the aortoiliac bifurcation, including the visceral aortic segment. A more

recent addition to the traditional Crawford classification, Extent V aneurysms arise in the distal descending thoracic aorta, distal to the sixth rib, but extends to include only the visceral segment of the abdominal aorta (19).

Indications for repair

Elective repair of asymptomatic aneurysms is based on the principle that repair should be undertaken when the annual risk of rupture exceeds the surgical mortality and morbidity. Therefore, patients with an unacceptably high surgical risk (i.e., cardiopulmonary or other end organ failure) or patients with shorter life expectancy for other reasons may not be candidates for repair. According to the 2022 AHA/ACC guidelines (9), asymptomatic TAAAs should be recommended for open repair, which is still the standard of care, at a threshold aortic diameter of 6.0 cm [Class of Recommendation (COR) 1, Level of Evidence (LOE) B-NR] or at a threshold of 5.5 cm if the repair is performed by an experienced surgeon working as part of a multidisciplinary aortic team (COR 2A, LOE B-NR); consideration should be given to endovascular repair for patients who are not able to tolerate open surgery (9). The 2022 guidelines also recommend consideration of repair at 5.5 cm or less in patients with high-risk features for rupture, including annual growth of the aneurysm by 0.5 cm or more, symptomatic aneurysms, significant change in the appearance of the aneurysm, saccular aneurysms, or aneurysms with penetrating atherosclerotic ulcers (COR 2A, LOE B-NR). The Society for Vascular Surgery guidelines also recommend repair at lower thresholds in populations with hereditary thoracic aortic disorders (20-22). Repair is recommended at a threshold of 5.5 cm for isolated descending thoracic aneurysms (DTAs) (COR 1, LOE B-NR) (9).

These recommendations are intended to prevent aortic rupture. A retrospective study by Zafar *et al.* of 907 patients with DTAs and TAAAs (8) found that at an aortic diameter of 6.0 cm, the annual rate of rupture, dissection, and death was 19%. 93% of ruptures occurred at a diameter greater than 5.0 cm, yet 80% of dissections occurred at a diameter less than 5.0 cm. Acute type B dissections occurred at a median aortic diameter of 4.1 cm, suggesting that diameter is a better predictor of rupture than of dissection and that current guidelines are not directed toward prevention of dissection.

The association of both institutional and individual surgeon volume with improved mortality and morbidity at high-volume centers and with high volume surgeons when compared with low-volume centers and surgeons has been reported (23-26). In a 2018 meta-analysis of 30 studies and 9,923 patients undergoing open TAAA repair by Moulakakis *et al.* (26), they found a statistically significant inverse association between mortality and the volume of cases at each vascular center (t=–2.00; P=0.005) on meta-regression analysis. Rocha *et al.* (25). found that institutional volume of over 60 cases in a ten-year study period (approximately six cases per year) was associated with a significant lower operative mortality after open TAAA repair compared with all other centers (13.8% versus 36.0%; P<0.01). In a 2003 retrospective study of 1,542 patients undergoing TAAA repair by Cowan *et al.* (23), high-volume surgeons (performing three to eighteen cases per year, median seven cases per year) had significantly lower operative mortality than their low-volume counterparts (11.0% versus 25.6%; P<0.001); the same was true for high-volume centers (five to 31 cases per year, median twelve cases per year) when compared with low-volume institutions (15.0% versus 27.4%; P<0.001) low surgeon volume (defined as one to two cases per year) was a significant predictor of mortality: OR 2.6; P<0.001), as was low institution volume (one to three cases per year) (OR 2.2; P<0.001). Therefore, surgeon and institutional volume have an important relationship to clinical outcomes and should be part of the decision-making process regarding whether, where, and how to undergo repair.

Outcomes of open repair

Single-lung ventilation with a left thoracoabdominal approach is the traditional approach for open TAAA repair; reconstruction of both the aorta and the vasculature to the viscera and kidneys is performed (27). To decrease the risk of recurrent visceral patch aneurysms, a multibranched graft may be preferred in patients with hereditary thoracic aortic disease or in young patients (18). Reimplantation of the intercostal arteries at the T10–T12 level is performed when anatomically indicated in order to limit the risk of spinal ischemic complications.

Early series of open TAAA repair reported high rates of ischemic complications. A 1993 retrospective cohort study by Svensson *et al.* of 1,509 TAAA patients undergoing 1,679 open TAAA repairs between 1960 and 1991 found a 30-day mortality of 8%, a spinal cord injury (SCI) incidence of 16%, and renal failure incidence of 18% with an incidence of dialysis requirement of 9% (28). On logistic

regression, significant predictors of paraplegia or paraparesis were total aortic clamp time, extent of the repair, aortic rupture, patient age, proximal aortic aneurysm, and history of renal dysfunction (P<0.05).

Results have been mixed in large-scale contemporary series, although overall the reported incidence of both SCI and dialysis requirement have decreased. In 2016, Coselli *et al.* (12). published the largest series to date of open TAAA repair in 3,309 patients and found an in-hospital mortality of 7.5%, permanent paraplegia incidence of 2.9%, paraparesis incidence of 2.4%, and an incidence of renal failure requiring dialysis of 7.6%. In further studies on this same patient cohort, the most important risk factors for mortality or ischemic injury were age, chronic pre-existing pulmonary or kidney disease, and need for urgent or emergent repair (29-32).

In the patients described by Coselli *et al.* (12), patients over 80 years old had almost quadruple the in-hospital mortality (26%) than the rest of the cohort (6.9%), and age was significantly associated with mortality and ischemic injury. However, a 2018 single-center study by Girardi *et al.* (33) of 783 patients (96 octogenarians) undergoing open repair of DTA or TAAA found no difference in operative mortality (octogenarians: 5.6% versus remaining cohort: 5.7%; P=0.852), SCI (2.0% versus 2.0%; P=0.715), renal failure requiring dialysis (5.3% versus 5.2%; P=1.00), or respiratory complications (26.3% versus 24.9%; P=0.651). The authors did note that a greater proportion of octogenarians' repairs (85.4%) were performed using the clamp-and-sew technique when compared with the remaining patients' repairs (61.6%; P<0.001), leading to a significantly shorter duration of aortic cross-clamp time (30.7 versus 26.6 minutes; P=0.04), which may have contributed to the better postoperative outcomes in this study.

Preoperative renal and pulmonary failure have been identified as risk factors for mortality and morbidity after open TAAA repair. A 2017 single-center retrospective study of 711 open DTA and TAAA patients included 202 with preoperative renal failure and found that these patients also presented with other significantly worse comorbidities, including smoking, chronic pulmonary disease, peripheral vascular disease, and diabetes, than patients without renal failure (34). The incidence of operative mortality was 14.2% in patients with renal failure versus 2.2% in those without, and five-year survival was significantly lower in patients with renal failure as well (45.0% versus 79.8%; P<0.001). Consistent with findings from prior studies (12,28), preoperative renal failure was found to be a significant predictor of operative mortality (OR: 4.91, 95% CI: 2.01–11.97; P<0.001). Another study by the same group included 149 propensity-matched pairs of patients undergoing TAAA and DTA repair with forced expiratory volume in 1 second (FEV1) either above or below 50%, and found an incidence of major adverse events of 33.1% in patients with FEV1 <50% compared with 19.5% in those with FEV1 >50% (P=0.08). In propensity-matched patients undergoing TAAA, there was a significant difference in operative mortality (FEV1 <50%: 12.2% versus FEV1 >50%: 3.5%; P<0.001). This difference was driven by operative mortality in patients undergoing Extent II repair (29.6% versus 7.0%; P=0.013) (35). A summary of the large TAAA repair studies from the last decade is presented in *Table 2*.

While open repair has well-described perioperative risk, as noted above, its long-term success is also well-described, with low reported rates of visceral patch aneurysms, branch-vessel occlusion, pseudoaneurysm, and, importantly, re-intervention, the last of which distinguishes it from endovascular repair. Coselli *et al.* reported a freedom from repair failure of 95.3%±0.6% at ten years and 94.1%±0.8% at 15 years in the aforementioned cohort (12). Graft infection represents a serious but rare late complication of TAAA repair, with Coselli *et al.* reporting just eighteen instances at fifteen-year follow up (12); other series have reported a similarly low incidence of graft infection at long-term follow-up, ranging from 0.42% to 2.32% (42,43).

Outcomes of endovascular repair

Endovascular repair of TAAA was described in 2001 (44), and was initially reserved for elderly or high-risk patients for whom open surgery was medically contraindicated. The approach and devices used have improved over time. Endovascular devices have evolved to include branched, fenestrated, and parallel grafts, leading to wider adoption of this technique (45-47). Parallel grafting uses bridging stents placed in parallel to the aortic graft and into the visceral and renal vessels. Small series on parallel grafting for TAAA have shown relatively good rates of technical success and an acceptably low incidence of adverse outcomes (48,49). However, this technique is limited by both aortic calcification and narrow aortic diameter, as well as flow between stent components that may cause endoleak (18). Fenestrated and branched grafts have replaced parallel grafts given the reduction of these limitations.

Table 2 Large studies in the last decade of open and endovascular repair of thoracoabdominal aortic aneurysms

Approach	Year	Study	Study type	Cohort (n)	Operative mortality, %	Permanent SCI, %	Renal failure, % (HD)	Reintervention, %
Open	2012	Lima *et al.* (36)	P, single-center	361	7.6%	7.3%	9.1%	NR
Open	2005	Estrera *et al.* (37)	R, single-center	1,896	15.9%	7.1%	16.6%	4.9%†
Open	2016	Coselli *et al.* (12)	R, single-center	3,309	7.5%	5.4%	7.6%	Freedom from reintervention: 94.1%
Open	2018	Girardi *et al.* (33)	R, single-center	783	5.6%	2.5%	5.2%	2.4%†
Open	2019	Geisbüsch *et al.* (38)	R, population	1,422	23.9%	NR	NR	NR
Open	2021	Rocha *et al.* (25)	R, population	361	17.4%	3.6%	12.5%	13.5%
Endo	2012	Guillou *et al.* (39)	R, single-center	89	10%	7.8%	6.7%	4.2%
Endo	2016	Eagleton *et al.* (40)	P, single-center	354	4.8%	4.0%	2.8%	Freedom from reintervention: 54%
Endo	2019	Geisbüsch *et al.* (38)	R, population	856	10.6%	NR	NR	NR
Endo	2019	Oderich *et al.* (41)	R, single-center	316	2.5%	2%	1%	30.3%
Endo	2021	Rocha *et al.* (25)	R, population	303	10.8%	4.3%	6.9%	23.9%

†, reintervention for bleeding only, not all-cause. SCI, spinal cord injury; HD, hemodialysis; P, prospective study design; R, retrospective study design; NR, not reported.

In a large single-center retrospective study of 354 patients treated by fenestrated and branched endografts for Extent II or III TAAAs, technical success was 94%, 30-day mortality was 4.8%, with an SCI incidence of 8.8%, a permanent SCI incidence of 4%, and an incidence of renal failure requiring dialysis of 2.8%. Similar to the data regarding open surgery, the factors associated with mortality included age [hazard ratio (HR) 1.031, 95% CI: 1.008–1.054; P=0.008], chronic pulmonary disease (HR 1.507, 95% CI: 1.05–2.1543; P=0.024), Extent II repair (HR 1.739, 95% CI: 1.226–2.467; P=0.002), cerebrovascular disease (HR 1.620, 95% CI: 1.096–2.394; P=0.016), and higher American Society of Anesthesiologists Physical Status Classification score (P=0.001) (40). Primary patency of the renal and visceral vasculature ranged from 92% (right renal artery) to 96% (celiac axis) at 36 months. Freedom from unplanned reintervention was 54% at 36 months (95% CI: 0.47–0.61), with 27 reinterventions to maintain branch vessel patency and 67 reinterventions for endoleak, highlighting need for reintervention as a main difference between endovascular and open repair. A summary of the large TAAA repair studies from the last decade is presented in *Table 2*.

Branched or fenestrated grafts can be standardized or custom-made to fit the individual patient's anatomy and aneurysm. This requires additional manufacturing time and obviates their urgent/emergent use (i.e., rupture, rapid aneurysm growth). Standardized grafts include branches for the celiac axis, superior mesenteric arteries, and bilateral renal arteries based on standard anatomy and can be used in 50–80% of patients with TAAAs (18,50,51). A meta-analysis by Konstantinou *et al.* (52) including seven observational studies and 197 patients undergoing TAAA repair using standardized t-Branch devices found a pooled success rate of 92.75%, with early mortality of 5.8%, permanent paraplegia of 1.3%, and acute renal failure of 18.7%; pooled reintervention rate at a mean follow-up of 15 months (±7 months) was 5.7% (95% CI: 1.70–11.4%). Notably, this study included 32 cases performed for ruptured aneurysm, which may have increased the rate of adverse events but also highlights the utility of standardized devices when compared with custom-made devices. Physician-modified endografts, by their very nature, do not have standardized quality control, and have also been associated with higher rates of adverse events compared with standardized and custom-made devices; their use has declined use of standardized and custom-made devices has increased (41).

Endoleak represents a primary mid- and long-term complication of endovascular TAAA repair, with the reported incidence ranging from 15–66.7% and the reported incidence of re-intervention for endoleak ranged from 3–33% in prior series (39,53,54). Management of endoleak varies depending upon the surgeon, the type of

endoleak, and its features; for instance, aneurysm sac growth may suggest the need for intervention, while regression of sac size or endoleak volume may favor observation (39,54). In prior series (39,53,54), type I endoleak was managed with cuff extension for proximal or distal seal, type II endoleak was most common and was managed either with observation or with glue embolization, and type III endoleak was managed with repeat stenting into the visceral or renal arteries to seal the modular joints.

Choosing the best approach: open or endovascular repair?

The open approach has historically been considered the gold standard of TAAA repair. Open repair is suitable for all aortic anatomies and has well-described outcomes, including long-term durability. Endovascular repair may be the only option in patients with severe medical comorbidities (cardiac, pulmonary, and renal) that render open surgery unacceptably high-risk. Endovascular repair has the further benefit of obviating the need for thoracotomy, extracorporeal circulatory support, and aortic cross-clamping. As the endovascular approach has become more common, its indications have grown to include younger and lower-risk patients. Yet despite its increased use, the need for appropriate preoperative aortic anatomy and the higher rate of reinterventions may present barriers to wider adoption.

Some patient groups are poor candidates for endovascular repair, such as those with connective tissue diseases, and should be offered open repair due to the underlying risk of continued and progressive aortic degeneration, as well as the risk of iatrogenic injury with endovascular techniques (2022 AHA/ACC guideline recommendation COR 1, LOE C-LD) (9). Additional aortic length is required for a landing zone in endovascular repair, as is an aortic lumen narrow enough to seal the endograft, thus some aneurysms may not be anatomically suited to endovascular repair. The passing of wires, catheters, and grafts demands adequate peripheral access, therefore poor access sites or severe peripheral vascular disease may render this approach impossible (9). The most recent AHA/ACC guidelines now state that in patients with intact degenerative TAAA and suitable anatomy, endovascular repair with fenestrated or branched grafts may be considered (COR 2B, LOE B-NR) (9).

Open repair remains the recommended approach for ruptured TAAA (COR 1, LOE B-NR), however, most recent guidelines do state that if a patient presenting with rupture is hemodynamically stable, endovascular repair may be considered (9). Gaudino *et al.* (55) compared the outcomes of 61 pairs of contemporary, propensity-matched patients undergoing open repair of either ruptured or intact DTA or TAAA. After matching, there was no significant difference in operative mortality, SCI, or any other major postoperative complications.

There are no prospective randomized trials directly comparing the two approaches, which limits the quality of the data that might guide clinicians and patients alike. Results of observational studies have been mixed. A 2018 meta-analysis by Rocha *et al.* included eight observational studies directly comparing the open and endovascular approaches. Two studies used propensity-matching to account for the large baseline heterogeneity between the patient populations undergoing endovascular or open repair (56-58) and found no difference in mortality. Yet pooled analysis of all unmatched and unadjusted studies found a lower mortality [relative risk (RR) 0.63, 95% CI: 0.45–0.87; P<0.01] and lower risk of SCI (RR 0.65, 95% CI: 0.42–1.01; P=0.05) with endovascular repair. A 2020 systematic review and meta-analysis (59) of 71 studies (24 on endovascular TAAA repair and 47 on open TAAA repair) by the same group found that there was no significant difference in operative mortality (endovascular: 7.4% versus open 8.9%; P=0.21), permanent paralysis (endovascular: 5.2% versus open: 4.5%; P=0.39) or long-term dialysis requirement (endovascular: 3.7 % versus open: 3.8%; P=0.93) despite significantly different preoperative patient characteristics.

A recent retrospective database study (38) including 2,607 cases (856 endovascular, 1,422 open, and 354 hybrid) found decreased in-hospital mortality among patients who underwent elective endovascular repair compared with open and hybrid repair (OR 0.35, 95% CI: 0.24–0.51; P=0.001); notably, only 18% of these cases were performed at a low-volume center. Another database study by Rocha *et al.* (25) of 664 patients undergoing surgical repair for TAAA (303 endovascular, 361 open, 241 propensity-matched patient pairs) found that open repair was associated with a higher incidence of in-hospital mortality (17.4% versus 10.8%; P=0.04) and complications, defined as the composite of SCI, permanent dialysis, or stroke (26.1% versus 17.4%; P=0.02), than endovascular repair, however, there was no difference in mortality at long-term (8 years) follow-up (HR 1.9, 95% CI: 0.78–1.50). However, these reported outcomes (in particular the in-hospital mortality rates) are less favorable than outcomes reported from single-center, high-volume series, once again reflecting the importance

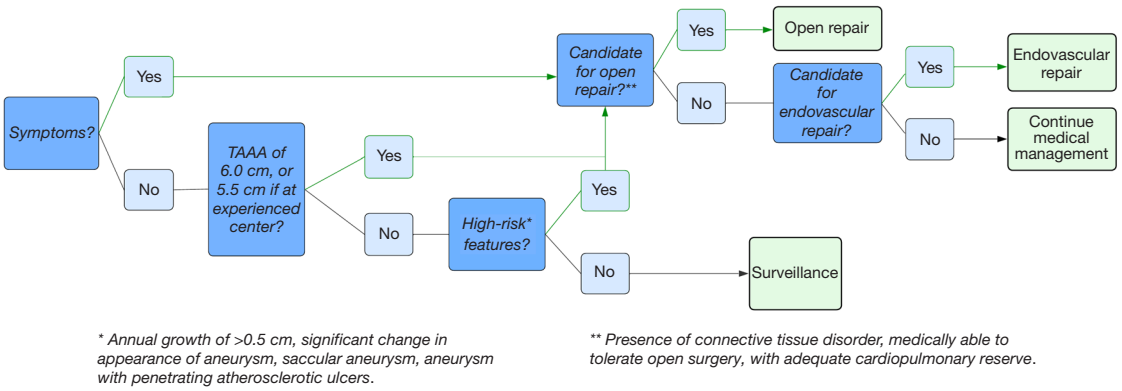

Figure 2 Algorithm for management of TAAAs. TAAA, thoracoabdominal aortic aneurysm.

of surgeon and center experience. There was no significant difference in the incidence of late adverse events between groups, with the exception of vascular reintervention, which was higher in the endovascular group (HR 2.64; P<0.01). A proposed algorithm for helping clinicians decide on the most appropriate surgical approach for TAAA repair is provided in *Figure 2*.

Ischemic injury

Strategies used to prevent ischemic injury after TAAA range from staged repair, preservation of the left subclavian and internal iliac arteries, and distal perfusion using left heart or cardiopulmonary bypass, to cold renal perfusion, hypothermia, and cerebral spinal fluid (CSF) drainage, among others (9,14,18). Improvements in the rate of ischemic complications in more recent series are a reflection of utilization of techniques that mitigate the risk of intraoperative ischemia. Current and past recommendations on strategies to prevent ischemic injury are provided in *Figure 3*.

SCI

SCI is the most dreaded complication of TAAA repair. A 2022 meta-analysis by Gaudino *et al.* (60) of 169 studies and 22,634 patients undergoing DTA and TAAA repair found a pooled SCI rate of 4.5% (95% CI: 3.8–5.4), 3.5% in DTA repair (95% CI: 1.8–6.7), and 7.6% in TAAA repair (95% CI: 6.2–9.3). Open repair had a permanent SCI rate of 5.7% (95% CI: 4.3–7.5), while endovascular repair had a permanent SCI rate of 3.9% (95% CI: 3.1–4.8; P=0.03); Extent II repair had the highest rate of permanent SCI (15%, 95% CI: 10.0–22.0; P<0.001).

Contemporary studies have reported that spinal cord drainage leads to decreased incidence of SCI, forming the basis for a COR 1, LOE A recommendation from the AHA/ACC guidelines for its use in both open and endovascular repair in patients at high risk for SCI (9). Coselli *et al.* (61) randomized 145 patients undergoing TAAA repair to CSF drainage for 48 hours postoperatively or no drainage, and found a significant reduction in paraplegia or paraparesis between the two groups (drainage: 2.6% versus no drainage: 13.0%; P=0.03); other studies have reported similar benefits with CSF drainage (62,63). Estrera *et al.* reported a decreased risk of neurologic deficit after DTA repair with the use of distal aortic perfusion and CSF drainage (OR 0.19; P=0.02) (37); Safi *et al.* reported similar benefits with the same intervention (63). CSF drainage is now the only technique for SCI prevention to receive a Class 1 AHA/ACC recommendation. In addition, guidelines provide a COR 1, LOE B-NR recommendation for management of delayed SCI by increasing arterial pressure and decreasing intrathecal pressure (with CSF drainage).

Lima *et al.* demonstrated in a retrospective study of 250 patients undergoing DTA and TAAA repairs (150 with intrathecal papaverine) that those receiving intrathecal papaverine had significantly lower rates of permanent paraplegia (3.6% versus 7.5%; 0=0.01) and paraparesis (1.6% versus 6.3%; P=0.01) than patients who did not (36). Intrathecal papaverine use was supported by a COR 2B, LOE B recommendation in the 2010 guidelines, but does not receive such a recommendation any longer (14,36). The 2010 ACC/AHA guidelines gave a COR 2A, LOE B recommendation for moderate systemic hypothermia for neuroprotection during open repair, based on a 2003

Strategy for prevention of ischemic injury	Approach	2010 AHA/ACC guideline (14)	2022 AHA/ACC guideline (9)
Distal perfusion (left heart bypass or CPB)	Open	COR 2A, LOE B	No recommendation
Reimplantation of segmental arteries	Open	No recommendation	No recommendation
Moderate systemic hypothermia	Open	COR 2A, LOE B	No recommendation
CSF drainage	Open and Endo	COR 1, LOE B	COR 1, LOE A
Preservation of left subclavian and internal iliac arteries	Open and Endo	No recommendation	No recommendation
Staged repair	Open and Endo	No recommendation	No recommendation
Preoperative segmental artery embolization	Open and Endo	No recommendation	No recommendation
Neuromonitoring	Open and Endo	COR 2B, LOE B	No recommendation
Intrathecal papaverine	Open and Endo	COR 2B, LOE B	No recommendation
Optimize spinal cord perfusion (CSF drainage, deliberate hypertension, anemia and hypoxemia)	Open and Endo	No recommendation	COR 1, LOE B-NR
Epidural irrigation with hypothermic solutions	Open	COR 2B, LOE B	No recommendation
High-dose glucocorticoids	Open and Endo	COR 2B, LOE B	No recommendation
Osmotic diuresis (mannitol)	Open and Endo	COR 2B, LOE B	No recommendation
Preoperative hydration	Open and Endo	COR 2B, LOE C	No recommendation
Cold renal perfusion	Open	COR 2B, LOE B	COR 1, LOE A
Blood perfusion	Open	COR 2B, LOE B	COR 1, LOE A
Intraoperative mannitol administration	Open and Endo	COR 2B, LOE C	No recommendation
Furosemide, mannitol, or dopamine for renal protection	Open and Endo	COR 3, LOE B	No recommendation

Figure 3 Updates to the recommendations for spinal and visceral protection against ischemic injury during thoracoabdominal aneurysm repair (9,14). AHA, American Heart Association; ACC, American College of Cardiology; CPB, cardiopulmonary bypass; COR, Class of Recommendation; LOE, level of Evidence; CSF, cerebrospinal fluid.

study of 132 patients that found that moderate systemic hypothermia had a lower risk of transient neurologic deficit (6.6%) compared with mild systemic hypothermia (32%, P=0.04); this recommendation is no longer made in the updated guidelines (64). Neuromonitoring with somatosensory and motor evoked potentials has been used by some groups to guide surgical strategy and to identify optimal perfusion pressure for patients at high risk of SCI, yet data have been mixed, and intraoperative neuromonitoring is no longer recommended (9,14,65-67).

Several strategies in spinal cord protection are based on the concept of a 'collateral network' of spinal cord perfusion, which posits that spinal cord nutrient flow is derived from an axial network of small arteries in the spinal canal and surrounding tissue that accept inputs from the hypogastric and subclavian arteries in addition to the known supply from the intercostal and lumbar (segmental) vessels (18,68,69). The natural corollary of the concept is that cord nutrient flow from one source can be increased in response to reduction from another source; conversely, nutrient 'steal' can also occur (68). This theory supports deliberate hypertension and CSF drainage in the postoperative period as the spinal cord develops its collateral nutrient supply (18). This theory also supports preoperative interventions such as staged aortic repair (70) and minimally invasive segmental artery coil embolization (18,71,72).

Renal failure

Cold renal perfusion has been found to be effective at minimizing renal injury, leading to its widespread use in open repair (73-75). A 2002 study by Köksoy *et al.* (73)

randomized 30 patients undergoing Extent II TAAA repairs to cold crystalloid perfusion or normothermic blood, and found on multivariable analysis that the use of cold crystalloid perfusion was protective against renal dysfunction (OR 0.133; P=0.02); however, a larger 2009 trial by Lemaire *et al.* randomized 172 patients to cold crystalloid perfusion or cold blood perfusion and found that there was no difference between groups in the incidence of renal failure (3% in each group) (74). Use of cold renal perfusion and blood perfusion is strongly recommended (COR 1, LOE A) (9), while the use of furosemide, mannitol, or dopamine is recommended against by the 2010 AHA/ACC guidelines when used solely for the purpose of renal protection (COR 3, LOE B) (14). In addition, patients who present with compromised visceral or renal perfusion may require further interventions to improve flow to the SMA, celiac axis, or renal arteries (COR 1, LOE B-NR) (9).

Follow-up and surveillance

Postoperative surveillance imaging should be performed annually for patients who have undergone open TAAA repair (COR 2A, LOE B-NR), with a decreasing frequency over time if imaging findings remain stable (9). Given the higher rate of needed reintervention with endovascular repair, these patients should undergo imaging at one and 12 months postoperatively (COR 1, LOE B-NR) (9), with subsequent annual surveillance provided the interval imaging is stable. Younger patients (i.e., patients with hereditary connective tissue disorders) or patients undergoing endovascular repair who may require more frequent imaging may utilize magnetic resonance imaging for surveillance given the risk of repeat exposure to radiation with computed tomography, which is the usual recommended modality (COR 2A, LOE B-NR) (9). Lifelong management of comorbidities with improved diet, smoking cessation, and medical control of hypertension and hyperlipidemia is necessary. Despite these measures, long-term survival after TAAA repair is poor (12,25,76). Coselli *et al.* (12) reported survival of 36.8% (±1) at 10 years and 18.3% (±0.9%) at fifteen years.

Conclusions

TAAA has had an increasing incidence over the last two decades, rendering a knowledge of its pathophysiology, surveillance, and management increasingly important. Open repair remains the standard of care, and is the preferred method of repair in appropriate risk patients given the comparable perioperative outcomes and reduced risk of reintervention. Advancements in endovascular repair have expanded the patient population to whom this approach may be offered and it is an acceptable option at centers experienced in these techniques. Ischemic injury remains a primary concern of both endovascular and open TAAA repair, and strategies for prevention of spinal and visceral ischemia continue to evolve, with spinal cord drainage and cold renal perfusion having the most evidence to support their use. Continued improvements in technique and innovations in endovascular technologies may further improve outcomes in the future. Irrespective of the approach chosen for TAAA repair, the relationship between surgeon and center experience and clinical outcomes cannot be understated, and significant consideration should be given to the choice of a high-volume center and a surgeon with expertise in TAAA repair, regardless of whether an open or endovascular technique is utilized.

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Footnote

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