

Review

Surgical Management of Acute Type A Aortic Dissection

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Abstract

Acute type A aortic dissection (ATAAD) is a life-threatening cardiovascular emergency characterized by disruption of the ascending aortic intima with subsequent propagation of a false lumen. Population-based studies report an annual incidence of approximately 3 cases per 100,000 patient-years. Despite advances in diagnosis and surgical management, 30-day operative mortality remains approximately 10–20%, with higher rates in the presence of malperfusion syndrome, cardiac tamponade, or aortic rupture. This narrative review summarizes the current understanding of ATAAD pathophysiology, classification systems, diagnostic modalities, and evolving management strategies, with particular emphasis on contemporary surgical techniques and organ protection strategies employed during repair. Hypertension remains the most significant modifiable risk factor for ATAAD, whereas hereditary thoracic aortic disease (HTAD) predisposes younger patients to dissection. The Penn classification stratifies mortality risk according to the pattern of malperfusion; coronary and mesenteric malperfusion are associated with the highest mortality. Surgical management has evolved from traditional hemiarch replacement to more extensive arch replacement, with or without frozen elephant trunk (FET) techniques, and emerging data demonstrate improved long-term durability. Moderate hypothermic circulatory arrest (MHCA) with antegrade cerebral perfusion (ACP) has emerged as the preferred neuroprotective strategy, offering outcomes comparable to those achieved with deep hypothermic circulatory arrest (DHCA) alone. Treatment at high-volume comprehensive aortic centers is associated with improved survival. Management of ATAAD continues to evolve through ongoing refinements in surgical techniques, organ-protection strategies, and perioperative care. While prompt surgical intervention remains the cornerstone of treatment, individualized management that incorporates patient-specific anatomy, malperfusion patterns, and institutional expertise is essential. Long-term surveillance and follow-up at specialized aortic centers are critical for detecting and managing late complications, and genetic evaluation is recommended to identify HTAD in appropriate patients.

Keywords: acute aortic syndrome; aortic dissection; ascending aorta; thoracic aorta

1. Introduction

Acute aortic syndrome (AAS) refers to a group of potentially fatal disruptions of the aortic intima, including intramural hematoma, penetrating aortic ulcer, and aortic dissection. In the setting of aortic dissection, an intimal tear allows blood to enter the media, forming a false lumen that propagates along the vessel [1,2,3]. Acute type A aortic dissection (ATAAD) describes any aortic dissection that involves the ascending aorta [4]. Emergency surgical evaluation is recommended due to the high risk of progression to aortic rupture, cardiac tamponade, acute aortic regurgitation (AR), and malperfusion syndrome [4,5,6,7]. The management of ATAAD has evolved substantially over recent decades, driven by advances in imaging and surgical techniques, including open and hybrid repairs [4,5,6].

2. Methods

Given the significant changes in our understanding of ATAAD in the last decade, the goal of this narrative review is to contextualize these new data, and explore emerging management strategies. References were selected for in-

clusion based on a query of Embase, PubMed, and Medline/OVID databases to identify studies published since 2010 using relevant keywords including “acute type A aortic dissection”, “acute aortic syndrome”, etc. Previous society-generated guidelines for management of ATAAD were reviewed, and an iterative process was used to add additional references to contextualize and evaluate these new publications [1,4,7,8,9,10].

3. Incidence and Severity

Meta-analysis of the incidence of ATAAD in population-based studies reports a mean annual incidence of about 3 per 100,000 patient-years [11]. However, given the rate of pre-hospital mortality for ATAAD, its true prevalence is likely higher. Post-mortem computed tomography was used to identify a 7% incidence of ATAAD in patients with pre-hospital cardiopulmonary arrest, and reported a 51% pre-hospital mortality rate for ATAAD [12]. Non-operative ATAAD management is associated with 57% mortality, while 30-day operative mortality for ATAAD ranges from 10–20% based on contemporary multi-center studies and registry reviews,



even higher (27% to 42%) in patients with associated malperfusion syndrome, pericardial tamponade, and aortic rupture [4,5,6,9,13].

4. Etiology and Risk Factors

Proposed mechanisms of ATAAD include a combination of underlying pathology that predisposes the aorta to mechanical failure, and a stimulating stress event that overwhelms aortic compliance leading to intimal tear formation [14,15]. Once an intimal tear develops, pressurized blood dissects into the media, forming a false lumen, separated from the true lumen by an intimal-medial flap. The release of chemokines from disrupted aortic smooth muscle cells leads to further inflammation, and weakening of the aortic wall [16].

Hypertension is the most significant modifiable risk factor for ATAAD development with an attributable risk of 54%, with tobacco smoking and dyslipidemia carrying attributable risks of 14% and 30%, respectively [17]. Notably, hereditary thoracic aortic disease (HTAD) that results in compromised integrity of the aortic media, including Marfan (0.8–12% prevalence of ATAAD), vascular Ehlers-Danlos (1.3–5.8%), Loeys-Dietz (16%), and Turner (1–5%) syndromes, is associated with early presentation of aortopathies [4,5,15,18]. Presence of bicuspid aortic valve, male sex, and advanced age are also associated with increased risk of ATAAD [5,6]. Pregnancy is a known trigger for ATAAD due to hemodynamic and hormonal changes that stress the aortic wall [19]. Before planned pregnancy, patients with HTAD are recommended to meet with a multidisciplinary team, undergo genetic counseling, and undergo surveillance aortic imaging [20,21,22]. In pregnant patients with HTAD, guideline-directed hypertension treatment can help prevent aortic dissection.

5. DeBakey, Stanford, TEM, and SVS/STS Classifications

The DeBakey classification categorizes aortic dissections based on the origin and longitudinal extent of the intimal tear [23]. The Stanford classification (Fig. 1A) is a widely adopted alternative that sorts aortic dissection into two groups based on the involvement of the ascending aorta. Both historic systems have limitations, especially in describing aortic dissections that begin in the aortic arch or involve complex propagation patterns. The Type-Entry-Malperfusion (TEM) classification (Fig. 1B) characterizes the dissection's extent, the entry location, and the presence of malperfusion [24]. These classification systems are described in Table 1.

The SVS and STS proposed a more granular classification (Fig. 1C) based on Ishimaru zones of proximal and distal extent of dissection as well as the location of the primary entry tear [7,20,25]. In this system, the aorta is broken up into zones 0–12 from the ascending aorta to the iliac arteries. The classical anatomic classifications remain essen-

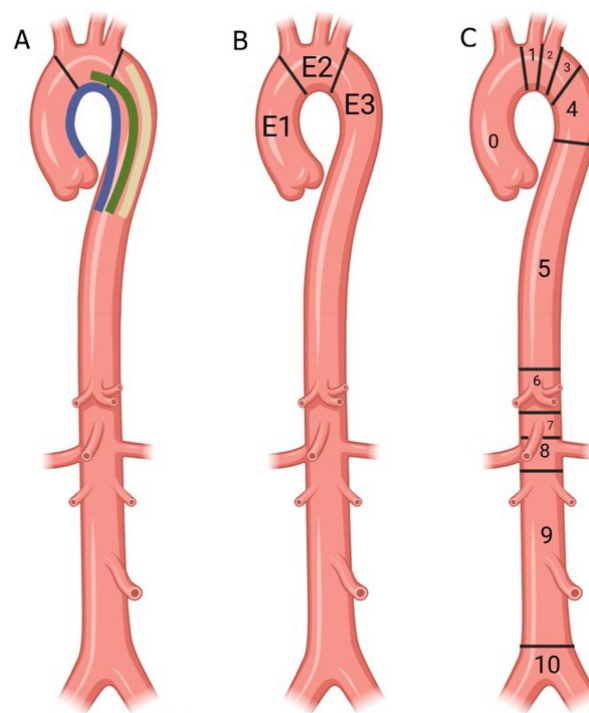


Fig. 1. Aortic dissection classifications. (A) Blue line: Stanford type A dissection; Beige line: Stanford type B dissection; Green line: non-A, non-B dissection. (B) TEM Entry locations. (C) STS/SVS zones. TEM, Type-Entry-Malperfusion; SVS/STS, Society for Vascular Surgery/Society of Thoracic Surgeons. Created in BioRender.

tial for rapid clinical triage and surgical decision-making, while the newer systems enhance precision in describing disease patterns that may influence management and outcomes, especially as endovascular management options of acute dissection are pursued. TEM is useful for prognostication and outcomes prediction, while STS/SVS provides detail for operative planning, with Stanford and DeBakey offering the fastest ways to communicate type of dissection.

6. Acuity

The timing of symptom onset in ATAAD has important implications for prognosis and management, with timing typically stratified as hyperacute, acute, subacute, or chronic [6,25]. Traditionally, dissections were dichotomized simply as acute (<14 days) versus chronic (≥14 days) from symptom onset to clinical presentation [23]. Investigators of the International Registry of Acute Aortic Dissection (IRAD) data further refined this approach by identifying four distinct inflection points in survival curves, regardless of management type, for ATAAD: hyperacute (symptom onset to presentation <24 hours), acute (2–7 days), subacute (8–30 days), and chronic (>30 days) [26]. These findings emphasize the importance of prompt identification and management of ATAAD [6,27,28].

Table 1. DeBakey, Stanford, and TEM classification systems.

DeBakey	Type I: originates in the ascending aorta and propagates through the arch and distally Type II: originates in, and is confined to, the ascending aorta Type III: originates in the descending aorta and rarely extends proximally
Stanford	Type A: dissection involving the ascending aorta, regardless of where the tear originates Type B: dissection doesn't involve the ascending aorta. Entry tear distal to the left subclavian Type non-A non-B: dissection involves the arch and descending but not the ascending
TEM	Type: Stanford A, B, or non-A non-B Entry: E0: no identifiable entry E1: entry in the ascending between the aortic valve and the proximal edge of the innominate E2: entry in the arch between the innominate and the distal edge of the left subclavian E3: entry in the descending aorta Malperfusion: M0: no clinical or radiologic malperfusion M1: dissection of at least 1 main coronary artery with or without myocardial malperfusion M2: dissection of at least 1 supra-aortic vessel or aortic arch true lumen collapse with or without cerebral or upper limb malperfusion M3: dissection of at least 1 visceral, renal or iliac artery or aortic true lumen collapse with or without mesenteric, renal, or lower limb malperfusion

7. Clinical Presentation and Diagnosis

Patients with ATAAD typically present with abrupt onset of severe pain, most commonly in the chest or upper back and often described as tearing, ripping, or sharp, with maximal intensity at onset. On physical examination, patients may exhibit widened pulse pressure, new diastolic murmur (representative of AR), asymmetrical or diminished peripheral pulses, and interarm blood pressure discrepancies. Muffled heart sounds, distended jugular veins, hypotension, and electrical alternans on electrocardiogram (ECG) may indicate aortic rupture into the pericardium [4,5,6,29]. End-organ malperfusion can result in varied presentation (see Malperfusion section).

When clinical suspicion for ATAAD is high, computed tomography (CT) angiography (CTA) of the chest, abdomen, and pelvis should be pursued promptly to confirm the diagnosis and identify the location of the intimal tear, the extent of the dissection, patterns of flow in the true and false lumens, branch-vessel involvement, and any associated pericardial or pleural effusions [29,30,31]. For stable patients, adjunctive tools such as echocardiography and biomarkers (e.g., lactate, troponins, and creatinine) may assess for malperfusion. In hemodynamically unstable patients who may not tolerate CTA, preparation for emergency surgical intervention should occur simultaneously with emergent imaging modalities such as bedside transthoracic echocardiography and transesophageal echocardiography (TEE) [29,31].

Once imaging confirms the diagnosis, prompt activation of the surgical team is essential [4,9]. Future directions of study include improving risk-prediction models to identify high-risk individuals for perioperative complications

based on imaging findings, biochemical markers, and patient factors, beyond those that exist currently (see Malperfusion syndrome section).

8. Initial Management and Stabilization

Initial management of ATAAD focuses on immediate medical stabilization to reduce aortic wall shear stress, limit propagation of the dissection, control pain-induced sympathetic activation, and preserve end-organ perfusion while preparing for definitive surgical repair [3,4,31,32,33]. Patients should undergo continuous telemetry and blood pressure monitoring, with prompt establishment of large-bore intravenous access [34]. Monitoring and documentation of peripheral pulses and neurologic exam are essential for early identification of malperfusion symptoms both in the preoperative and postoperative setting. Placement of arterial lines in both the upper and lower body may be helpful, particularly in patients with concern for arch vessel or lower extremity malperfusion [7,35]. This allows for monitoring of peripheral perfusion during and after intervention [34]. However, placement of these lines should not delay definitive operative management. Adequate analgesia, typically with intravenous opioids, should be administered early to relieve pain and blunt the catecholamine surge that can exacerbate hypertension and shear stress [1].

The cornerstone of medical therapy for ATAAD is anti-impulse therapy, with intravenous β -blockers as first-line agents, targeting a heart rate of less than 60 beats per minute and systolic blood pressure of 100–120 mmHg [9,29]. Esmolol is favored for its rapid onset and short half-life, but labetalol can be used as an alternative [9,32,36]. For patients with cardiac tamponade, emergent surgery

should be performed, with consideration of the use of controlled pericardiocentesis only in the case of tamponade-related hemodynamic instability, as a bridge therapy in the case of substantial delay from presentation to appropriate definitive surgical management [1,37,38].

9. Timing of Intervention

Given the high risk of aortic rupture, cardiac tamponade, and death associated with ATAAD, current guidelines recommend immediate surgical evaluation and emergent operative repair once the diagnosis is confirmed or strongly suspected in high-risk patients [4,9]. Older studies have consistently demonstrated a clear time-dependent risk of mortality with delays to definitive repair, with mortality increasing by approximately 1–2% per hour in the first day in the absence of surgery, with a recent report describing a 2.6% hourly mortality rate for the first day [6,9,26,27,28,39,40].

10. Transfer Considerations to Dedicated Aortic Surgical Centers

Prior studies have demonstrated that patients with ATAAD experience reduced risk of morbidity and mortality when undergoing surgery at high-volume aortic institutions [41]. When ATAAD is confirmed at a non-surgical facility, rapid transfer to a dedicated aortic surgery center with ongoing medical stabilization is recommended for hemodynamically stable patients [42,43,44]. In unstable patients, emergent local surgery is appropriate if immediately available; otherwise, carefully selected patients may still experience significantly improved survival outcomes after expedited transfer to an aortic surgery center following limited stabilization, coordinated closely with the receiving center [42,43,44,45,46,47]. Efficient interfacility transfer, with direct clinician communication and prompt transmission of imaging and clinical data, reduces delays. In fact, patients transferred alive for definitive repair may achieve outcomes comparable to direct admissions when transfer is timely and well-coordinated [48]. While time from admission to surgery at a dedicated aortic surgery center for ATAAD has significantly decreased from a median of 7.1 hours to 6.5 hours, there continues to be room for improvement with education and quality control efforts at centers of all levels of care [49].

Given the emergent nature of ATAAD management, organizations have implemented dedicated aortic emergency phone numbers for referring hospitals, direct-to-operating room aortic emergency transfer programs, and algorithms designed to streamline steps between the presenting emergency department and operating room of the definitive aortic center [50]. When the external site refers a patient to the aortic surgical team, the decision to accept transfer to the OR immediately activates the OR team such that, when the patient arrives, the site of definitive management is ready for immediate management [50]. This results in significantly decreased time from transfer acceptance to

OR and time from hospital arrival to OR [50]. Organizational development of aortic surgery teams is critical in providing timely care for patients with aortic pathologies [42,51].

11. Malperfusion Syndrome

Pre-operative malperfusion in patients with ATAAD occurs with an incidence of 15–40% and is an independent risk-factor for in-hospital mortality [6,9,13,52]. In patients with ATAAD, malperfusion syndrome is associated with a significantly higher rate of in-hospital mortality than patients without malperfusion (30.5% vs 6.2%) [53]. Malperfusion, or inadequate blood supply to an end organ, can lead to malperfusion syndrome, which results from sustained ischemia of one or more end-organs, often as a result of branch vessel obstruction.

Physiologic presentations of malperfusion include neurologic, cardiac, renal, mesenteric, and limb. Cerebral malperfusion may present with syncope and neurological deficits, including coma and stroke [5,6,29]. Spinal malperfusion may present with motor or sensory deficits in the lower extremities [9]. Coronary malperfusion may present with chest pain and cardiogenic shock in the setting of myocardial ischemia or infarction [9]. Renal malperfusion may manifest as decreased urinary output. Mesenteric malperfusion presents with abdominal pain, and limb ischemia may present with limb pain, and motor and/or sensory deficits [13,53,54,55,56,57].

Preoperative malperfusion represents an important predictor of early mortality after surgical repair [13,53,58,59,60,61]. The University of Pennsylvania (Penn) classification system is a tool used to understand the impact of malperfusion on surgical outcomes. Patients are classified into one of four classifications (A, B, C, and B-C). A represents hemodynamic stability without signs or symptoms of ischemia. B represents hemodynamic stability with evidence of branch vessel malperfusion or symptoms of localized ischemia (e.g., stroke, ST segment changes, abdominal pain, limb ischemia). C represents global malperfusion from circulatory collapse. B-C involves global malperfusion from circulatory collapse plus evidence of local ischemia [52]. In a recent single-center review validating the Penn classification, Patrick and colleagues [52] identified the following 30-day mortality risk, stratified by Penn classification: A–5.2%, B–10.3%, C–15.2%, B-C–35.3%. There are several risk stratification tools validated for predicting mortality risk in patients with ATAAD with or without malperfusion that perform reasonably well including Penn (area under the curve (AUC) 0.77), German Registry for Acute Aortic Dissection Type A (GERAADA) (0.73), and ALICE (0.83) [1,5,13,27,52,59,62].

In addition to classification systems that stratify patients by degree of malperfusion, such as the Penn classification, specific malperfusion location is highly asso-

ciated with mortality risk. A recent review of the STS database identified the following rates of malperfusion in a contemporary series of surgically repaired ATAAD from 2017–2020: cerebral (9.8%), coronary (5.0%), renal (10.2%), mesenteric (5.7%), extremity (14.9%), and spinal malperfusion (1.0%) [13]. In this study, the authors identify the highest adjusted odds of 30-day mortality associated with coronary (OR 2.36) and mesenteric (OR 1.84) malperfusion, which is consistent with other large series [9,13,53,54,55,60].

Traditionally, immediate proximal aortic repair has served as the primary management of pre-operative malperfusion, restoring true lumen flow and resolving branch-vessel occlusion [9,53]. Surgical intervention mitigates the immediate risk of fatal complications, corrects AR and myocardial ischemia, and often reestablishes flow to malperfused vessels [9,53]. In some cases, peripheral revascularization may be performed in the setting of ongoing malperfusion following aortic repair.

More recently, select centers have advocated for targeted revascularization of mesenteric malperfusion in stable ATAAD patients using endovascular fenestration, branch-vessel reperfusion, or hybrid before primary open repair [63,64]. By restoring mesenteric perfusion via endovascular approaches before proximal aortic repair, the risk of ongoing mesenteric ischemia during cardiopulmonary bypass (CPB) is minimized. Various centers have reported reduced risk of in-hospital mortality associated with this approach [9,63,64,65]. However, it is important to note that delayed proximal aortic repair is not without risk, and patients may experience increased risk of aortic rupture during complex mesenteric reperfusion procedures [9,63]. For example, Yang and colleagues [64] describe their approach to endovascular intervention on mesenteric malperfusion with delayed proximal aortic repair; in the first decade of their series, 16% died from aortic rupture before open repair, improving to 4% in their second decade. However, those who reached open surgical intervention for proximal repair experienced a remarkably low operative mortality of 2% [64]. Additionally, Leshnower and colleagues [63] performed a single-institution retrospective review of a thoracic endovascular aortic repair (TEVAR)-first approach to ATAAD with mesenteric malperfusion. Patients with mesenteric malperfusion without shock, tamponade, rupture, or severe AR were selected to undergo TEVAR-first; while this likely represents a lower risk cohort, the TEVAR-first patients experienced a significantly reduced in-hospital mortality of 30%, versus 69.2% in the cohort of patients undergoing primary open repair first [63]. TEVAR-first management of mesenteric malperfusion is an investigational strategy, with expert aortic care centers demonstrating reasonable outcomes, while current guidelines support proximal aortic surgery first [63,64,65,66]. Future multicenter prospective investigations into TEVAR-first management will help inform future guideline development.

The case of ATAAD complicated by cerebral malperfusion is associated with a significant increase in morbidity and mortality [52,55,60,67]. In the case of cerebral malperfusion with carotid occlusion, no evidence-based consensus has been established [67,68,69]. Surgical options include restoration of cerebral blood flow via conventional central aortic repair or early restoration of cerebral perfusion (either via early operative branch perfusion, stenting, or via transcatheter carotid artery perfusion cannula placement) [68]. While central aortic repair continues to be the most common practice, in cases of occlusion of the common carotid arteries, cannulation or anastomosis distal to the carotid occlusion to establish early antegrade cerebral perfusion (ACP) has been performed to minimize cerebral ischemia time before central repair. This is often followed by carotid transection and false lumen thrombectomy then aortic reconstruction [68]. Others have found preliminary success using MRI to evaluate for salvageable cerebral tissue, and if salvageable, performing early carotid artery stenting [69].

In addition to tailoring repair approach based on presence and type of malperfusion, other centers have advocated for stratifying patients based on timing of presentation. A recent, single-center retrospective review assessed the outcomes of a traditional approach to an individualized approach with situational reperfusion-first ATAAD complicated by malperfusion [67]. In-hospital mortality in the traditional cohort was 33.9%, versus 10.2% in the individualized cohort [67]. These data from select expert aortic care centers are promising but expectations should be tempered until multi-center prospective evidence emerges. Guideline-supported practice continues to be traditional central repair first. A suggested algorithm is presented for operative decision making in Fig. 2.

12. Cannulation Strategies

Prompt, safe, and reproducible arterial and venous cannulation strategies to establish CPB flow are key to reestablishing true lumen systemic perfusion. Given the variation in anatomic features and presentations of ATAAD, there is not a one-size-fits-all approach to arterial cannulation. Historically, the femoral artery was the preferred site of arterial cannulation given ease of access prior to sternotomy; however, more recently, right axillary, innominate, and central aortic cannulation have gained popularity with evidence of excellent reproducibility and safety [70]. Regardless of arterial cannulation strategy, it is vital to ensure that CPB flow is established in the true lumen.

While its use has declined over time, percutaneous or open femoral artery cannulation can be useful in the setting of hemodynamic instability or when rapid CPB is required, due to the ease of rapid access and exposure, before or during sternotomy. However, the femoral approach carries inherent risks associated with its retrograde perfusion, including the risk of retrograde embolization and false lumen pressurization. A 2021 meta-analysis found that ax-

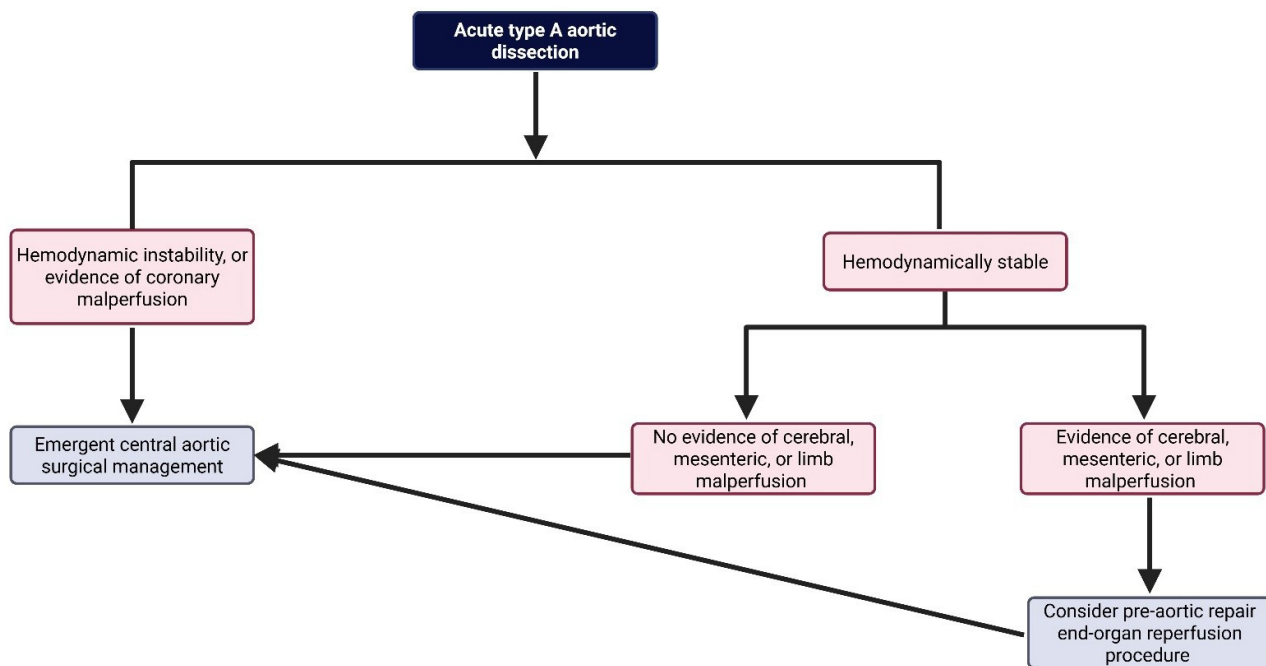


Fig. 2. Surgical decision-making algorithm. Created in BioRender.

illary arterial cannulation was associated with significantly reduced odds of stroke when compared with femoral artery cannulation [71]. Despite the risks associated with femoral arterial cannulation for CPB, it remains a relatively fast option for cannulation of patients with hemodynamic instability or challenging dissection anatomy [72,73].

A recent review of International Registry of Acute Aortic Dissection (IRAD) data has identified the axillary artery as the most common arterial cannulation site, with global use of 51% but significant regional variation in practice patterns [73]. The right axillary artery is often favored as a cannulation site given the ability to establish prompt antegrade true lumen flow prior to sternotomy, as well as the ability to provide ACP during circulatory arrest via clamping of the innominate artery [72,74]. The axillary artery is typically approached by a second incision near the deltopectoral groove, with cannulation either via side-graft technique or direct cannulation [75]. Retrospective data comparing direct cannulation of the axillary to side-graft approach demonstrated significantly reduced incidence of cannulation-related complications (stroke and iatrogenic axillary artery dissection) in the side-graft group [76]. Axillary cannulation is a safe and effective option if the dissection extends into the origin of the innominate, but not if it extends into the axillary artery itself. Delivery of ACP via axillary cannulation should be avoided in the case of anomalous right subclavian artery originating from the descending thoracic aorta [75].

Similar to axillary artery cannulation, cannulation of the innominate artery eases the delivery of ACP during hypothermic circulatory arrest, without the need for an additional incision [73]. By clamping the proximal innominate

artery, the same cannula can be used to deliver ACP during hypothermic circulatory arrest.

Central aortic cannulation can be performed using the Seldinger technique and utilizing epiaortic ultrasound or TEE, surgeons can carefully deliver a wire and cannula into the true lumen. A single-institution retrospective cohort demonstrated an in-hospital mortality rate of 10%, and an overall stroke rate of 13% [70]. In the case of inadvertent false lumen cannulation, elevated pump pressures will be noted after initiation of CPB [72]. In extreme cases, such as with aortic rupture or circumferential dissection of the ascending aorta, the aorta may be opened and the wire or cannula is introduced under direct visualization (“Samurai” cannulation) [77]. While previously debated, several recent series have demonstrated comparable surgical outcomes among femoral, axillary, direct aortic, and branch vessel cannulation in terms of stroke, mortality and bleeding, while others show increased risk of postoperative stroke with femoral artery cannulation during ATAAD repair with total arch replacement (TAR) with frozen elephant trunk (FET) [78,79,80,81].

13. Management of the Proximal Aorta—Aortic Root and Valve Interventions

The aim of surgical repair of ATAAD is resection of the entire ascending aorta and the primary intimal tear, with restoration of true lumen flow [9]. Additionally, any aneurysmal portion of the aorta should be replaced, and aortic valve competence should be ensured.

The dissection must be evaluated pre-operatively to determine the complexity of the arch dissection, considering the time that definitive repair will take, and parsing

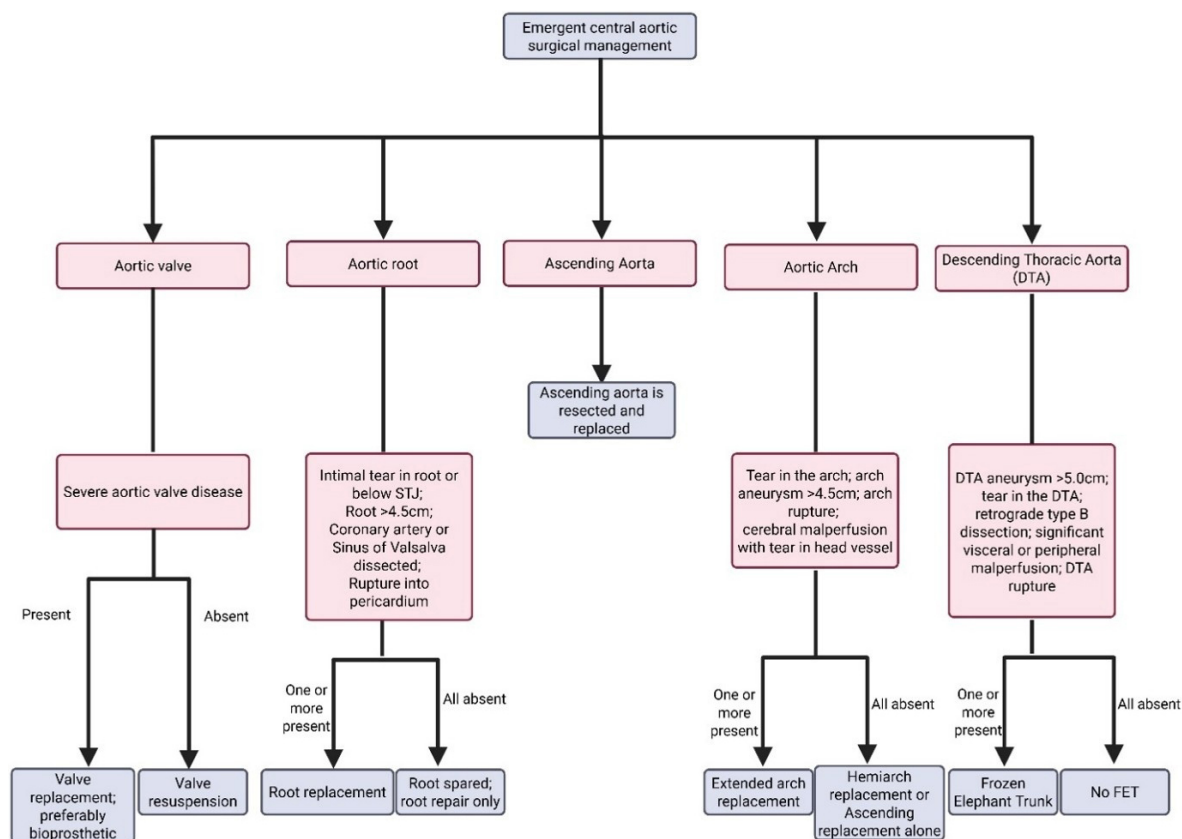


Fig. 3. Central figure: surgical management of the aorta in ATAAD. ATAAD, Acute type A aortic dissection; STJ, sinotubular junction; FET, frozen elephant trunk. Created in [BioRender](#).

out which steps must be taken under circulatory arrest, and which can be performed during CPB (often while cooling or rewarming) [9,82,83]. This can guide the order of proximal and distal repair – generally the proximal intervention is completed during CPB (especially during cooling and, if necessary, warming), with distal management undertaken during hypothermic circulatory arrest. An algorithmic approach to the surgical management of ATAAD is presented in Fig. 3.

The timing and decision for cross clamp placement is key in managing the proximal aorta. If aortic cross clamping will cause malperfusion, it is unsafe and cannot be performed. In cases of relatively simple arch dissection and clamping will not cause malperfusion, then aortic cross clamping can be performed and repair should begin proximally. In the presence of severe AR, which may manifest with left ventricular distension, aortic cross clamping can assist in preventing retrograde flow from distending the ventricle. However, if cross clamping is deemed unsafe due to high complexity, risk of malperfusion, or concomitant intramural hematoma, then the patient should be cooled and circulation should be arrested without cross clamp placement, and repair should begin in the arch, not proximally [9,82,83].

Proximally, the surgeon must closely evaluate the root for pathology that warrants replacement. If a primary inti-

mal tear is located in the aortic root, below the sinotubular junction (STJ), aortic root replacement is indicated. Additionally, if the aortic root meets size criteria for replacement (typically >4.5 cm), or the coronary arteries are severely dissected, an aortic root replacement should be performed [66].

Next, the aortic valve must be closely inspected. Retrograde propagation of the intimal flap often extends to the level of the root. When this occurs, it is possible to observe catastrophic failure of the aortic valve or valve-supporting structures, either via distortion of root geometry or dynamic prolapse of the intimal flap across the aortic valve. Retrograde propagation, in addition to native valve pathology that is contributing to AR or aortic stenosis, should guide surgical repair strategies [84,85,86,87]. Other key factors that may influence management of the aortic root include compression or deformation of coronary ostia by the false lumen, which can cause coronary malperfusion and myocardial infarction (MI) in up to 5% of patients [88]. Ostial reconstruction or a Cabrol procedure may need to be performed to preserve coronary perfusion [88,89]. If there are any concerns about patency of the coronary arteries, or there are regional wall motion abnormalities on the pre-operative (or post-operative) TEE, this should be addressed with coronary artery bypass before the patient leaves the operating room [88,90].

Proximal aortic rupture into the pericardial space and subsequent cardiac tamponade can also be devastating, increasing the risk of in-hospital mortality among those receiving surgery 2.58-fold [91]. In these cases, the aorta is often friable and malperfused, increasing the technical difficulty of repair, guiding surgeons towards root replacement [66,84,92].

Once the surgeon has decided that the root should be replaced, the next question is how. The main options are valve sparing aortic root replacement (VSARR) and composite valve-graft root replacement. Time from diagnosis to completion of operative intervention is an essential risk factor for post-operative morbidity, and composite valve-graft root replacement offers improved operative speed [28,93]. Recent retrospective cohort studies report that in the setting of ATAAD, the Bentall procedure was associated with shorter CPB times (260 minutes vs 291) and aortic cross clamp times (199 minutes vs 259) when compared to VSARR [94,95,96,97]. If root replacement is indicated in patients who are unstable, Bentall is the preferred method.

Candidates for VSARR should be young, hemodynamically stable, have pliable native aortic valves (bicuspid or tricuspid and without calcification or dissection), and be performed by surgeons who are able to perform VSARR [21,84,85,97]. ATAAD patients with elevated potential for morbidity, such as those with cardiogenic shock, myocardial ischemia, or severe cardiac tamponade, require expeditious definitive repair and therefore composite valve-graft aortic replacement may be more appropriate [9,84,97,98].

VSARR is performed via one of two techniques—the David (reimplantation) procedure or the Yacoub (remodeling) procedure. Given that the Yacoub procedure is often contraindicated in ATAAD, due to its reliance on annular structural integrity, the David procedure is more frequently performed [99]. After VSARR, the native annulus is contained within a Dacron graft, theoretically reducing the risk of future annular dilation [87]. A 2024 analysis of the European registry of ATAAD identified 139 patients who underwent the David procedure with a 10-year mortality rate of 30.1% and a proximal aortic reoperation rate of 3.9% [95]. Other cohorts have reported operative mortality between 0% and 15.9% for VSARR in patients with ATAAD [84,98,99]. The durability, long term quality of the aortic valve, and lack of need for anticoagulation, make VSARR an ideal solution for ATAAD requiring root replacement in young patients, and those with connective tissue disorders [7,9,66,84].

The majority of patients requiring aortic root replacement at the time of ATAAD repair will undergo a composite valve-graft conduit placement, initially described by Bentall and De Bono [100]. Older patients unlikely to benefit from VSARR, those who are unstable at the time of intervention, those with pre-existing valve disease or severe calcification, endocarditis or irreparable root destruction should undergo composite root [97,100,101]. Briefly,

the surgeon excises the aortic root including the valve, and implants a composite valve-graft to the left ventricular outflow tract [100]. This process produces a technically simpler approach to aortic root replacement that is less dependent on procedure-specific expertise when compared to valve-sparing root replacement and is more reproducible [84,102]. The Michigan experience comparing the Bentall procedure to VSARR demonstrated that operative mortality outcomes were not significantly different but the composite outcome of MI, stroke, renal failure, and operative mortality significantly favored the David procedure [97]. A single-center study of 314 patients found a short-term mortality rate of the Bentall procedure of 26%, which exceeds the rate of other studies that range from 9.7%–12.9% [95,101]. Furthermore, a 2025 meta-analysis of 4212 aortic root replacement patients found that freedom from reoperation rates were 98.9%, 95.9%, and 90.2% at 1, 5, and 10 years, respectively [103]. Both the Bentall procedure and VSARR are viable options for root replacement, and electing to pursue one or the other must be tailored to fit patient- and surgeon-specific situations, with the emergency setting encouraging many to pursue Bentall given its significantly shorter CPB and aortic cross clamp times.

After evaluation of the aortic root, if the decision is made not to undergo aortic root replacement, aortic root repair should occur. Briefly, the native aortic sinus is preserved, the aortic root is trimmed, false lumen thrombus is removed, the valve is resuspended, and the root is reconstructed and reinforced with inverted intima or felt [66,92,103]. In reapproximating dissected aortic layers, most surgeons have success with neomedia or Teflon felt support, while biologic glue has fallen out of favor due to increased complications of re-intervention [66,92,94,103,104].

A 2014 University of Pennsylvania retrospective study of 489 ATAAD patients analyzed the efficacy of resuspension with felt neomedia repair and found survival rates of 69%, 50%, and 36% at 5, 10, and 15 years, respectively [104]. Impressively, freedom from reoperation at the proximal anastomotic site was 96%, 92%, and 89% at 5, 10, and 15 years, respectively [104]. These data suggest that valve resuspension with adequate aortic wall support remains durable in the long-term. However, leaving the native aortic root tissue that may be weakened by dissection and inflammation can lead to long-term aneurysmal dilation of the root, eventually requiring a complex redo root replacement [92,102,103]. When technically feasible, prioritizing aortic valve repair over replacement is preferred [4,84].

Once the management strategy of the root is selected, a critical decision regarding the management of the valve remains—can the valve be spared or does it need to be replaced? As alluded to above, this often relies on the health and structure of the valve itself [84,85,96,99]. If the valve needs to be replaced, the surgeon needs to decide whether a mechanical or bioprosthetic valve is ap-

appropriate. Modern bileaflet mechanical valves have excellent hemodynamics and long-term survival. Yet, mandatory lifelong warfarin use increases the risk of major bleeding, even with lower INR targets (1.5–2.0) [8,86,105]. Bioprosthetic valves avoid lifelong warfarin requirement, but there remains a concern regarding their durability in young patients, as they are more prone to requiring reoperation within 10 to 15 years [106]. Single-institution data comparing mechanical Bentall to bioprosthetic Bentall for ATAAD in patients younger than 65 demonstrated no survival advantage of mech-Bentall compared with bio-Bentall at 10 years, and mech-Bentall was associated with a significantly higher rate of stroke and major bleeding [107]. When the aortic valve cannot be spared, bio-Bentall should be strongly considered in ATAAD, even in young patients.

Aortic root management in ATAAD necessitates rapid evaluation of anatomic, individual, and institutional factors. A reasonable approach involves deciding whether the root needs to be replaced. If the root needs to be replaced—determine which type of replacement is most appropriate (VSARR or Bentall). If the root does not need to be replaced, repair the root and resuspend the valve. If the native valve is damaged beyond repair, then valve replacement and ascending intervention are appropriate.

14. Distal Aortic Repair Strategies and Considerations

After assessing the proximal aorta, surgeons must examine the distal aorta on CTA and intra-operatively, evaluating arch diameter, arch vessel anatomy, and location of intimal tear as part of a tear-oriented approach [7,9]. Most patients with ATAAD can be effectively treated with complete resection of the ascending aorta and hemiarch replacement to manage the arch. However, in patients with ATAAD, extended arch intervention is warranted if the primary entry tear is in the arch [9,108]. It may also be warranted if the tear is in the proximal descending aorta, there is brain or peripheral malperfusion, or if there is arch or descending thoracic aneurysm or rupture [108,109,110,111]. Further, extended aortic arch replacement with FET should be considered to provide favorable aortic remodeling and extended aortic arch replacement may be considered in cases of ATAAD in HTAD patients [109,112,113,114,115,116,117].

The fundamental difference between hemiarch replacement and extended arch intervention lies in the extent of aortic tissue replacement, and the consequent complexity of the repair, which is reflected in the operative metrics. Hemiarch replacement involves excising the ascending aorta and extending the graft obliquely into the proximal aortic arch, the lesser curvature, with the distal anastomosis being performed slightly inferior to the origin of the innominate artery [118,119,120]. Conversely, extended arch intervention typically refers to TAR with or without FET. TAR involves the excision of the aortic arch and replacement. This requires the reimplantation of some or all 3

arch vessels onto either a new graft individually (branched graft technique) or all together on shared tissue (island technique) [110,121]. This can be performed with distal anastomosis in zones 1–3, with zone 3 being the most common [83,122]. Typically, a zone 2 anastomosis involves more robust aortic tissue with a better aorta-to-graft size match, while zones 0–1 have improved exposure and are more feasible [83,123,124,125]. With zone 2 arch anastomosis, the left subclavian artery (LSCA) may be covered by concomitant FET or classic elephant trunk (cET). Therefore, the LSCA should either be debranched (locally or extra-anatomically), managed with FET endoprosthesis that includes a subclavian branch, or managed with subsequent TEVAR [83,112,126]. Zone 2 anastomosis is less extensive, results in reduced lower body perfusion arrest time, improved hemostasis, and reduced rate of recurrent laryngeal nerve palsy [122,125]. In the event of hemodynamic instability, or significant comorbidity precluding more complex repair, one can consider less-extensive repair options individualized to the patient. For a proximal tear in the lesser curvature of the arch, with distal extension, one can consider proximal hemiarch replacement with distal TEVAR with partial left subclavian coverage [108,111]. Branched stented anastomosis FET repair, with various configurations selected based on anatomy of dissection, has demonstrated safe and effective early outcomes for ATAAD with multi-segment aortic dissection including a tear in the arch [126]. Finally, while a tear in the arch should be managed with an arch replacement, in extreme cases of hemodynamic instability in relatively poor operative candidates, some may consider hemiarch replacement with simple primary tear repair with felt to perform the fastest possible repair—accepting the increased risk of subsequent aneurysmal degeneration, malperfusion, or rupture with the knowledge that subsequent arch management will be needed in more stable conditions [9]. However, whenever possible—it is essential to attempt to resect or exclude the primary entry tear and perform arch replacement when appropriate [7].

One of the primary concerns with TAR is its potential to increase perioperative morbidity due to increased duration of circulatory arrest time and, traditionally, increased duration with inadequate cerebral perfusion [108]. Meta-analysis of studies comparing TAR versus hemiarch replacement in ATAAD found TAR to be associated with a statistically significant increase in 30-day mortality (OR 1.79), renal failure requiring dialysis (OR 1.34), and stroke (OR 1.49). TAR was also associated with a non-statistically significant increase in long-term mortality (HR 1.25) non-statistically significant decrease in unplanned aortic reoperation (HR 0.53), and a statistically significant decrease in late aortic events (HR 0.53), though subgroup analyses showed higher rates of preoperative malperfusion in the TAR group [111]. A 2021 retrospective cohort study with 365 patients compared the time stratified risks of mortality and neurological injury between the two groups and found

that in the post 2009 era, risks were comparable [127]. One study found actuarial survival at 10 years was 49% versus 66% for hemiarch and TAR, respectively, and freedom from reoperation at 10 years was 89% versus 98%, respectively [128]. Similarly, a 2022 meta-analysis of 5243 patients found a significant decrease in the risk of reoperation after 7 years in those who underwent TAR when compared to hemiarch, and TAR yielded a significantly lower risk of mortality at 3 months post-operation [129]. A recent meta-analysis of long-term outcomes suggests significantly improved overall survival with TAR compared with proximal aortic replacement in ATAAD, with significantly lower reoperation rates in patients who underwent TAR [129]. Hemiarch and TAR both play an essential role in the management of ATAAD. The decision to perform a TAR should be individualized to the patient's situation based on the presence of indication (tear in the arch, arch aneurysm, circumferential arch dissection, or connective tissue disease), patients' comorbidities and likelihood of living long enough to see a long-term benefit, and the surgeon's ability to perform a TAR safely [111].

When performing TAR, the order of operations can impact the time of distal organ, and cardiac ischemia and the duration of hypothermia. For example, in branch-first TAR, a trifurcated graft is utilized, and the other head vessels are perfused and provide cardiac perfusion during the third vessel reconstruction. However, others prefer to address the head vessels from distal to proximal during circulatory arrest. Ultimately, the decision of branch vessel order should be individualized to the patient's anatomy and the dissection pathology [130].

While performing ATAAD repair that does not require extended arch intervention, there exists some debate regarding the role of a true hemiarch replacement under circulatory arrest, versus a "clamp distal", or an ascending aortic replacement without circulatory arrest [82,118,119,120,131,132]. The conventional approach is to perform the distal anastomosis via an open-distal technique, which permits extension of the resection to the lesser curvature (and extirpation of the cross-clamp site when applicable) and may reduce the risk of false aneurysm formation, false-lumen pressurization, and creation of new entry tears [9,118,119,120]. For most patients, hemiarch replacement remains the preferred strategy. On rare occasions when a patient is unlikely to tolerate circulatory arrest, the operative plan must be highly individualized—for example, limited ascending replacement without circulatory arrest may be chosen as a temporizing measure, with acknowledgment of the increased long-term risk of aneurysmal degeneration or rupture and the likely need for future intervention [7,9,51].

For patients with aneurysmal or dissected descending thoracic aortas, performing a FET will provide an adequate landing zone for staged TEVAR to cover additional tears distal to zone 3, stent open the true lumen in the setting of compression and malperfusion, and encourage healthy

aortic remodeling [112,114,115]. The FET technique was initially described using antegrade deployment of a typical TEVAR device into the descending thoracic aorta, and now often utilizes a specialized hybrid FET graft, a composite of a proximal, non-stented fabric tube and a distal, self-expanding stent graft (e.g., Thoraflex Hybrid FET device) [133,134]. The FET evolved from a two-stage cET procedure involving replacement of the ascending aorta and aortic arch with a non-stented vascular graft followed by a second stage in which a new graft was sewn into the free "trunk", thus extending the repair distally [113,135]. The obliteration of the false lumen promotes its eventual thrombosis, which helps prevent late aneurysmal degeneration [127]. False lumen thrombosis has been reported in up to 92% of patients with ATAAD who underwent FET, with positive remodeling occurring in up to 80% of patients [116].

There are risks associated with performing FET. A 2013 meta-analysis of 8 studies found the overall mortality for patients with ATAAD who underwent TAR with FET was 7.7% (range, 0–18.2%) [109]. A 2025 Taiwanese study of 5769 patients found that when compared to non-FET groups, the in-hospital mortality for those who underwent TAR with FET was significantly higher (22.4% vs 18.0%) [115]. The risk for new onset stroke significantly increases in FET populations [115]. There is also a risk of spinal cord injury/ischemia as a result of using longer, multi-level covered stent grafts [9]. The risk of spinal cord injury is reported to be 2.4% (95% CI, 1.3–4.2%) in patients with ATAAD managed by FET, and this can be significantly decreased by limiting the degree of aortic coverage by the FET to at, or above, T8, or by limiting use of commercial stent grafts to 10-cm length, and avoiding 15-cm length [136,137,138,139]. For postoperative spinal cord ischemia, lumbar cerebrospinal fluid drainage can be used to lower intrathecal pressure and thereby improve spinal cord perfusion pressure; prophylactic use of drains is currently under investigation [140].

In contrast to traditional FET techniques utilizing covered aortic stent grafts, the Ascyrus Medical Dissection Stent (AMDS) is a recently developed hybrid prosthesis meant to augment hemiarch replacement procedures with a proximal cuff of polytetrafluoroethylene (PTFE) and a distal uncovered nitinol stent. This device is primarily indicated for ATAAD with a primary entry tear in the ascending aorta and either clinical or radiographic distal malperfusion. The goal of the AMDS prosthesis is to augment true lumen flow and expansion using an uncovered stent, and seal the distal anastomosis and prevent distal anastomotic new entry tears (DANE) [141,142]. Initial outcomes demonstrate similar operative mortality and in-hospital morbidity, with increased false-lumen thrombosis and decreased rate of DANE, compared to hemiarch alone [143]. The early PERSEVERE results are encouraging: in this particularly sick cohort (ATAAD with a zone 0 tear and malperfusion), the device achieved high technical success and was asso-

ciated with substantially lower 30-day mortality (9.7% vs 34.6%) and fewer patients experiencing one or more major adverse events (26.9% vs 58.0%) compared with the reference cohort [144].

Additional emerging investigations into the management of the arch include the ARTIZEN trial evaluating the safety and effectiveness of the Arcevo™ (Artivion, Inc) self-expanding aortic stent graft with an integrated stented left subclavian branch preloaded onto a delivery system for use during the FET procedure, preventing coverage of the left subclavian [126,145]. Finally, pre-clinical studies demonstrate the technical feasibility, decreased time for technical anastomosis, and improved anastomotic quality in aortic surgery with aortic end-to-end anastomotic stapler device use [146]. While these developments from select expert aortic care centers for the ARTIZEN trial and use of AMDS are promising, further study is essential before they drive evidence-based guidelines.

15. Endovascular Management of ATAAD

While endovascular management of type B aortic dissection is well described, endovascular management of ATAAD is not guideline-directed or the standard of care. Nevertheless, for high-risk patients deemed unsuitable for open repair, there is growing interest in developing and refining endovascular management strategies [147,148,149]. The anatomic feasibility of commercially available stent grafts to treat ATAAD is low. The ascending aorta is frequently dilated and the proximal landing zone short, with the entry tear often very close to the STJ—conditions that make secure sealing difficult and increase the risk of endoleak, coronary encroachment, aortic valve distortion, or compromise of innominate flow [148]. Fenestrated or branched valve-carrying conduits may overcome these problems, creating a more proximal landing zone because they can be anchored in the annulus [150]. Endovascular aortic root repair in the setting of ATAAD has been executed successfully using physician-modified composite grafts [151]. While reviews describe achievable anatomy for endovascular approach and there are case series with reasonable short-term outcomes, the technique remains experimental. Until long-term outcomes are available, and better-fit devices are commercially available, open surgical management will likely be the gold standard supported by guidelines. The ARISE multicenter investigation is evaluating the efficacy of an ascending stent graft for use in patients with ATAAD who are deemed high-risk surgical candidates with amenable anatomy, with promising early results (20% 30-day mortality, 6.7% stroke with a high degree of technical success) [152].

16. Organ Protection During Aortic Cross Clamping and Circulatory Arrest

ATAAD has the potential to compromise blood flow to all major organs, including the brain, spinal cord, heart, abdominal viscera, kidneys, and extremities. In addition

to restoring true lumen flow, the objective of the operation should be protection and restoration of function of these end-organs.

Primary repair of the dissection—with adjunctive branch-vessel interventions when needed—is required to restore and maintain perfusion to the heart, abdominal viscera, kidneys, and extremities. Strategies for cerebral protection, however, vary substantially because hemiarch replacement and more extensive arch work require a period of circulatory arrest, necessitating additional measures to preserve brain perfusion and metabolism. While visceral organ protection is solely centered on minimizing metabolic demand, cerebral protection during ATAAD repair also relies on alternative perfusion while antegrade aortic blood flow is halted. One of the key decisions in end-organ protection is the degree of hypothermia. Systemic hypothermia reduces end-organ metabolic rate, lowering the oxygen demand of the brain and other ischemic organs and thereby decreasing the risk of organ injury during the period of circulatory arrest required for ATAAD repair [153]. Cardioplegia delivered into the coronary ostia and via a retrograde coronary sinus catheter should be given at regular intervals determined by the cardioplegia solution and institutional protocol with continuous confirmation of adequate cardiac arrest [154]. Deep hypothermic circulatory arrest (DHCA), defined as core body temperature below 20 °C, began as the standard of care for metabolic protection. However, prolonged deeper hypothermia is associated with increased risk of coagulopathy, neurologic dysfunction, and other risks. Therefore, moderate hypothermic circulatory arrest (MHCA), defined as core body temperature between 20.1–28 °C, is becoming increasingly utilized. Based on IRAD data, before 2010, significantly more patients underwent dissection repair using DHCA than MHCA (57.7% vs 42.3%), whereas between 2010 and 2018, patients underwent dissection repair using MHCA significantly more frequently than DHCA (62.5% vs 37.5%) [155]. Recent studies demonstrated that MHCA provides similar visceral end-organ protection to DHCA, and should be preferred for periods of visceral ischemia less than 60 min, as MHCA may be associated with a reduced inflammatory response and reperfusion injury [156].

ACP and/or retrograde cerebral perfusion (RCP) during circulatory arrest is a popular adjunct used to provide cerebral blood flow while the pump is off. The two main methods for perfusion delivery are ACP and RCP. ACP is defined as perfusion that supplies the brain in a normal physiological direction via direct cannulation of the right axillary artery, innominate artery, or carotid arteries, once circulatory arrest has begun. By maintaining forward arterial inflow, ACP preserves oxygen tension, glucose delivery, and acid-base balance within the cerebral microcirculation, thus helping sustain aerobic metabolism and prevent ischemic cytotoxic shift [153,157]. The physiologic orientation of flow allows ACP to match metabolic demand more effectively than cooling alone, which has enabled the use

of MHCA for complex aortic surgeries, given that cerebral metabolic suppression from MHCA alone would be insufficient [158].

In RCP, cannulation of the superior vena cava allows oxygenated blood to flow superiorly through the cerebral capillaries and through the arterial system, eventually returning centrally via retrograde flow [159]. Although RCP has demonstrated effectiveness in enhancing cerebral protection and in reducing the rate of embolic events by particulate washout, it demonstrates limited capacity to provide meaningful nutrient flow to the brain. This limitation arises from the venous counterpressure and collapse of venous channels under hypothermic circulatory arrest [158]. A single-center ATAAD series found similar 30-day mortality, new neurological dysfunction, and intensive care unit (ICU) or in-hospital stay between ACP and RCP with a standardized protocol for TAR [160].

In the last 15 years, MHCA, used in conjunction with ACP, has proven to be associated with outcomes comparable to DHCA in several studies. A 2021 IRAD analysis of patients with ATAAD who underwent surgical repair in DHCA vs MHCA showed no significant difference in propensity matched stroke, or short-term mortality [155]. A 2023 randomized clinical trial found that lower ranges of MHCA with ACP provided similar protection to DHCA with ACP as evidenced by statistically equivalent rates and severity of global cognitive ability of patients 4 weeks after aortic repair requiring circulatory arrest [161]. A 2016 study found that MHCA and ACP were associated with a 76.5% significantly decreased risk of postoperative neurologic complications and all-cause mortality when compared to DHCA and RCP [162]. A further topic of investigation is whether unilateral or bilateral ACP is superior. Recent literature has suggested that there is no significant difference between the two ACP techniques as the unilateral method is non-inferior to that of bilateral. Therefore, the unilateral ACP method is recommended as it involves less manipulation of arch branch vessels and is simpler [58,163].

Many studies suggest that DHCA alone is safe for up to 40 minutes, MHCA is safe for up to 20 minutes, and ACP prolongs the duration of safety for both temperature approaches [161,164,165,166,167,168]. Due to the heterogeneity of the results in these studies, neuromonitoring methods have been developed to confirm minimal cerebral activity before circulatory arrest. Identifying and correcting cerebral malperfusion by modifying operative technique was associated with improved neurologic outcomes [169,170]. In practice, temperature management and cerebral perfusion in ATAAD repair are complementary tools that must be used mindfully to match the planned surgery to the protection strategy [9,155,161].

17. Postoperative Care

Though the rate of in-hospital mortality for ATAAD has been down-trending from 31% to 22%, likely as a re-

sult of down-trending surgical mortality from 25% to 18%, ATAAD continues to be a devastating diagnosis [6]. As such, effective postoperative management of patients recovering from ATAAD is critical for optimizing outcomes. In the immediate post-operative period, patients recover in the intensive care setting. The intensive care setting offers increased nursing and provider attention, such that the likelihood that common yet devastating complications (stroke, mesenteric ischemia, renal failure, bleeding, paralysis, and acute limb ischemia) are found promptly, with the resources necessary to manage them. Similar to the acute medical management of ATAAD, post-operative management of ATAAD involves anti-impulse therapy and ensuring adequate perfusion [4,32,36,171,172]. Postoperative malperfusion is associated with high in-hospital mortality rates [173]. Early neurologic exam can screen for cerebral and spinal cord malperfusion [34,136,137,138].

Bleeding represents a critical post-operative complication that requires early identification and treatment. ATAAD is associated with coagulopathy for a multitude of reasons. Pre-operatively, blood enters the false aortic lumen, which is not covered by endothelium, resulting in massive secretion of tissue factor that results in extensive coagulation activation and hyperfibrinolysis [174]. In the early post-operative period, hypothermia, hemodilution, and administration of heparin all contribute to the coagulopathy experienced by patients. Temperature management, close activated clotting time monitoring and goal-directed therapy, and judicious blood product utilization help optimize coagulopathy in patients with ATAAD [174]. Prospective randomized data demonstrated that thromboelastography-guided transfusion was associated with a decreased use of allogeneic blood units and reduced incidence of massive transfusion compared with other transfusion strategies [174,175]. Rescue therapy for refractory bleeding following ATAAD repair with factor VIII inhibitor bypassing activity (FEIBA) resulted in reduced transfusion requirement in the first 48 hours after surgery and should be considered as an adjunct to thromboelastography-guided transfusion [176].

18. Long-Term Management and Follow-Up

After surgical ATAAD management, computed tomographic imaging or magnetic resonance imaging of the aorta with transthoracic echocardiography is recommended before discharge, 1, 6, and 12 months post-dissection, and if stable, annually thereafter [1]. Ideally, this imaging would utilize the same modality and be performed at the same institution for optimal comparison [1]. The goal of this imaging surveillance is to detect enlargement and/or aneurysmal degeneration of the distal aorta in a timely fashion, and monitor visceral vessel dissection. Visceral vessel dissection does not necessarily warrant descending aortic intervention at the time of emergent ATAAD repair, but patients should be followed closely to identify distal aortic aneurys-

mal degeneration early [177]. Patients who underwent isolated ascending aortic repair remain at risk for progressive enlargement of unrepaired segments of residual dissected aorta and potential growth of non-dissected native aortic segments because of underlying medial degeneration [177]. Were this to occur, it would likely necessitate repeat intervention on the root, arch, or descending aorta [4]. Patients with residual distal dissections after ATAAD repair, especially those with patent distal false lumen and initial very proximal tear, are at risk for aortic growth and reintervention [178]. Those with chronic residual thoracoabdominal dissection and an aneurysm with total aortic diameter of at least 5.5 cm should undergo elective thoracic aortic repair [4].

Care for patients with ATAAD includes screening for HTAD. Indications for HTAD workup include: (1) thoracic aortic disease (TAD) in patients younger than 60 years old, (2) TAD in patients with syndromic features of Marfan syndrome, Loeys-Dietz syndrome, or vascular Ehlers-Danlos syndrome, (3) TAD with a family history of TAD or peripheral/intracranial aneurysms in a first- or second-degree relative, and (4) TAD with a history of unexplained sudden death at a relatively young age in a first- or second-degree relative [4]. All these patients should receive genetic testing for *FBNI*, *LOX*, *COL3A1*, *TGFBR1*, *TGFBR2*, *SMAD3*, *TGFB2*, *ACTA2*, *MYH11*, *MYLK*, and *PRKG1* to identify pathogenic or likely pathogenic variants, and if any are established, it is recommended to counsel patients about their variant, and recommend cascade genetic testing for their family members [179]. If family members have the pathogenic or likely pathogenic variants from the genetic screening—TTE or CT is recommended to visualize the aorta [4]. Any patients with identified genetic variants or who experienced ATAAD should be followed by a multidisciplinary aortic care center [1,4,7,9,10,180].

19. Outcomes and Prognosis

As previously mentioned, IRAD data report an overall in-hospital mortality of 10–20% for ATAAD, with a 30% in-hospital mortality rate among patients presenting preoperatively with tamponade, shock, heart failure, stroke, coma, MI, acute renal failure, or mesenteric ischemia [5,6]. Cardiac tamponade, often associated with aortic rupture, has been reported in 8–31% of ATAAD cases [181]. Patients with ATAAD with associated MI experience significantly higher mortality rates (33%) compared to those who do not have MI (15.6%) [182]. Among hospital survivors, 10-year survival after ATAAD repair ranges from about 45–70% across studies, reflecting the high burden of comorbidity and progressive aortic disease. Predictors of poorer long-term outcome include renal dysfunction requiring dialysis, cardiogenic shock or malperfusion at presentation, older age, preoperative frailty or impaired mobility, and lower socioeconomic status [183,184,185,186].

After successful repair of ATAAD, distal aortic pathology remains among the most common causes of late mortality [187,188]. This distal aortic pathology (including aneurysmal degeneration of residual arch dissection) can be associated with the need for distal re-intervention. Single-institution prospective data found that re-operative aortic arch reconstruction following a structured algorithm matching the extent of repair with patient pathology resulted in low rates of in-hospital and 30-day mortality (2.8% and 5.6%, respectively) [189]. When these interventions are performed in a controlled setting, operative outcomes are promising—risk of mortality is around 4–6% for both redo root and redo TAR with FET following ATAAD repair [190,191]. This demonstrates the importance of close follow-up with a multidisciplinary team specializing in the care of patients with aortic disease [192].

20. Conclusion

Despite decades of advances in the diagnosis and management of ATAAD, it continues to be associated with significant morbidity and mortality. This review underscores several key themes that improve care of this complex pathology. Optimizing time from presentation to surgery at a high-volume center, individualizing treatment based on malperfusion patterns and surgeon expertise, and employing targeted revascularization strategies in select cases are key to improving outcomes. An algorithmic approach to evaluating ATAAD allows surgeons to individualize the management technique (including cannulation strategy, extent of resection and reconstruction, and end-organ protection techniques) to patient pathology and surgeon expertise. A brief operative video demonstrating the execution of our operative management of ATAAD algorithm (Fig. 3) is shown in **Supplementary Video 1**. There are several promising innovations in development including novel hybrid prostheses and endovascular approaches for select high-risk patients, that have the potential to further improve outcomes. It is essential that we continue to develop and improve tools for managing patients with this complex aortic pathology at aortic centers.

Author Contributions

AHK, MAC, BT, IK, and MD contributed to the writing of the original draft of this manuscript. All authors contributed to the writing, reviewing, and editing of this manuscript. OAJ, SSP, and DRB contributed to the supervision of this manuscript. All authors contributed to the conceptualization of this manuscript. All authors have read and approved the final version of the manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Supplementary Material

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