

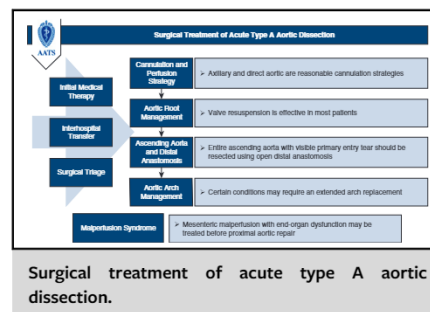
2021 The American Association for Thoracic Surgery expert consensus document: Surgical treatment of acute type A aortic dissection



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Acute aortic dissection (AD) involves the ascending aorta in approximately two-thirds of patients.^{1,2} Chest pain is the most common feature, but clinical presentation can be varied, and algorithms have been developed to facilitate timely diagnosis.^{3,4} Nevertheless, delays in diagnosis comes at a severe cost to the patient.⁵ The complications of AD that involve the ascending aorta have been known for well over 60 years and include aortic rupture, cardiac tamponade, aortic regurgitation (AR), and organ malperfusion.^{6,7} The risk of death is estimated to be 1% to 2% per hour and nonoperative treatment is associated with mortality in nearly 60% of patients.^{8,9} Open surgical repair of the ascending aorta is a life-saving operation and remains the standard of care for patients with acute type A AD (ATAAD). The surgical care of these patients continues to evolve through innovation, guided by clinical evidence with long-term follow-up.

Our goal was to develop an expert consensus document (ECD) for the surgical treatment of ATAAD. The intent of



CENTRAL MESSAGE

The management of ATAAD continues to evolve but emergency cardiac surgery remains the standard of care.

PERSPECTIVE

Acute, type A aortic dissection remains a challenging emergency in cardiovascular surgery. The management of life-threatening complications has evolved to include endovascular options and complex aortic repair, but expeditious open replacement of the ascending aorta remains the preferred approach. This expert consensus offers recommendations based on current clinical evidence and expert opinion.

this ECD is to improve quality of care through evidence-based recommendations for a true surgical emergency. These recommendations, on the basis of systematic methods to evaluate and classify evidence, should serve surgeons, emergency room physicians, and any health care provider who care for patients with ATAAD.



Scanning this QR code will take you to the table of contents to access supplementary information.



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Abbreviations and Acronyms

| | |
|-------|---|
| AATS | = The American Association for Thoracic Surgery |
| ACP | = antegrade cerebral perfusion |
| AD | = aortic dissection |
| AR | = aortic regurgitation |
| ATAAD | = acute type A aortic dissection |
| CAC | = comprehensive aortic center |
| CPB | = cardiopulmonary bypass |
| CT | = computed tomography |
| ECD | = expert consensus document |
| FET | = frozen elephant trunk |
| HTAD | = hereditary thoracic aortic disorder |
| IMH | = intramural hematoma |
| IRAD | = International Registry for Aortic Dissection |
| MRI | = magnetic resonance imaging |
| RCP | = retrograde cerebral perfusion |
| SCI | = spinal cord injury |
| STS | = Society of Thoracic Surgeons |
| TEVAR | = thoracic endovascular aortic repair |
| TTE | = transthoracic echocardiography |
| VSARR | = valve-sparing aortic root replacement |

Definitions and Scope

- AD is a disruption of the tunica intima that results in blood flow into the tunica media that can propagate throughout the entire length of the aorta and into branch vessels. AD is part of a spectrum of acute aortic syndromes including penetrating aortic ulcer and intramural hematoma (IMH). These syndromes are life-threatening, inter-related conditions with similar clinical characteristics.
- Type A AD involves the ascending aorta (proximal to brachiocephalic artery), regardless of primary entry tear location according to the Stanford classification and are also considered type I/type II AD depending on the distal extent according to the DeBakey classification.^{7,10,11} Newer classifications endorsed by surgical societies have characterized AD according to site of primary entry tear, extent of false lumen, and malperfusion.^{12,13} Standardization of these classifications has not been uniform, and definitions have been conflicting. For example, an AD with the primary entry tear in the proximal descending thoracic aorta with proximal extension of the false lumen into the ascending aorta (retrograde type A AD according to the Stanford definition) would be classified as a type B AD according to the Society of Thoracic Surgeons (STS)/Society for Vascular Surgery definitions and a type A AD according to the European Association for Cardio-Thoracic Surgery/European Society of Cardiology definitions.^{12,14}
- ATAAD is a dissection that is diagnosed <14 days from the onset of symptoms and is standard in all classification schemes.^{4,12,15} For the purposes of this document, any AD that involves the ascending aorta (proximal to the brachiocephalic artery) and is diagnosed within 14 days of symptom onset are considered ATAAD regardless of the location of the primary entry tear. Subacute and chronic AD are beyond the scope of this document.
- The scope of this document is the management and treatment of ATAAD. A detailed review of the evaluation and diagnosis was not performed.
- Although surgical management is the main focus of this document, concise perioperative medical management and postoperative follow-up are included.
- The writing group acknowledges and endorses the evidence-based approaches to ATAAD management including the 2010 American College of Cardiology Foundation/American Heart Association/The American Association for Thoracic Surgery (AATS)/American College of Radiology/American Stroke Association/Society of Cardiovascular Anesthesiologists/Society for Cardiovascular Angiography and Interventions/Society of Interventional Radiology/STS/Society for Vascular Medicine guidelines for the diagnosis and management of patients with thoracic aortic disease, 2011 Japanese Circulation Society guidelines for diagnosis and treatment of aortic aneurysm and aortic dissection, 2014 European Society of Cardiology guidelines on the diagnosis and treatment of aortic diseases, and the 2016 Canadian Cardiovascular Society/Canadian Society of Cardiac Surgeons/Canadian Society for Vascular Surgery joint position statement on open and endovascular surgery for thoracic aortic disease.^{3,4,16,17}
- Patient preferences and values should be combined with evidence-based, clinical judgment in the management of ATAAD.
- When new data become available, the recommendations in this document are subject to revisions.

METHODOLOGY

We followed the recommendations of the AATS/STS position statement on developing clinical practice documents.¹ An expert panel consisting of 15 cardiac surgeons, all members of AATS, followed an ECD pathway to develop consensus statements according to class of recommendation and the level of evidence. Class of recommendation specifies the strength of recommendation, including the estimated magnitude and certainty of benefit compared with risk, and level of evidence is used to assesses the quality of scientific evidence supporting the recommendation, graded on the basis of the type, quantity, and consistency of the data from clinical trials and other sources.²

Literature searches were conducted by a scholarly communications librarian using Ovid MEDLINE, and all articles published since 2000

were reviewed (Appendix E1). Important, older landmark publications were also included. After these initial searches, section-specific literature was reviewed by the writing committee and recommendations were created on the basis of discussion, overseen by an epidemiologist/methodologist. Recommendations were submitted for voting among writing committee members. To approve recommendations, a 75% consensus rate was required with 80% participation to ensure validity of voting. This process was repeated for each section. Controversies were discussed and resolved via regular, online video meetings and re-voting, in accordance with the Delphi method process.^{3,4} Each author contributed substantially to the writing of 8 document sections and the document was finalized by the writing committee chairs. The writing committee and a group of 10 external reviewers were given an opportunity to review, comment, and approve the document before it was submitted for final approval by AATS Cardiac Clinical Practice Standards Committee and AATS Board of Directors.

SECTION 1: INITIAL MEDICAL THERAPY

Initial medical therapy (Table 1) should optimize patient stabilization during interhospital transfer and surgical triage. As soon as a diagnosis of ATAAD is made, treatment of blood pressure, pulse rate, and pain should be initiated. Medical therapy should be directed at decreasing excessive shear stress on the dissected layers of the affected aorta, thereby reducing false lumen propagation. Additionally, patients with ATAAD can present with various complications, such as severe AR, hypotension, and cardiac tamponade. Hence, the choice of medical treatment should be individualized to the presenting clinical condition to minimize exacerbation of ATAAD complications. Medical treatment is not a substitute for surgery, but used appropriately, can be crucial in achieving initial stabilization.

Focused Clinical Questions

What are appropriate agents for anti-impulse therapy?

Goals of initial medical therapy in normotensive or

hypertensive patients should be focused on decreasing shear stress on the aortic wall, suppression of false lumen propagation, and ultimately prevention of rupture. Forces acutely propagating the dissection process include the heart rate, blood pressure, and velocity of ventricular contraction. These parameters might be stabilized through early β -blockade to systolic blood pressure targets of 100 to 120 mm Hg and heart rate below 60 bpm.¹¹⁻¹³ Through diminished afterload and decreased rate of oxygen consumption, anti-impulse therapy also provides improvement in myocardial perfusion. Exemplary agents are intravenous propranolol, esmolol, labetalol, or metoprolol. Esmolol has a uniquely short half-life, and labetalol, a β -blocker with intrinsic partial sympathomimetic activity, gives a potent advantage of achieving heart rate and blood pressure control, potentially reducing the need for additional agents. Some of the contraindications to β -blockers are asthma, chronic obstructive pulmonary disease, or atrioventricular block. Potential alternatives in patients with contraindications or in those not able to tolerate β -blockers are nondihydropyridine calcium channel blockers—verapamil or diltiazem. Both drugs should be used with caution in a setting of severe AR because they might cause reflex tachycardia.

What are appropriate second-line agents? Additional agents might be required when hypertension is refractory to one agent. Vasodilators should be used with caution, ensuring chronotropic control with β -blockers is first established, to avoid reflex tachycardia.³ Intravenous nicardipine (dihydropyridine calcium channel blocker) has been shown to rapidly decrease blood pressure, without reflex tachycardia or exaggerated depression of diastolic pressure.⁴ Intravenous nitroprusside has also been effectively used in

TABLE 1. Initial medical therapy recommendations

| Recommendations | COR | LOE | References |
|---|-----|-----|------------|
| Initial Medical Therapy | | | |
| 1. β -blockers are recommended in the initial management of ATAAD without severe aortic regurgitation. | I | B | 1-3 |
| 2. Calcium channel blockers are a potential alternative. | IIa | B | 1,3,4 |
| 3. When multiple agents are required, it is reasonable to start vasodilators after initial rate control. | IIa | C | 1,3 |
| 4. In the setting of hypotension, volume resuscitation is reasonable to achieve systolic blood pressure of 90 mmHg. | IIa | C | 5-7 |
| 5. Pain relief is recommended in patients with ATAAD. | I | C | 8-10 |

COR, Class of recommendation; LOE, level of evidence; ATAAD, acute type A aortic dissection.

a setting of acute AD, however, cyanide toxicity during prolonged administration is a serious adverse effect. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are also reasonable options, but more difficult to titrate.

How should hypotension in patients with ATAAD be managed? Hypotension in ATAAD patients is a critical condition, associated with increased in-hospital mortality and neurologic events.^{6,7} Underlying causes include acute AR, cardiac tamponade, and myocardial infarction. Each of these conditions require definitive operative management. The most reasonable first approach is to administer intravenous fluids to improve blood pressure, increase preload and cardiac output, and ensure adequate end-organ perfusion. Permissive hypotension is reasonable with a target systolic pressure of 90 mm Hg, because mortality increases below that value.^{5,6} Vasopressors might be an adjunct to volume therapy but can contribute to further propagation of false lumen. Pericardiocentesis remains controversial in dissection-related hemopericardium and should not delay surgery. However, in patients with imminent cardiac arrest, hemopericardium may be treated with pericardiocentesis to restore sufficient end-organ perfusion.¹⁴

How to manage pain in patients with ATAAD? Patients with ATAAD might present with acute pain and anxiety.^{8,9} Pain affects the sympathetic system, increasing blood pressure and heart rate through the release of catecholamines, which directly undermines anti-impulse therapy. Analgesia augments the effects of β -blockers and vasodilators, and opioids provide not only beneficial effects on anti-impulse therapy, but also decrease agitation and respiratory distress. Intravenous opioids should be started early after the diagnosis and rapidly titrated for pain relief.

SECTION 2: INTERHOSPITAL TRANSFER

Current data suggest that ATAAD patients might benefit from transfer to a comprehensive aortic center (CAC). The metrics to define such centers is beyond the scope of this

document but might include case volume and expertise in emergency and elective aortic cases. Surgery for ATAAD is an uncommon occurrence in most hospitals with a median case volume of 3 per year.¹ Moreover, only approximately 10% of hospitals perform more than 10 ATAAD repairs annually.^{2,3} The effect of case volumes has consistently shown that higher-volume centers achieve better patient survival after ATAAD repair compared with lower-volume centers.⁴

Outcomes for patients with ATAAD might be further improved when patients are managed at centers that perform a high volume of elective aortic cases.^{5,6} CACs have committed significant resources to develop an advanced and innovative infrastructure as well as multidisciplinary expertise that can offer an array of options to these very complicated patients.⁷⁻¹¹ Efficiency in diagnosis and triage and improved quality metrics are additional benefits.^{10,11} Therefore, the regionalization of care to CACs has merit, but should be balanced with the need for appropriate access to timely care with optimal patient outcomes (Table 2).

Focused Clinical Questions

When should patients with ATAAD be transferred to a CAC? When patients with ATAAD present to hospitals without on-site cardiac surgery or when cardiac surgery is not immediately available, interhospital transfer should be considered. Decision-making for transfer is simplified when the next closest hospital is also a CAC. An increasing body of evidence supports the relationship between greater hospital^{2,3,13-15} and surgeon^{2,16,17} experience in treating ATAAD and a decline in operative mortality and morbidity. Recent reports from large data sets show operative mortality after repair of ATAAD as between 17% and 20% with individual high-volume aortic centers reporting rates consistently below 10%.^{1,18,19} A study of more than 5000 patients with ATAAD from the National Inpatient Sample database divided annual hospital volume with ATAAD into quartiles: lowest (<3), low (>3-8), high (>8-13), and

TABLE 2. Interhospital transfer recommendations

| Recommendations | COR | LOE | References |
|--|-----|-----|------------|
| Interhospital Transfer | | | |
| 1. When cardiac surgery is not immediately available, it is reasonable to transfer patients with ATAAD to a Comprehensive Aortic Center. | IIa | B | 3,6,10,12 |
| 2. It may be reasonable to transfer patients with complicated ATAAD to a Comprehensive Aortic Center. | IIb | C | 7,10,11 |

COR, Class of recommendation; LOE, level of evidence; ATAAD, acute type A aortic dissection.

highest (>13) and showed a significant reduction in operative mortality when low-volume hospitals were compared with high-volume hospitals (27% vs 16%; $P < .001$).² Dramatic reductions in operative mortality (33.9% to 2.8%) were reported when a focused thoracic aortic surgery program not only increased institutional ATAAD volume but also increased total proximal aortic procedure volume, standardized perioperative care, and reduced the number of participating surgeons, thus tripling the experience individual surgeons obtained in performing these procedures.⁹

Bypassing a low-volume hospital for the benefits of a CAC that might be further away must be balanced with the risk of death during transfer and consequences of delayed treatment. A recent study of 16,886 Medicare beneficiaries with ATAAD between 1999 and 2014 showed that 40% of patients were transferred to another hospital.⁶ Approximately half of the transferred patients went to a high-volume hospital, defined by the highest decile of open thoracic aortic (elective and emergency) annual volume (>105 cases) and the other half to a low-volume center that comprised the comparator group. The median additional distance between high- and low-volume hospitals was 50 miles. Despite a delay in transfer because of increased travel distance, surgery at a high-volume hospital conferred a 7.2% (95% confidence interval, 4.1%-10.3%) absolute risk reduction in operative mortality compared with a low-volume hospital, and this benefit persisted on long-term follow-up. Although death during transfer was unknown, complex sensitivity analysis determined that 4.4% of patients would need to die during the transfer process to negate the benefits of transfer to high-volume hospitals.

When should patients with complicated ATAAD be transferred to a CAC? Regional care models linking rural and community hospitals to high-volume tertiary level facilities with expertise across a spectrum of aortic disease lend further support to the safety of transferring stable patients with ATAAD. A regional system based in Ohio reported their results with 359 patients transferred from 84 different hospitals over a 3-year period.⁷ Because of a median transfer distance of 66 km, there were no transfer-related mortalities. Another regional system based in Minnesota implemented a multidisciplinary program with 101 patients across 32 hospitals over 7 years in the US Midwest. Despite regionalization of cases, a 43% reduction in the time to ATAAD diagnosis (279 minutes to 160 minutes; $P = .014$) and a 55% reduction in the time from presentation to surgical intervention was observed.¹⁰ Moreover, the percentage of patients who received proper anti-impulse therapy also increased dramatically as did the percentage of hospital survivors who participated in long-term follow-up after the index event.

Patients with ATAAD complicated by shock secondary to cardiac tamponade, acute AR, or acute myocardial infarction might be too unstable to tolerate interhospital transfer. However, ATAAD complicated by comorbidities, previous cardiac surgery, or distal malperfusion are candidates for escalation of care to a CAC. Options including aortic root replacement and aortic arch reconstruction are discussed in the following sections, but some surgical and endovascular therapies require an additional level of expertise that might warrant transfer to a CAC.

SECTION 3: SURGICAL TRIAGE

Surgical triage is on the basis of clinical findings and cross-sectional imaging: computed tomographic angiography, magnetic resonance angiography, or transesophageal echocardiography. The imaging must be scrutinized for (1) the diagnosis of AD or IMH, (2) the location of the primary entry tear, (3) the proximal and distal extent of false lumen, and (4) radiologic signs of malperfusion or rupture. Clinical findings are correlated with imaging features to diagnose complications of ATAAD, including pericardial tamponade, AR, malperfusion syndrome, or shock.

Surgery remains the standard of care for ATAAD on the basis of accumulated worldwide data that surgical management significantly outperforms medical management.¹⁻⁴ The stark contrast between the 60% 30-day mortality of medical management as reported by Masuda and colleagues⁵ and the 26% 30-day mortality of surgical management reported in the same era by Fann and colleagues⁶ continues to be true today, and emergency surgery is recommended for most patients with ATAAD without delay. However, recent data suggest some notable exceptions, including IMH, catheter-induced ATAAD, retrograde ATAAD, and select cases of malperfusion (discussed in Section 8). These circumstances might warrant delayed intervention or expectant management (Table 3).

Focused Clinical Questions

How should IMH be managed? Acute type A IMH can regress spontaneously with medical management alone.⁷ However, medical management of IMH is associated with a 37% rate of complications and 29% to 46% risk of conversion to surgery.^{7,11} Two meta-analyses have shown that overall survival is better with immediate surgery than expectant management.^{7,8} A few single-institution studies have identified risk factors for failure of expectant management that include total aortic diameter >50 mm, hematoma thickness >11 mm, pericardial effusion, AR, and ulcer-like projections.^{7-9,11-13} In the absence of these features, it might be reasonable to pursue expectant management of acute type IMH in

TABLE 3. Surgical triage recommendations

| Recommendations | COR | LOE | References |
|---|-----|-----|------------|
| Surgical Triage | | | |
| 1. Emergency surgery is recommended for patients with acute type A aortic dissection. | I | B | 1-4 |
| 2. Surgery is recommended in patients with type A IMH and one or more high-risk features*. | I | C | 7,8 |
| 3. Expectant management may be reasonable for type A IMH patients with significant comorbidities in the absence of high-risk features*. | IIb | C | 9-13 |
| 4. Surgery can be effective in patients with ATAAD and brain malperfusion. | IIa | B | 14-20 |
| 5. Nonoperative management is reasonable in catheter induced ATAAD, if limited to the aortic root. | IIa | B | 21-23 |
| 6. Descending TEVAR alone may be reasonable in selected patients with retrograde ATAAD. | IIb | C | 24,25 |

COR, Class of recommendation; LOE, level of evidence; ATAAD, acute type A aortic dissection; IMH, intramural hematoma; TEVAR, thoracic endovascular aortic repair. *Aortic diameter >50 mm, hematoma thickness >11 mm, pericardial effusion, aortic regurgitation, ulcer-like projection.

patients who have significant comorbidities with close inpatient monitoring and serial imaging.^{7-11,13}

How should ATAAD with brain malperfusion be managed? The International Registry for Aortic Dissection (IRAD) registry has suggested that the incidence of brain malperfusion in ATAAD is 15%.²⁶ Patients with ATAAD and brain malperfusion can present with asymptomatic, radiographic findings to profound coma.^{15,19,27-30}

The potential for conversion of ischemic to hemorrhagic stroke must be recognized, however, endovascular neurointervention has become the standard of care for acute ischemic stroke in quaternary care centers in response to several randomized clinical trials.³¹⁻³⁴ No such data exist for patients with ATAAD and stroke; however, evidence continues to favor immediate ATAAD surgery, and extrapolation of the acute ischemic stroke literature is reasonable.¹⁴⁻¹⁸ Surgery can reduce acute mortality in selected ATAAD patients with stroke commensurate to ATAAD without stroke.^{14-18,20,35} Moreover, reversal of deficits, including coma, after immediate surgery has been shown in several reports.^{19,27,36,37} Nevertheless, ATAAD with brain malperfusion has been associated with a 37% to 50% incidence of persistent neurological deficit despite proximal aortic repair.^{15,18} A recent study from Michigan suggested the presence of internal carotid occlusion was associated with cerebral edema and herniation in all patients regardless of management strategy.³⁸ Extent of repair (including carotid artery replacement), as well as initial carotid reperfusion have

been proposed as strategies to improve outcomes.^{39,40} Until more studies become available to inform patient selection and treatment options, proximal aortic repair is reasonable in most patients with brain malperfusion.

How should iatrogenic ATAAD be managed? Limited published data on catheter-induced ATAAD suggests that medical management is reasonable for dissections that are limited to the aortic root in the area immediately surrounding the coronary ostium with careful observation and blood pressure control, unless myocardial ischemia occurs.²¹⁻²³

These localized catheter-induced lesions often heal or stabilize spontaneously after termination of a percutaneous coronary intervention. The success of medical management is largely predicated on very limited tears that are incited in a retrograde fashion, an orientation that might naturally facilitate restored apposition of the torn wall. If a catheter-induced ATAAD extends beyond the aortic root, immediate surgical intervention after confirmation of the diagnosis is warranted.

What is the preferred management of retrograde ATAAD? De novo, retrograde ATAAD occurs when the primary entry tear is distal to the left subclavian artery and the false lumen propagates retrograde into the ascending aorta. Retrograde ATAAD has been successfully treated with endovascular coverage of the primary entry tear in the descending aorta with a covered stent graft in carefully selected patients in a few small series.^{24,25} Anatomy favorable for thoracic endovascular aortic repair (TEVAR) is determined using cross-sectional imaging and includes

an adequate proximal landing zone for the stent graft, often necessitating coverage and revascularization of the left subclavian artery.

Iatrogenic, retrograde ATAAD as a consequence of TEVAR performed for either descending thoracic aortic aneurysm or dissection is often due to injury by the stent graft to the intimal surface of the lesser curve of the aortic arch.⁴¹⁻⁴³ Poor apposition of the proximal aspect of the stent (bird's beaking), hereditary thoracic aortic disorder (HTAD), and ascending aortic dilatation >4 cm might be important risk factors.^{41,42,44-46} Iatrogenic, retrograde ATAAD should be treated by immediate proximal aortic repair unless patient anatomy allows for proximal extension of the TEVAR into the aortic arch.

Can ATAAD after previous coronary artery bypass grafting be delayed for coronary imaging? ATAAD patients with a history of previous coronary artery bypass grafting might benefit from additional preoperative coronary imaging including coronary angiography or computed tomography (CT) coronary angiography.^{47,48} Previous studies have suggested that pericardial adhesion from previous cardiac surgery might prevent aortic rupture and pericardial tamponade,⁴⁹ but recent studies have shown that patients remain at significant risk for all complications of ATAAD.⁴⁷ Moreover, the diagnostic CT angiography for ATAAD might also show patency of bypass grafts in patients with previous coronary artery bypass grafting, obviating the need for coronary angiography.

SECTION 4: CANNULATION AND PERFUSION STRATEGY

The complexity of circulatory management and cardiopulmonary bypass (CPB) during ATAAD repair depends

on the extent of aortic reconstruction. Guiding principles for cannulation and perfusion strategy are the establishment of antegrade systemic perfusion whenever possible and the alleviation of malperfusion and end organ ischemia by restoration of true lumen flow (Table 4). Organ protection during periods of aortic cross-clamping and circulatory arrest can be achieved with systemic temperature management, cardioplegia, and brain perfusion strategies.

Blood flow can be affected by the choice of arterial cannulation site for CPB and lead to a dynamic circulation pattern within the lumens of the AD, sometimes requiring adaptation to different sites during the course of the operation.¹ Cannulation for CPB also has implications on how cerebral perfusion can be provided during circulatory arrest required during arch reconstruction for ATAAD repair. Furthermore, with refinement in brain perfusion and protection strategies, there has been an evolution of the degree of hypothermia when concomitant antegrade cerebral perfusion is used.

Focused Clinical Questions

What is the preferred cannulation site for ATAAD surgery? Right axillary artery cannulation is more commonly performed during ATAAD repair with over 50% usage in Europe and 36% in North America.¹⁸⁻²⁰ To date, there is no randomized study on the outcomes of the different sites of arterial cannulation. Several retrospective studies have shown decreased risk of stroke and mortality with axillary cannulation compared with other techniques of cannulation. In an analysis of 775 patients, Rosinski and colleagues reported that axillary cannulation was associated with a decreased risk of mortality.¹ In a recent analysis of the STS database of 7353 patients, axillary

TABLE 4. Cannulation and perfusion strategy recommendations

| Recommendations | COR | LOE | References |
|---|-----|-----|------------|
| Cannulation and Perfusion Strategy | | | |
| 1. Right axillary artery cannulation is reasonable for stable patients undergoing ATAAD repair. | IIa | B | 1-5 |
| 2. Direct aortic cannulation with imaging guidance is reasonable for ATAAD repair. | IIa | B | 6-9 |
| 3. Femoral artery cannulation may be reasonable for ATAAD repair. | IIb | B | 8,10 |
| 4. The addition of either ACP or RCP during circulatory arrest is reasonable for ATAAD repair. | IIa | B | 11-13 |
| 5. Circulatory arrest with ACP and moderate or deep hypothermia is reasonable for ATAAD repair during extended arch reconstruction. | IIa | C | 14-17 |

COR, Class of recommendation; LOE, level of evidence; ATAAD, acute type A aortic dissection; ACP, antegrade cerebral perfusion; RCP, retrograde cerebral perfusion.

cannulation was associated with the lowest risk of stroke.² In another analysis by Benedetto and colleagues, a meta-analysis of 8 studies including 793 patients, axillary cannulation was associated with a reduced risk of in-hospital mortality and permanent neurologic deficit compared with femoral artery cannulation.³ However, technical considerations remain and the right axillary artery should be reconsidered if dissected.

Increasing experience has been recently reported with direct ascending aortic cannulation.¹⁹ This technique provides physiologic antegrade perfusion by central cannulation of the ascending aorta. Nonetheless, cannulation of a dissected ascending can be challenging and is performed using the Seldinger technique to access the true lumen.²¹ Most importantly, imaging (epiaortic ultrasonography or transesophageal echocardiography) is required to confirm cannulation of true lumen.^{8,9} In a study of 584 patients, Kreibich and colleagues reported that central aortic cannulation is safe and associated with similar outcomes with regard to stroke and mortality compared with axillary and femoral artery cannulation.⁸ In a series of 309 patients, Jormalainen and colleagues reported central aortic cannulation was also associated with a similar outcome of stroke and mortality compared with peripheral cannulation.⁷

Classically, femoral artery cannulation has been widely used in the past and remains relevant today.^{18,19} The concern regarding femoral artery cannulation is the unpredictable nature of retrograde arterial flow in the dissected aorta resulting in potential worsening of organ malperfusion, especially the brain.^{1,2,6} In an analysis of cannulation strategies in 25,462 patients, Helder and colleagues reported femoral artery cannulation was associated with a significantly greater risk of stroke compared with axillary or central aortic cannulation.⁶ In the previously cited STS Adult Cardiac Surgery Database study by Ghoreishi and colleagues, femoral artery cannulation was associated with the highest risk of stroke.² However, with the recent increasing trend in axillary and central aortic cannulation in stable patients, there might be an unfavorable selection bias for femoral cannulation. Femoral arterial cannulation remains a rapid and effective technique for arterial cannulation during ATAAD repair, especially in hemodynamically unstable patients.

Should adjunctive brain perfusion be used during circulatory arrest in the ATAAD repair? Over the past 2 decades, brain protection strategy has evolved from the use of isolated deep hypothermic circulatory arrest in initial early experience to currently include retrograde cerebral perfusion (RCP) or antegrade cerebral perfusion (selective unilateral vs bilateral). With regard to comparison of the 2 perfusion strategies in elective arch surgery, there has been no evidence to suggest superiority of either perfusion strategy.^{22,23} To date, there has been only 1 randomized study to examine outcome differences between antegrade cerebral perfusion (ACP)

and RCP in arch reconstruction during elective surgery, and it showed no difference in stroke, or magnetic resonance imaging (MRI) or neurocognitive studies after total aortic arch replacement.²⁴ Nonetheless, the trend in Europe and North America has been an increasing use of ACP with varying degrees of hypothermia.^{11,18,25}

Multiple retrospective analyses have been performed to examine whether the different brain protection strategies have a significant effect on outcomes in ATAAD repair. In the largest series to date, O'Hara and colleagues examined the STS Database in an analysis of 6387 patients who underwent ATAAD repair.¹¹ Brain perfusion during ATAAD repair with either ACP or RCP was associated with reduced risk of death and stroke compared with hypothermic circulatory arrest without cerebral perfusion.

How should systemic temperature be managed during circulatory arrest during ATAAD repair? When extended arch reconstruction requiring longer duration of circulatory arrest is anticipated, the use of ACP has been advocated by many.^{5,26-29} With regard to the degree of hypothermia during ATAAD repair,³⁰ there is evidence to suggest that warmer temperature (moderate hypothermia) is safe and similar to colder temperatures (deep hypothermia) when used with antegrade cerebral perfusion.^{14-17,25,29} There has been no evidence to support that either unilateral or bilateral brain perfusion has superiority in terms of brain protection and outcomes.^{5,26,29,31-33}

SECTION 5: AORTIC ROOT MANAGEMENT

Management of the aortic root in patients with ATAAD is on the basis of the patient's clinical condition and anatomy defined using cross-sectional imaging. Guiding principles for management of the aortic root are restoration of a well functioning aortic valve, obliteration of the false lumen, and alleviation of coronary malperfusion (Table 5). Detailed examination of the structure and function of the root can direct therapy with assessment of the severity and mechanism of the AR, the extent of the root dissection, and the pattern of coronary artery involvement. Surgical experience should also be considered in the final choice of aortic root preservation or replacement. The need to maximize long-term benefit should be balanced with the primary goal of limiting early operative risk.

Focused Clinical Questions

What is the preferred management of the aortic root and for the aortic valve in the ATAAD? For most patients, a sinus segment-preserving approach to aortic root repair with resuspension of the aortic valve and obliteration of the false lumen within the aortic root is appropriate. AR in patients with ATAAD is often caused by a dissection flap extending below the sinotubular junction, causing detachment and prolapse of one or more of the aortic valve

commissures (Figure 1). All 3 commissures should be re-suspended using transmural, pledgeted sutures to reattach the commissures to the aortic wall (Figure 2). The dissection flap often extends into the noncoronary and part of the right coronary sinuses, requiring obliteration of the false lumen within the sinus segment. This may be accomplished by using one or a combination of the following techniques: creation of a neomedia with felt, pericardium, graft, or other material (Figure 2); judicious use of a surgical adhesive; or direct suture repair with a fine monofilament suture.¹⁵⁻¹⁷ Surgical adhesives should be used sparingly to avoid tissue necrosis and late pseudoaneurysms.^{18,19} Furthermore, neurotoxic effects on the conduction system can result in heart block.^{18,19} If the aortic sinuses are minimally dissected and not enlarged, but the aortic valve cusps are diseased, then the aortic valve should be replaced along with a supracoronary ascending aortic graft.

Aortic root replacement is recommended in the presence of a root aneurysm or intimal tear within the root. If the tear is in the noncoronary sinus, replacement of a single sinus might be considered.²⁰ Root replacement is reasonable for patients suspected to have Marfan syndrome or other HTAD. In patients with extensive dissection in all 3 sinuses, aortic root replacement might be unavoidable because of the quality of the tissues. If the aortic valve cusps themselves are calcified or diseased, then the root should be replaced with a composite biological or mechanical valve graft, depending on the patient’s age and other clinical considerations. If the cusps are of good quality, then a valve-sparing aortic root replacement (VSARR) with either a reimplantation or remodeling technique may be considered in experienced centers.

Several retrospective studies have compared the early and late outcomes after root replacement versus limited root repair (valve resuspension with various techniques of false lumen obliteration) for ATAAD.^{1,3-9,14} Root replacement was necessary in approximately 30% to 35% of cases. Indications for root replacement were similar across most studies and included an intimal tear in the root, root diameter >45 mm, HTAD, and unreparable valve pathology. Although several studies have shown equivalent early and late outcomes of the 2 approaches,^{1,3,4,14} others have reported that a root-preserving approach is associated with an increased risk of late AR, root dilatation, and proximal aortic reoperation.^{2,5,6,8} Nevertheless, for most patients, valve resuspension with obliteration of the proximal false lumen within the root and a supracoronary graft replacement of the ascending aorta provides satisfactory results. Of note, in the setting of normal cusps and no root aneurysm, valve resuspension and tailoring of the sinotubular junction can treat even severe AR caused by the dissection.

What is the preferred management of the aortic root in patients with ATAAD and HTAD? Root replacement with a composite valve graft might be required in patients with extensive root pathology as described previously. For patients with Marfan syndrome and those with HTAD, aortic root replacement might be particularly beneficial because supracoronary ascending aortic graft replacement is associated with a high (>40%) need for subsequent reintervention.¹⁰ In single-center series, excellent early and late outcomes have been reported after VSARR with either a reimplantation or remodeling technique in ATAAD patients.^{11,13,14} In a recent systematic review and meta-

TABLE 5. Management of aortic root recommendations

| Recommendations | COR | LOE | References |
|--|-----|-----|------------|
| <i>Aortic Root Management</i> | | | |
| 1. Aortic valve resuspension is recommended for most patients with ATAAD. | I | B | 1-4 |
| 2. Aortic root replacement is recommended in ATAAD with root aneurysm or primary entry tear in the root. | I | B | 3-9 |
| 3. Aortic root replacement is reasonable in patients with ATAAD and Marfan syndrome or other hereditary thoracic aortic disorders. | IIa | C | 10 |
| 4. Valve-sparing aortic root replacement may be reasonable in selected patients with ATAAD. | IIb | C | 11-14 |
| 5. Expedient coronary artery bypass grafting should be performed in patients with ATAAD and persistent coronary malperfusion after repair. | I | C | - |

COR, Class of recommendation; LOE, level of evidence; ATAAD, acute type A aortic dissection.

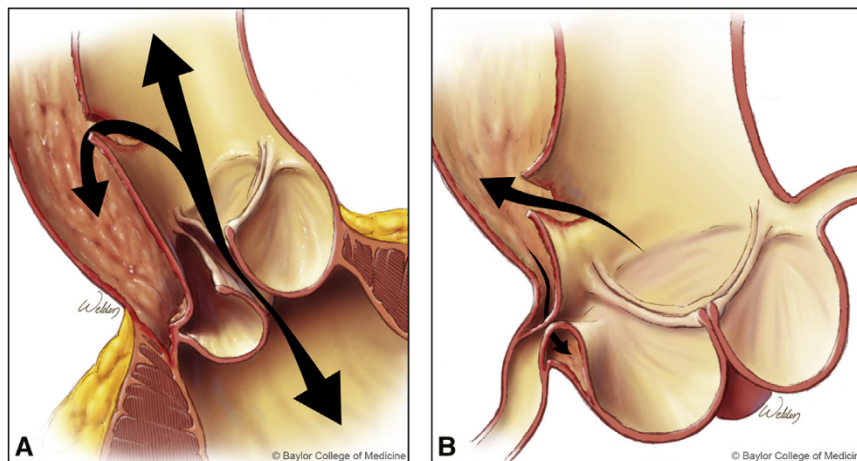


FIGURE 1. Disruption of the aortic root by acute type A dissection. Illustration of potential anatomic consequences of acute type A aortic dissection. A, The dissection flap extends below the sinotubular junction, causing detachment and prolapse of the valve commissure, resulting in aortic regurgitation. B, Malperfusion of the coronary artery from pressurization of the false lumen.

analysis outcomes of VSARR with reimplantation of the aortic valve was compared with composite valve graft procedures in patients with ATAAD; improved survival and less aortic valve reintervention were reported after VSARR in carefully selected patients.¹² Because of the additional operative complexity and myocardial ischemia time, however, this approach should be undertaken only by experienced surgeons. Despite a growing body of literature, these reports remain highly selective with relatively small sample sizes, and typically only young and otherwise healthy patients who stand to gain the greatest benefit from valve preservation are offered VSARR in the emergency setting.

How should bicuspid aortic valve be managed in a setting of ATAAD? Patients with a normally functioning bicuspid aortic valve or with AR caused by the dissection itself and without extensive root pathology might be amenable to valve resuspension and limited root repair techniques. Retrospective series have reported that patients with ATAAD and bicuspid aortic valve present at a younger age and undergo root replacement more often than those with tricuspid valves.²¹⁻²³ Early and late results after ATAAD repair are not affected by valve morphology.

What is the preferred management of the ATAAD complicated by persistent coronary malperfusion? ATAAD might lead to coronary malperfusion caused by either static or dynamic obstruction of coronary blood flow (Figure 1). Coronary malperfusion is typically diagnosed by changes in the electrocardiogram, echocardiographic regional wall motion abnormalities, malperfusion on preoperative CT imaging, or intraoperative visualization of a disrupted coronary artery. Malperfusion with myocardial dysfunction is associated with high mortality and must be addressed expeditiously with restoration of

coronary blood flow to all parts of the myocardium. Although the literature is sparse, several techniques have been described, including: obliteration of the false lumen as part of the neomedia repair during valve resuspension; reinforcement of the coronary buttons with pericardium, felt, or other material during root replacement; and direct coronary artery repair or bypass grafting.²⁴⁻²⁶ Coronary artery bypass grafting for persistent coronary malperfusion despite successful direct coronary artery repair is necessary before leaving the operating room.

SECTION 6: ASCENDING AORTA AND DISTAL ANASTOMOSIS

The ascending aorta is the most likely segment of aorta to rupture in ATAAD. The surgical objectives during ATAAD repair are resection of the vulnerable aorta including the primary entry tear and restoration of flow into the true lumen. An open distal anastomosis to the proximal arch permits complete resection of the tubular ascending aorta and provides the option of a beveled anastomosis (hemiarch reconstruction) when the primary entry tear extends into the lesser curve of the aortic arch (Figure 3).

Various techniques have been described for preparing the distal anastomosis and includes simple suturing, multi-layer reconstruction with felt, pericardial gasket reinforcement, and buttressing with folded redundant adventitia (Figure 3).¹⁻³ The specific preparation technique for the distal anastomosis is less important than the actual quality of the suture line. During construction of the distal anastomosis, meticulous suturing is important to avoid new intimal tears, minimize operative bleeding complications, and provide late durability.⁴ Recommendations for management of the ascending aorta and distal anastomosis are outlined in Table 6.

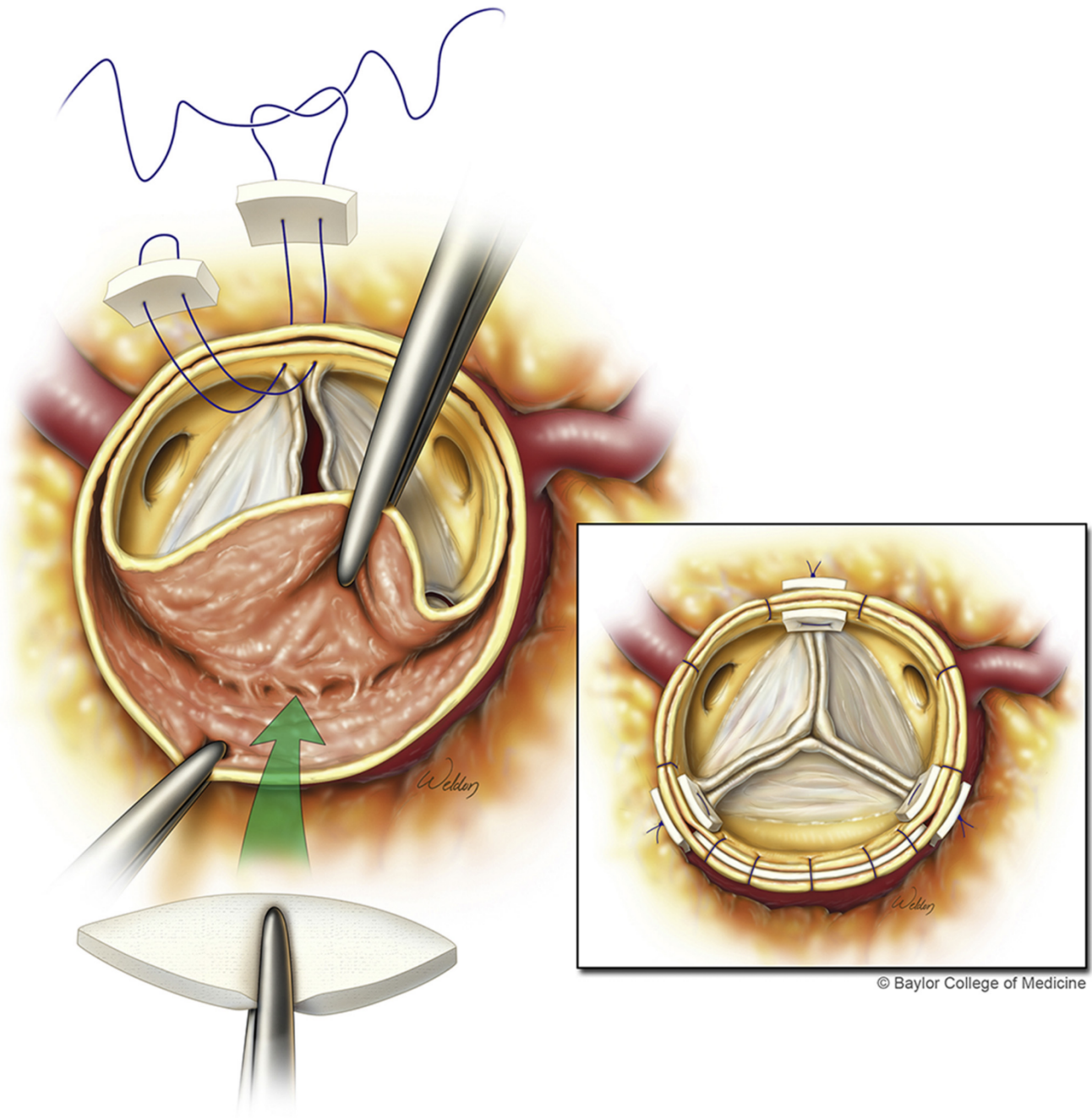


FIGURE 2. Aortic root management. The technique for valve resuspension involves 2 objectives highlighted in the figure. The first is to obliterate the false lumen in the aortic root by construction of neomedia (shown) with Teflon felt or bovine pericardium, use of a surgical adhesive, or simple suture repair. The second is to resuspend the very tips of the commissures with transmurals, pledged horizontal mattress sutures without leaving any tubular ascending aorta below the proximal suture line (*inset*).

Focused Clinical Questions

Should the distal aortic anastomosis be performed open or clamped? Because circulatory arrest was first described to facilitate aortic arch surgery, an open distal anastomosis for ATAAD repair has become well accepted for AD involving the arch, but not universally practiced. Advantages of circulatory arrest include optimal exposure of the anastomotic site and complete visualization of the

arch to rule out additional entry tears. An analysis by Lawton and colleagues showed no difference in early outcomes of open and clamped aortic anastomosis, however, they showed that open anastomosis with circulatory arrest was associated with a long-term survival benefit.⁹ In a single-center study, Malvindi and colleagues reported no difference in early outcomes of open and clamped anastomosis, but better downstream aortic

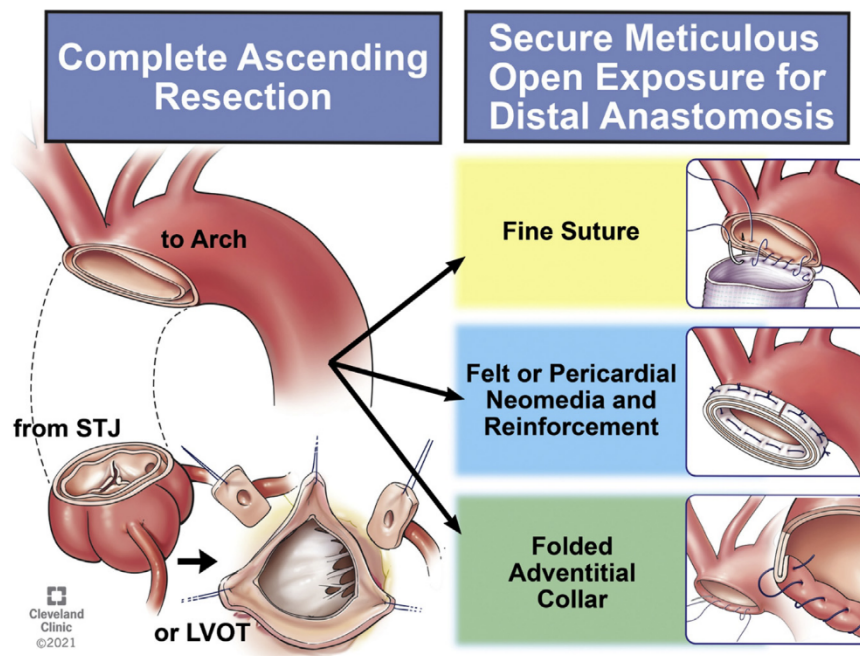


FIGURE 3. Ascending aorta and distal anastomosis management. The management of the ascending aorta and distal anastomosis involves 2 objectives shown in the illustration. The first is to completely resect the ascending aorta and primary entry tear, which requires circulatory arrest for an open distal anastomosis. The second is to restore the true lumen flow by reapposition of the dissected aorta during reconstruction of the distal anastomosis. *STJ*, sinotubular junction; *LVOT*, left ventricular outflow tract.

remodeling in the open anastomosis cohort.¹⁰ In a multi-center analysis of the Nordic Consortium for Acute Type A Aortic Dissection, Geirsson and colleagues reported that >13% of patients underwent repair with the clamped anastomosis.⁷ Compared with those who underwent open anastomosis under circulatory arrest, the clamped group had higher mortality and worse survival despite being younger with more bicuspid aortic valves. Although the clamped group had a lower risk of stroke, they also had more coronary disease and shock at presentation, therefore clamp versus open anastomosis was not an independent predictor of outcome. The authors concluded that open anastomosis with circulatory arrest was preferred, but in selected situations, such as patients in cardiogenic shock

at lower-volume hospitals, clamped anastomosis is a reasonable approach.

What extent of resection should be performed in the ATAAD? Several single-center and registry experiences have provided important guidance about basic principles regarding management of the ascending aorta during ATAAD repair, but no randomized clinical studies are available to evaluate the effectiveness of various techniques. Resection of the entire ascending aorta can reduce the risk for proximal aortic reoperation by excising the most vulnerable section of the thoracic aorta and primary entry tear.¹¹ In a recent analysis of reoperations after ATAAD repair, an aneurysmal aorta beyond the distal anastomosis was associated with a higher risk

TABLE 6. Management of ascending aorta and distal anastomosis recommendations

| Recommendations | COR | LOE | References |
|---|-----|-----|------------|
| Ascending Aorta and Distal Anastomosis Management | | | |
| 1. Circulatory arrest with open distal anastomosis is preferred in ATAAD. | I | B | 5-13 |
| 2. The extent of aortic resection should include the entire ascending aorta and primary entry tear. | I | C | 9,11,14 |

COR, Class of recommendation; *LOE*, level of evidence; *ATAAD*, acute type A aortic dissection.

or reoperation.^{15,16} Moreover, in DeBakey type II ADs where the tear and extent of intimal separation is limited to the ascending aorta, complete resection of the proximal aorta offers the potential for a curative resection.^{17,18}

In a comparison of ascending only (many with clamped technique) versus hemiarch reconstruction (all under circulatory arrest), Moon and colleagues reported 97% freedom from reoperation for the hemiarch group versus only 76% in the ascending only group at 10 years and no difference in survival.¹¹ Tamura and colleagues described a 20% incidence of new entry tears at the distal anastomotic site in their series despite the routine use of felt reinforcement and circulatory arrest.⁴ The presence of new entry tears was associated with greater distal aortic growth and event rates particularly when it involved the ascending aorta compared with re-entry tears in the descending aorta.

As newer currently investigational endovascular devices designed to treat the arch and ascending aorta have become available, the ascending aortic graft used for ATAAD repair can serve as the proximal landing zone for TEVAR. In such situations, a longer, kink-free landing zone will become increasingly important to optimize seal and fixation and might be best achieved by a complete resection of the ascending aorta.

What is the role of ascending TEVAR in ATAAD? Up to 20% of patients with ATAAD do not undergo surgery.^{19,20} Although ascending TEVAR might provide a treatment option for inoperable ATAAD patients, no devices are commercially approved; however, results of the early feasibility trials are pending.²¹ Initial clinical evidence has shown operative mortality of the procedure using first-generation devices to range from 0% to 13%.²²⁻²⁴ Long-term outcomes are unavailable. Until more evidence is available regarding safety and efficacy, ascending TEVAR for ATAAD should be considered investigational.

SECTION 7: AORTIC ARCH MANAGEMENT

Optimal management of the aortic arch in patients with ATAAD should balance the risk of extended aortic arch replacement with the benefit of additional resection of dissected aorta (Table 7). Most patients with ATAAD can be effectively treated with an open distal anastomosis and complete resection of the ascending aorta and hemiarch as recommended in the previous section. However, some clinical conditions might require an extended arch replacement to address primary entry tears not resectable with hemiarch alone, restore true lumen flow, or resect ruptured distal aorta. Different techniques for extended aortic arch replacement ranges from aortic arch replacement with reconstruction of arch branch vessels, to aortic arch replacement with classical or frozen elephant trunk (FET). Moreover, hybrid arch procedures using surgical and endovascular grafts have been used to address these complex ATAAD. Additional risks of extended aortic arch procedures might result from increased in circulatory arrest times and consequences of prolonged hypothermia, but these risks might be mitigated in experienced centers.¹⁻⁴ Multiple factors, such as patient anatomy, presenting features, and comorbidities should be considered when planning extended aortic arch replacement.

A sequential strategy for patients with ATAAD starts with addressing the life-threatening conditions at the initial operation and ends with the management of the lifelong complications of residual dissection in the distal aorta. Although extended aortic arch replacement has not been shown to reduce the risk of downstream aortic surgery, aortic arch replacement with FET has been shown to promote favorable aortic remodeling and facilitate future completion descending aortic surgery. Moreover, patients with HTAD potentially face a lifetime of aortic operations for replacement of residual, chronically dissected aorta. It is common for these patients to undergo multiple operations, so an extended aortic arch replacement with FET at the initial operation might

TABLE 7. Management of the aortic arch

| Recommendations | COR | LOE | References |
|--|-----|-----|------------|
| Aortic Arch Management | | | |
| 1. Extended aortic arch replacement is reasonable in patients with ATAAD and: - primary entry tear in the arch or proximal descending thoracic aorta, - brain or peripheral malperfusion, - arch or descending thoracic aortic aneurysm or rupture. | IIa | B | 1-9 |
| 2. Extended aortic arch replacement with frozen elephant trunk may be reasonable in ATAAD to promote favorable aortic remodeling. | IIb | B | 10-23 |
| 3. Extended aortic arch replacement may be considered in young patients with Marfan syndrome or hereditary thoracic aortic disorders. | IIb | C | 24-30 |

COR, Class of recommendation; LOE, level of evidence; ATAAD, acute type A aortic dissection.

reduce the total number of future operations. Performing surgery in a thoughtful sequence might therefore benefit young patients who are at high risk for future aortic complications, but acceptable surgical risk for extended aortic arch surgery at the time of ATAAD repair.

Focused Clinical Questions

When should extended arch surgery be performed in patients with ATAAD? Experience in aortic arch surgery is recommended when considering extended aortic arch replacement in patients with ATAAD. The additional risk of these procedures might be justified for patients with the clinical conditions listed in the recommendation. In 13% to 32% of ATAAD patients, the primary entry tear occurs in the aortic arch or proximal descending thoracic aorta,³¹⁻³⁴ and in many of these cases the primary entry tear cannot be resected with a hemiarch procedure. An extended aortic arch replacement might therefore be reasonable to resect or exclude the primary entry tear, especially when distal malperfusion is present.³⁵ In IRAD studies, brain malperfusion was reported in 6% to 15% of patients with ATAAD.³⁶⁻³⁸ Static occlusion of arch branch vessels might not be relieved by proximal aortic repair alone but might be suitable for arch branch reconstruction during aortic arch replacement.³⁹⁻⁴¹ Arch aneurysm is present in approximately 6% of ATAAD patients.⁴² Furthermore, rupture of the aortic arch might preclude safe hemiarch repair at the time of ATAAD and an extended aortic arch replacement might be required to secure a hemostatic distal anastomosis.

Is it reasonable to use FET in addition to ATAAD surgery? Aortic arch replacement with FET has the immediate benefit of true lumen expansion and coverage of entry tears into the false lumen. Several large series have shown a significant rate of false lumen thrombosis of the treated aorta as high as 85%, ensuring early aortic remodeling.¹⁷ Because the graft used for the aortic arch replacement is continuous with the stent graft of the proximal descending thoracic aorta, migration and type 1a endoleak are avoided in patients who undergo a total arch replacement with FET. Hence, the FET might prevent future operations in the treated segment.²¹

Although FET has the advantage of facilitating either an open or endovascular aortic repair, spinal cord injury (SCI) remains a concern especially when longer stent grafts are used. Several studies have shown that SCI can be avoided by limiting aortic coverage by the FET to at or above the sixth intercostal arteries.¹¹⁻¹³ A convenient echocardiographic and radiographic landmark is the level of the native aortic valve for the sixth thoracic vertebra. A more useful criteria might be the length of the stent graft because most commercial stent grafts are available in 10- or 15-cm lengths. A meta-analysis of more than 3000 patients has shown that 10-cm stent grafts were associated with a significantly lower rate of SCI.¹⁵

How should the arch be managed in patients with ATAAD and HTAD? Patients with HTAD such as Marfan syndrome are at risk of AD at a young age compared with the general population.^{43,44} Even after a successful ATAAD repair, the residual dissection in the distal aorta continues to dilate and half of these patients will face further aortic surgery.²⁶⁻³⁰ Moreover, in >40% of patients, the entire aorta will require replacement throughout the patient's lifetime because of aneurysmal degeneration or redissection.²⁸ Therefore, it might be reasonable to perform an extended aortic arch procedure in young HTAD patients with uncomplicated ATAAD. A strategy of arch replacement with elephant trunk (classical or frozen) followed by an elective thoracoabdominal aortic aneurysm repair might minimize the number and risk of combined aortic interventions for patients with HTAD and ATAAD.^{24,25}

SECTION 8: MALPERFUSION MANAGEMENT

Malperfusion is a life-threatening complication of ATAAD, with a reported incidence of 15% to 40%.¹⁻⁴ Coronary and brain malperfusion have been discussed in previous sections. Peripheral malperfusion is less common (spinal cord, 2%-5%; mesenteric, 4%-6%; renal, 6%-9%) but can be equally devastating.² Defined as ischemia of the vascular bed, malperfusion can lead to malperfusion syndrome characterized by end organ dysfunction.⁴ Moreover, end organ dysfunction can exist on a spectrum from systemic inflammation and metabolic derangement to overt organ infarction.

The traditional strategy for treating ATAAD complicated by malperfusion has been immediate proximal aortic repair to restore true lumen flow and resolve branch-vessel occlusion of the vascular bed (Figure 4).^{3,5} Peripheral revascularization is reserved for ongoing end organ ischemia after successful proximal aortic repair. An alternative strategy in stable patients with ATAAD and malperfusion syndrome reverses the order of interventions in a staged approach and features percutaneous or surgical revascularization as the initial intervention before proximal aortic repair (Table 8).⁶⁻¹¹ Using mesenteric malperfusion as the paradigm, this approach aims to resolve malperfusion syndrome, reduce operative risk of proximal aortic repair, and improve overall survival. However, this strategy might risk aortic rupture because proximal aortic repair is necessarily delayed, and an individualized approach is essential.

Focused Clinical Questions

How should ATAAD patients with malperfusion and end organ dysfunction be managed? Mesenteric malperfusion complicating ATAAD is lethal, and operative mortality after proximal aortic repair ranges from 41% to 100%.¹ An accurate diagnosis of malperfusion syndrome can be a challenge.

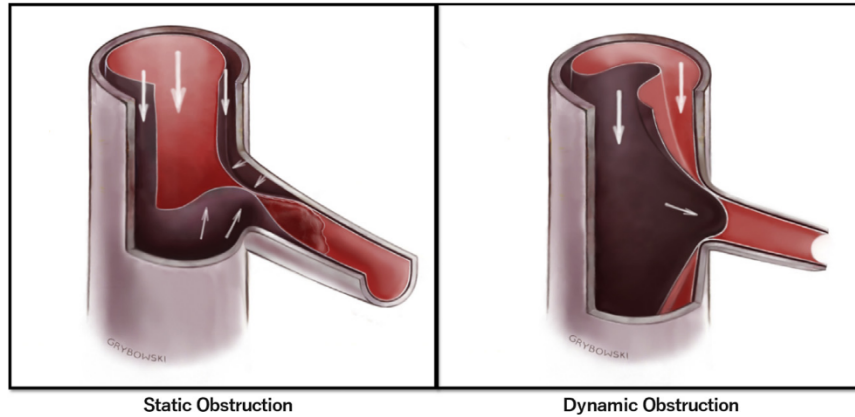


FIGURE 4. Branch vessel obstruction. Aortic dissection can obstruct the branch vessel by either static or dynamic obstruction. In static obstruction (*left panel*) the pressurized false lumen circumferentially narrows the branch vessel to the point of complete obstruction and distal thrombosis. In dynamic obstruction (*right panel*), the pressurized false lumen intermittently displaces the mobile dissection flap over the orifice of the branch vessel. Although dynamic obstruction can be relieved by proximal aortic repair and restoration of true lumen flow, static obstruction may not, because of resulting thrombosis.

Only 60% of patients present with abdominal pain and no laboratory study can completely confirm the presence or absence of mesenteric, end organ dysfunction. ATAAD and mesenteric malperfusion is often associated with liver, renal, and iliofemoral malperfusion due to true lumen compression throughout the abdominal aorta. Multicenter studies have suggested that mortality increases with an increasing number of affected vascular beds. A delay in diagnosis often results in prolonged reduction in blood flow to the splanchnic circulation, irreversible microvascular vasospasm, and transmural intestinal ischemia despite eventual reperfusion.

Historically, the treatment of ATAAD complicated by mesenteric malperfusion has been proximal aortic repair followed by exploratory laparotomy and bowel resection. Despite restoration of true lumen flow after an otherwise successful proximal aortic repair, patients died of metabolic abnormalities and end organ failure. In a report by the

IRAD, mesenteric malperfusion was detected in 68 (4%) of 1809 patients with ATAAD and mortality in this group was 63%.¹ Improving outcomes for this high-risk cohort requires rapid diagnosis and reversal of ischemia while minimizing the risk of aortic rupture.²

Because of the poor outcomes associated with traditional strategies, alternative approaches have been described that restore end organ perfusion before proximal aortic repair.^{2,3,6-8,10,12,16} A hybrid strategy allows treatment of mesenteric malperfusion and proximal aortic repair at the same setting.¹⁷ The rationale of proximal aortic repair immediately after mesenteric reperfusion is the avoidance of an interval of expectant management. Operative mortality is reported at 25% to 42% after combined open and endovascular treatment.^{1,18} This approach requires expertise in open and endovascular surgery, which might involve a team approach. There are no randomized studies on

TABLE 8. Management of malperfusion

| Recommendations | COR | LOE | References |
|--|-----|-----|------------|
| Malperfusion Management | | | |
| 1. It is reasonable to delay proximal aortic repair until after definitive treatment of mesenteric malperfusion. | IIa | B | 6-11 |
| 2. Descending TEVAR, aortic fenestration, and branch vessel stenting are reasonable treatment options for mesenteric malperfusion. | IIa | C | 9,12 |
| 3. It may be reasonable to perform revascularization for leg ischemia with sensory/motor deficits before proximal aortic repair. | IIb | C | 13-15 |

COR, Class of recommendation; LOE, level of evidence; TEVAR, thoracic endovascular aortic repair.

immediate aortic repair compared with initial treatment of mesenteric malperfusion followed by a period of medical stabilization before proximal aortic surgery.

Delay of proximal aortic repair in favor of initial treatment of malperfusion syndrome was first described by Deeb and colleagues; 20 patients were treated with percutaneous aortic fenestration and branch vessel stenting followed by a period of medical stabilization and then proximal aortic repair.¹⁰ Improved outcomes were observed in patients who had malperfusion addressed before aortic repair compared with historical controls, who had immediate aortic repair (75% survival vs 11% survival). Three patients who had initial treatment for mesenteric malperfusion died before operation including 1 patient who died of aortic rupture on day 12 of medical treatment. The 20-year experience of this strategy was recently updated by Yang and colleagues; mesenteric malperfusion in 82 patients was treated using endovascular reperfusion followed by delayed proximal aortic repair.⁸ In-hospital mortality for the entire cohort was 39%. Eleven patients (13%) died from aortic rupture before open aortic repair; all of these occurred in the first decade of this experience. In patients who recovered from malperfusion after endovascular therapy and went onto open surgical therapy, operative mortality of aortic repair was 2%.

Using lessons learned from experience with TEVAR for complicated acute type B AD, Leshnowar and colleagues reported their experience for treatment of mesenteric malperfusion using a “TEVAR-first” approach.⁶ Patients who had a traditional surgical strategy of initial proximal aortic repair had a 66% mortality compared with a 30% mortality in patients who had a TEVAR-first approach followed by surgical treatment of their dissection. One patient died because of aortic rupture after TEVAR.

How should ATAAD patients with limb ischemia be managed? The mechanisms of limb ischemia in ATAAD are secondary to either static or dynamic obstruction (Figure 4). In the setting of static obstruction (often as a consequence of false lumen thrombosis and true lumen compression in the iliofemoral vessels), proximal aortic repair is unlikely to result in restoration of limb perfusion, and adjunctive procedures are often necessary.^{13,14} However, dynamic limb malperfusion is associated frequently with associated visceral ischemia. Restoration of limb perfusion after prolonged ischemia can lead to consequences of ischemia reperfusion injury and result in metabolic derangements, renal failure, and loss of limb. Recognizing the association of increasing numbers of vascular beds affected by malperfusion and mortality, restoration of limb perfusion before aortic repair might lead to reduction in renal failure, and improvements in early mortality.^{1,12,15}

POSTOPERATIVE CONSIDERATIONS

Optimal postoperative management for patients recovering from ATAAD consists of blood pressure control and

lifelong radiologic follow-up with cross-sectional imaging of the entire aorta. Several aspects of long-term management should be taken into consideration. First, medical therapy aimed at reducing blood pressure as well as aortic wall stress should be initiated and properly explained to patients to ensure adequate medication adherence. Second, imaging tests should be scheduled at the appropriate time intervals. Diagnostic imaging should be used to assess the aortic valve and ventricular function as well as aortic remodeling. Finally, patients' first-degree relatives should be screened with transthoracic echocardiography (TTE) and if certain clinical features are present, the patient should be offered genetic testing for an HTAD.

Medical Therapy

Blood pressure management of patients after ATAAD repair has a dramatic effect on long-term outcomes. β -Adrenergic blockade reduces not only heart rate and blood pressure, but also provides anti-impulse therapy to decrease aortic wall stress independent of blood pressure effects.¹ In a recent nationwide study, Chen and colleagues showed beneficial effects of postoperative β -blocker use in 396 propensity score-matched pairs of patients.² Patients treated with β -blockers experienced lower mortality rates (16% vs 27%; $P < .001$) and strict medical compliance was also associated with improved outcomes. An analysis of the IRAD registry also showed improved survival ($P = .006$) in patients after ATAAD repair who were subsequently treated with β -blockers.³ This study showed no association between angiotensin-converting enzyme inhibitor use and mortality. Other studies have also confirmed the importance of β -blocker usage on outcomes after ATAAD repair.^{4,5}

A growing body of evidence has identified fluoroquinolones, one of the most commonly used group of antibiotics, as causing aortic complications in patients with AD, aneurysm, or HTAD.⁶⁻⁸ Experimental studies have also confirmed that fluoroquinolones can exacerbate aortic wall damage in the setting of underlying aortic disease.^{9,10} These findings have been confirmed in population-based studies, leading the US Food and Drug Administration to issue a warning against prescribing systemic fluoroquinolones for patients who have or are at risk of developing an aortic aneurysm because of the increased risk of aortic rupture or dissection.

Postoperative Imaging

Long-term surveillance of patients who underwent successful repair of ATAAD is imperative. Aortic events including rupture and reoperation are a major cause of late morbidity and mortality. Patients with residual distal dissections and particularly those with a patent distal false lumen and proximal tears are at greatest risk for aortic growth and reintervention.¹¹ Monitoring of the aortic valve and heart function with echocardiography is also critical.

Cross-sectional imaging with either CT or MRI may be used to identify aortic growth and other aortic complications. We suggest that if the renal function permits, predischarge CT or MRI should be performed in addition to scans at 6 months and 1 year. These studies are best performed in multiple phases including noncontrast, arterial phase, and delayed venous phase modes of acquisition to allow for a more complete assessment of false lumen perfusion. Subsequent imaging intervals should be determined according to the condition and size of the residual native aorta, with annual scans being sufficient for most patients.⁵

Genetic Testing and Family Screening

All first-degree relatives of patients who have suffered an ATAAD should be screened with a TTE to rule out the presence of an aortic root or ascending aortic aneurysm. TTE is often sufficient as a diagnostic modality for aortic assessment in family members and should be repeated at 5- to 10-year intervals if no aortic pathology is identified.¹²⁻²¹ Patients with a suspected HTAD including those with syndromic features, a family history of aortic aneurysm or dissection, or young age at diagnosis (younger than 50 years of age or age 50-60 years without hypertension) should be offered genetic assessment and testing. If a pathogenic variant is identified in the patient, then cascade testing of family members should be offered.

CONCLUSIONS AND FUTURE DIRECTIONS

Because of the lethal nature of ATAAD, a condition that is relatively uncommon and not frequently encountered, this writing committee sought to create a set of reasonable evidence-based recommendations aimed at aiding the practicing surgeon in the management of the condition. A well established first principle is the use of anti-impulse therapy before surgical intervention. Second is that expeditious surgery is the preferred approach to ATAAD in almost all instances, including for patients with neurologic deficits. Third, the conduct of the operation should be performed in a manner that is familiar and comfortable for the surgeon. The results of a variety of approaches to arterial cannulation and body temperature management have been reported in nonrandomized studies, with advocates for each; however, the importance of adequate end organ perfusion during repair as a goal is undisputed. The use of circulatory arrest for open distal anastomosis is accepted as is the establishment of antegrade flow after distal repair. Although there is continued controversy over the optimal proximal and distal extent of repair as a means to reduce the risk of late reoperation, there is clear consensus that the highest priority of ATAAD repair is safe conduct of the index operation. Significant root and isolated valve pathology, whether due to the dissection process or underlying aneurysmal disease, should be dealt with in a safe manner either by simple

aortic valve resuspension or root replacement/reconstruction. The extent of distal repair is dictated primarily by the location of the entry tear and the imperative to resect or cover the primary tear. The ultimate role of stent graft techniques including the FET remain uncertain. Malperfusion remains the single greatest challenge in the management of ATAAD, with increasing consideration given to reperfusion of the compromised bed before proximal aortic repair if involving the viscera or limbs. In most other instances, however, proximal aortic repair first is still preferred. Finally, the importance of medical optimization to complement operative intervention cannot be overemphasized. Late postoperative β -blockade and imaging surveillance have the potential to greatly affect the disturbingly high late mortality associated with this condition. Similarly, genetic testing and family counseling might identify individuals for prompt prophylactic interventions that prevent this dreaded disease.

Future directions should be targeted at system-level development of timely and rational triage of the patients, improved operative techniques, innovative technology, and stratagems to improve adherence to postoperative management. Finally, prevention and management of ATAAD complications will require detailed databases with long-term outcomes and randomized studies to characterize interventions associated with survival and to inform clinical decision-making.

Conflict of Interest Statement

S.C.M. is a consultant for Terumo Aortic, Cryolife, Edwards, and Medtronic, and has received speaker fees from Terumo Aortic, Cryolife, Edwards, and Medtronic. W.Y.S. is a consultant for Edwards Lifesciences, Medtronic, Terumo Aortic, Aquedon Medical, and Microinterventional Device, has received speaker fees from Edwards Lifesciences, Medtronic, and Terumo Aortic, and has received research funds from Edwards Lifesciences, Medtronic, Terumo Aortic, WL Gore, Boston Scientific, and Cardiac Dimensions. J.S.C. is a consultant for and has received speaker fees from Terumo Aortic, and has received research funds from Medtronic, Abbott, CytoSorbents, Edwards Lifesciences, and WL Gore. T.G.G. is a consultant for Abbott. M.O. is a consultant for Medtronic. E.E.R. is a consultant for Cryolife, WL Gore, and Medtronic, has received speaker fees from Abbott, Cook, Edwards Lifesciences, LivaNova, and Terumo Aortic, and has received research funding from WL Gore. M.L.S. is a consultant for and has received speaker fees from Terumo Aortic. M.R.M. is a consultant for Medtronic. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Section 8: Malperfusion Management

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Postoperative Considerations

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Key Words: acute type A aortic dissection, ATAAD, expert consensus document, ECD

**APPENDIX E1: LITERATURE REVIEW
METHODOLOGY**

a. Detailed results of literature search

| Literature search author | | |
|--|-----------------------------|--|
| Michelle R. Demetres, MLIS | | |
| Scholarly Communications Librarian | | |
| Weill Cornell Medicine | | |
| Samuel J. Wood Library and C.V. Starr Biomedical Information Center, New York, New York | | |
| | Total Ovid MEDLINE searches | Total Ovid MEDLINE publications reviewed |
| Section 1 | 3 | 347 |
| Section 2 | 1 | 126 |
| Section 3 | 6 | 1356 |
| Section 4 | 3 | 2418 |
| Section 5 | 6 | 3962 |
| Section 6 | 10 | 1006 |
| Section 7 | 8 | 1656 |
| Section 8 | 8 | 557 |
| Total | 35 | 11,428 |

b. MEDLINE search – exemplary details

Transfer to Aortic Center

Ovid MEDLINE (ALL – 1946 – October 1, 2020)
Searched on October 5, 2020
No language, publication date, or article type restriction

| Line | Search | Number of results |
|------|--|-------------------|
| 1 | Aneurysm, Dissecting/ | 17,888 |
| 2 | Aortic Aneurysm/ | 21,503 |
| 3 | Aortic Aneurysm, Thoracic/ | 12,521 |
| 4 | (acute type A adj2 (aortic or aorta or dissecti*)).tw. | 1834 |
| 5 | acute Stanford type A aortic dissection*.tw. | 110 |
| 6 | (ATAD or ATAAD or TAAD).tw. | 476 |
| 7 | acute aortic syndrome.tw. | 373 |
| 8 | thoracic aortic dissecti*.tw. | 639 |
| 9 | or/1-8 | 40,298 |
| 10 | Hospitals, High-Volume/ | 1605 |
| 11 | (high* volume adj3 (surgeon* or hospital* or center*)).tw. | 6144 |
| 12 | (aortic center* or aortic treatment center*).tw. | 84 |
| 13 | aortic dissection team*.tw. | 3 |
| 14 | or/10-13 | 6985 |
| 15 | 9 and 14 | 125 |

Surgical Repair of the Distal Anastomosis of the Aorta

Ovid MEDLINE (ALL – 1946-December 8, 2020)
Searched on December 8, 2020
No language, publication date or article type restrictions

| Line | Search |
|------|--|
| 1 | (distal anastomosis or ((distal aorta or distal aortic) adj3 anastomosis)).tw. |
| 2 | (surgery or surgeries or surgical or operation or operate or operations).ti. |
| 3 | 1 and 2 |

TOTAL: 307

Initial Step for Visceral Malperfusion Occurring After Initial ATAAD Surgery?

Ovid MEDLINE (ALL – 1946-present)
Searched on January 29, 2021
No language, publication date or article type restrictions

| Line | Search |
|------|---|
| 1 | Aneurysm, Dissecting/ |
| 2 | Aortic Aneurysm/ |
| 3 | Aortic Aneurysm, Thoracic/ |
| 4 | (acute type A adj2 (aortic or aorta or dissecti*)).tw. |
| 5 | acute Stanford type A aortic dissection*.tw. |
| 6 | (ATAD or ATAAD or TAAD).tw. |
| 7 | acute aortic syndrome.tw. |
| 8 | thoracic aortic dissecti*.tw. |
| 9 | or/1-8 |
| 10 | (visceral malperfusion* or visceral ischaemia* or end organ maleperfusion*).tw. |
| 11 | 9 and 10 |

TOTAL: 78

Aortic Root Management in Patients With ATAAD and Marfan Syndrome

Ovid MEDLINE (ALL – 1946 – November 30, 2020)
Searched on December 1, 2020
Nolanguage, publication date or article type restrictions

| Line | Search |
|------|--|
| 1 | Aneurysm, Dissecting/ |
| 2 | Aortic Aneurysm/ |
| 3 | Aortic Aneurysm, Thoracic/ |
| 4 | (acute type A adj2 (aortic or aorta or dissecti*)).tw. |
| 5 | acute Stanford type A aortic dissection*.tw. |
| 6 | (ATAD or ATAAD or TAAD).tw. |

(Continued)

ADULT

Continued

| Line | Search |
|------|---------------------------------------|
| 7 | acute aortic syndrome.tw. |
| 8 | thoracic aortic dissecti*.tw. |
| 9 | or/1-8 |
| 10 | Marfan Syndrome/ |
| 11 | (marfan or marfans or "Marfan's").tw. |
| 12 | 10 or 11 |
| 13 | 9 and 12 |
| 14 | ((aortic or aorta) adj3 root).tw. |
| 15 | 13 and 14 |

TOTAL: 563