Critical Review of Guidelines for Type B Aortic Dissection

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The management of type B aortic dissection is one of the most challenging and debated topics in contemporary cardiovascular surgery practice. Patients with acute or chronic dissection-related complications face high morbidity and mortality if not treated promptly. For most patients requiring intervention, thoracic endovascular aortic repair (TEVAR) is considered the gold standard. However, both early and late TEVAR-related complications make decision-making complex, even for experienced clinicians. In many cases, optimal medical management with longitudinal imaging surveillance may be preferred. In response to these challenges, several societal guidelines have recently been published to provide evidence-based or expert consensus "best practice" recommendations. Although these guidelines share many commonalities, they also highlight key unresolved clinical questions. For example, debates persist over the appropriate use of TEVAR for "uncomplicated" TBAD, defining "high-risk" criteria for uncomplicated presentations, and management of the false lumen, among other topics. Despite recent updates, a critical evaluation of the nuanced differences between these guidelines is lacking. Therefore, the purpose of this review is to compare current clinical practice guidelines, highlight their similarities and differences, and offer a comprehensive evaluation of the evidence surrounding management of TBAD. Moreover, this analysis will provide recommendations to address important knowledge gaps.

INTRODUCTION

1. **Description of Current Guidelines:** Since 2017, at least 5 different major updates have been published on the management of type B aortic dissection (TBAD) by various European and North American cardiology, cardiothoracic and vascular societies.[1−5] Among these guidelines, several key similarities emerge. They all define TBAD as one occurring distal to the left subclavian artery, without involvement of the ascending aorta. Additionally, all guidelines

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agree that an acute dissection is diagnosed within 14 days of symptom onset, a subacute dissection is defined as occurring between 14 and 90 days, and a chronic dissection refers to cases identified beyond 90 days. The initial medical management of uncomplicated acute dissections is consistent across guidelines, with antihypertensive therapy recommended as the first-line treatment. For complicated acute TBAD—such as cases involving rupture, malperfusion, or rapid aortic expansion—surgical or endovascular intervention is universally advised, with TEVAR as the preferred approach. Long-term management across all guidelines emphasizes the importance of strict blood pressure control and longitudinal surveillance imaging.

2. **Significance and Epidemiology:** Aortic dissection (AD) is one of the most common catastrophic events affecting the thoracoabdominal aorta, with an incidence that frequently exceeds that of ruptured degenerative infrarenal abdominal aortic aneurysm.[6] Historical population-based studies estimate the annual incidence of acute AD to be 2.9−3.5 per 100,000 person-years.[6,7] Recent reports suggest an increasing incidence among certain subgroups, often attributed to an aging population, improved diagnostic capabilities (e.g., ubiquitous exposure to computed tomography (CT)/magnetic resonance imaging (MRI)/echo, etc.), and greater prevalence cardiovascular risk factors.[8] Aortic dissections more commonly occur in the ascending aorta (∼65%) compared to the descending thoracic aorta (∼25%) and the aortic arch (∼10%)[9]; however, this review focuses specifically on dissections involving the descending thoracic aorta, distal to the left subclavian artery. While dissection can affect any patient population, 60% of those affected are males and the mean age at presentation typically is in the seventh decade of life.[10] Among younger patients, genetic vulnerabilities, hypertension and recent drug use are important inciting risk factors.[10]
3. **Pathophysiology and Classification:** TBAD is a dynamic process with variable clinical presentations. In the acute phase, a transverse intimal tear, often localized just distal to the left subclavian artery, allows blood to enter a false lumen bound by the adventitia. This blood flow can result in either a longitudinal or spiral injury to the aortic wall. Aortic dissections are typically classified by the location of the tear and the time between symptom onset and presentation, with designations of acute, subacute, and chronic. In the acute phase (<14 days from symptom onset), the aortic wall is fragile due to the recent tear and ongoing false lumen pressurization. This creates a risk for rapid aortic expansion, rupture, and malperfusion due to either dynamic or static obstruction of branch vessel origins. In contrast, the chronic phase involves thickening, fibrosis, and remodeling of the aortic wall, leading to reduced elasticity. Persistent perfusion of the false lumen increases the risk of aneurysm formation, which can lead to rupture in the long term.

CLINICAL DEFINITIONS AND CLASSIFICATION

1. **Acute Type B Dissection: Complicated versus Uncomplicated:** All guidelines uniformly define ''complicated'' acute TBAD by life-threatening features including rupture, clinical malperfusion, and/or rapid aortic expansion. However, there is limited clarification surrounding ''subclinical'' malperfusion, in the context of imaging findings such as renal or mesenteric branch vessel compromise without overt clinical symptoms (e.g., no creatinine rise or abdominal pain). In the European Association for Cardiothoracic Surgery (EACTS)/Society for Thoracic Surgery (STS) 2024 guideline, there is suggestion for close monitoring and further imaging before escalating management to intervention unless clinical symptoms manifest. Similarly, the American College of Cardiology (ACC)/American Heart Association (AHA), European Society of Cardiology (ESC), and European Society of Vascular Surgery (ESVS) guidelines acknowledged radiographic malperfusion but emphasize the need for aggressive medical management, serial imaging, and clinical correlation before TEVAR. Notably, the Society for Vascular Surgery (SVS)/STS reporting standards identify radiographic malperfusion as a ''precomplication'' state and advocate that management in this scenario be individualized and context-dependent and concede that some experts consider early intervention appropriate to prevent progression to overt clinical malperfusion.

The definition of refractory pain is often described in all the documents as persistent or worsening pain that does not respond to initial medical therapy. However, there is little detail that clarifies this distinction. Further, uncontrolled hypertension is usually described as persistent hypertension despite aggressive medical management but the number/

dose of medications or exact blood pressure thresholds and time over which this determination is made is ambiguous or missing in most descriptions. The categorization of refractory pain and/or uncontrolled hypertension in acute TBAD varies slightly among the 5 guidelines. Across all 5 guidelines, refractory pain and uncontrolled hypertension are generally categorized as a high-risk phenotype of acute TBAD. These symptoms are seen as indicators of impending clinical deterioration, potentially leading to rupture or malperfusion. TEVAR or surgical intervention is often recommended if these signs are not controlled, reflecting a consensus that they represent a higher risk for poor outcomes. The nuanced variation is rooted in differences in the definitions, as well as the general interpretation and philosophy of the consensus statements surrounding TEVAR management of this scenario in acute TBAD.

Key differences include the EACTS/STS and SVS/STS guidelines that are more explicit in defining symptoms and attributing this to a ''complicated'' presentation, with clear recommendations for early TEVAR. The ACC/AHA follow a similar approach but leave some flexibility for managing borderline cases and advocate use of additional clinical judgment before intervention. The ESC guidelines tend to favor a slightly more conservative approach, emphasizing that medical management really must be maximized before TEVAR but submit that with persistent symptoms, escalation of management is justified. Finally, the ESVS document identifies these clinical features as being ''complicated'' but recommends urgency of TEVAR to a greater extent relative to the ESC guideline.

2. **Chronic Type B Dissection: Stable versus Progressive:** A ''stable'' chronic TBAD is generally characterized across the different guideline documents by having no radiographic evidence of aortic enlargement or new symptoms and are recommended to receive ongoing best medical therapy and regular surveillance. In contrast, ''progressive'' dissection involves increasing aortic size, new symptoms, or persistent false lumen perfusion, prompting consideration for intervention. While all guidelines emphasize the importance of imaging surveillance to assess stability, there are nuanced differences in the criteria for defining progression and recommending treatment. Most guidelines (EACTS/STS, SVS/STS, ESC, and ESVS) define progression with an annual growth rate of >5 mm/year, whereas the ACC/AHA guidelines set a higher threshold of ≥ 10 mm/year. Regarding absolute aortic diameter, the ESC guidelines recommend intervention at ≥ 60 mm, the highest threshold, while the EACTS/STS, SVS/STS, ACC/AHA, and ESVS guidelines advise intervention when the diameter reaches ≥ 55 mm.

COMPLICATED AND UTBAD

1. **Risk Assessment and Classification:** A closer examination of the guidelines reveals that differences in recommendations for treating TBADs stem from how risk is assessed, and, crucially, whether ''uncomplicated'' TBAD truly exists.[11–16] While the terms ''uncomplicated'' and ''complicated'' are commonly used, some experts argue that ''uncomplicated'' is a misnomer, a pretense of stability masking the risk of future complications. The ESVS differentiates between complicated and uncomplicated dissections mainly based on clinical features (Table I). However, their interpretation of ischemic features, such as elevated serum creatinine, as a marker for renal ischemia is not clearly defined.[15] The SVS and STS reporting standards clarify that acute kidney injury alone does not define malperfusion and recommend following the Acute Kidney Injury Network grading scheme with careful monitoring for oliguria or anuria.[16] These guidelines stress that an ''uncomplicated'' dissection must lack high-risk clinical and radiographic features (Table I).

While the STS/AATS and ACC/AHA guidelines mention these features, they do not elaborate. In contrast, the EACTS/STS guidelines refrain from using the term ''uncomplicated'' and instead categorize TBADs as complicated or as having high-risk features, as defined in Table II. A controversial aspect of these guidelines is that they recommend intervention (TEVAR or frozen elephant trunk [FET]) in dissections with high-risk features, even in the absence of clinically documented complicating features.

2. **Medical Therapy:** There is strong consensus across guidelines on the role of optimal medical therapy, particularly blood pressure and heart rate control, aiming for systolic blood pressure below 120 mm Hg and heart rate below 60 beats per min. Although this recommendation is intuitively sound and supported by evidence,

Table I. Defining features of a complicated acute type B aortic dissection according to the European Society of Vascular guidelines on the Management of Descending Thoracic Aorta Diseases[15]

Aortic rupture and/or hypotension/shock
Rapid aortic expansion
Visceral, renal, or limb ischemia
Paraplegia/paraparesis
Periaortic hematoma
Recurrent or refractory pain
Refractory hypertension despite adequate medical therapy

Table II. High-risk type B aortic dissection features, according to the reporting standards from the Society for Vascular Surgery and Society of Thoracic Surgeons[16]

Refractory pain
Refractory hypertension
Bloody pleural effusion
Aortic diameter >40 mm
Radiographic only malperfusion
Readmission
Entry tear: lesser curve location
False lumen diameter >22 mm

it is based on relatively limited studies, such as the 2010 AHA guidelines.[15,17]

It should also be noted that the guidelines emphasize treatment as goal- or target-directed therapy, and B-blockade is the unanimous first-line agent. While not guideline-oriented, one recent analysis compared strict systolic target blood pressure levels below 120 mm Hg versus more liberal levels up to 140 mm Hg.[18] It found that stricter targets required more medication, led to higher rates of acute kidney injury, and showed no difference in overall survival. This finding, along with other nuances of antihypertensive therapy, is not addressed in the guidelines. In addition to antihypertensive regimens, all of the guidelines emphasize the importance of adequate pain management, with the ACC/AHA specifically recommending intravenous morphine. In the acute phase, there are no additional medical requirements. Over time, once clinical stabilization occurs, a shift from intravenous to oral medications is recommended. Long-term therapy should focus on lifestyle-related risk reduction, discussed further below.

3. **Indications and Modalities for Intervention:** The concept of "uncomplicated" AD provokes discussion surrounding the appropriate and evidence-based indications for intervention. If a dissection is categorized strictly as being uncomplicated, intervention would no longer be justified in most scenarios due to the lack of Level I evidence, especially since this classification implies the absence of complications. However, due to the often debated terminology and the dynamic nature of the associated pathophysiology which contributes to significant patient heterogeneity, it is understandable why there is dissent among providers about how this term is applied in real-world practice. This may be why the EACTS avoids using the term. Intervention recommendations across guidelines often reflect varying risk categories, particularly in terms of preventing mortality. All guidelines agree that TEVAR is the first-line treatment for complicated dissections, with open surgery considered when endovascular techniques are unsuitable due to anatomical constraints. Interestingly, the ACC/AHA guidelines note the lack of randomized controlled trials (RCTs) comparing open surgery with endovascular repair for complicated TBADs.[11]

For uTBADs, including those with high-risk features, the indications for intervention are less clear. The ESVS guidelines cite the Acute Dissection Stent graft OR Best medical treatment trial as evidence showing morphological improvement following TEVAR in uTBAD patients.[19] In contrast, they do not cite the only other RCT, the extended Investigation of STEnt Grafts in Aortic Dissection-XL trial, and its improved aorta-specific survival at 5 years.[20] Because of its original lack of statistical power and the use of a Landmark analysis, there have been calls for renewed RCTs with focus on long-term survival, 2 of which are clinically underway.[21–23] The Investigation of STEnt Grafts in Aortic Dissection-XL trial is, however, cited by all of the other guidelines, as are numerous other retrospective studies, as evidence that prophylactic TEVAR delays disease progression and has a reasonable perioperative safety profile. For example, the EACTS/STS guidelines highlight the low 30-day mortality and perioperative complications from the Global Registry for Endovascular Aortic Treatment registry among patients with or even without risk features. Because of these data, the EACTS/STS guidelines condense their perspective, suggesting that ''TEVAR might be considered in those patients with suitable anatomy.''[13]

Given the attention rendered to these high-risk features, it is interesting to summarize the evidence

levels that are ascribed from the different societies. The ESVS (class IIb, level B) and ESC (class IIa, level B) note that intervention ''may'' be considered to prevent aortic complications, whereas the EACTS (class IIa, level C) endorses that intervention ''should'' be considered, although with no specified objective. Furthermore, it is worth highlighting that the ESVS cites the Acute Dissection Stent graft OR Best medical treatment trial as their reference for their recommendation, while the EACTS/STS provides none. An important factor in early TEVAR is anatomical suitability, a consideration that the guidelines do not fully address, as newer devices and techniques, such as branched TEVAR devices for zone 2 deployment, are emerging.[24,25]

All guidelines advocate for left subclavian revascularization, primarily to prevent stroke and spinal cord ischemia (SCI). However, the EACTS/STS guidelines go further by recommending that FET arch repair be considered in cases with less favorable proximal landing zones. Such anatomically challenging cases are associated with increased procedural complexity and higher perioperative risks, particularly SCI. While many guidelines suggest considering prophylactic cerebrospinal fluid (CSF) drainage, the more recent EACTS/STS guidelines reflect a shift in paradigm, emphasizing rescue CSF drainage and blood pressure control protocols over prophylactic measures. Notably, none of the guidelines fully address or quantify the balance of risks and benefits in increasingly complex procedures, especially for patients with clinically uncomplicated dissections.

Moreover, for elderly patients with comorbidities, even when clinically uncomplicated, the risks of prophylactic surgery may outweigh the benefits. This dilemma is not fully addressed in the EACTS/STS guidelines, which suggest continued medical therapy or a FET for patients with high-risk features and an ascending aorta $\geq$4.5 cm.

An important consideration in the treatment of AD patients is the presence of confirmed or suspected connective tissue disease (CTD), often referred to as heritable thoracic aortic disease (HTAD). The ESVS guidelines specifically recommend that patients younger than 40 years presenting with an AD be thoroughly evaluated for an underlying HTAD, a distinction not explicitly emphasized in other guidelines. Although evidence regarding the optimal indications and methods for repair in HTAD patients remains limited, all guidelines agree that lower thresholds for early intervention—such as reduced aortic diameter criteria—are appropriate in this population.

4. **Timing of Intervention:** The timing of intervention is another area of variability among guidelines, largely influenced by how risk is classified. The ESVS guidelines discuss the subacute window (3–7 days), where the aortic septum is pliable but the aorta is not overly fragile, and they suggest this period as the optimal time for intervention. The SVS adds a ''hyperacute'' phase (<24 hr) based on Vascular Quality Initiative national registry data showing worse outcomes in this early phase.[26] Finally, the EACTS has no specific recommendation for the timing of intervention, emphasizing a general philosophy of early and proactive treatment.
5. **Surveillance and Follow-up:** All guidelines stress the importance of monitoring both in the early phase and for lifelong surveillance, regardless of whether surgical intervention has occurred. Standard follow-up includes imaging at 1 month, 6 months, 1 year, and then annually thereafter. More frequent monitoring may be necessary during the acute phase, especially for patients with precarious anatomical or clinical features. Long-term management focuses on maintaining blood pressure control and addressing cardiovascular risk factors such as smoking and weight management. The ESC notes that statin therapy may improve survival.[12] While interventions and reinterventions are common, the role of additional therapies like antithrombotic medications or statin agents is unclear, particularly for patients with concomitant cardiovascular comorbidities.

CHRONIC TYPE B AORTIC DISSECTION

1. **Definition and Diagnostic Criteria:** Chronic Stanford Type B aortic dissection (CTBAD) is defined when time from symptom onset exceeds 90 days.[16,27] This distinction between subacute (15–90 days) and chronic TBAD are based on clinical outcomes, changes in dissection flap architecture and plasticity, expansion rates, and therapeutic response (such as endograft-induced aortic remodeling). In the acute and subacute phases, the aorta undergoes rapid dilation followed by a slower growth, eventually plateauing around 3 months after symptom onset. Concurrently, the dissection flap thickens, stiffens, and becomes less mobile during this time.[28] Fluorodeoxyglucose positron emission tomography (FDG-PET)/MRI studies indicate enhanced FDG uptake in the acute and subacute

phases of AD, which stabilizes in the chronic phase.[29] These dynamic structural changes increase the risk of aortic-related complications and death during the first 90 days, justifying separate consideration of CTBAD.

Imaging may reveal predictors for aortic growth or late mortality. An entry tear larger than 10 mm in diameter or located in the aortic arch or the proximal descending aorta has been associated with increased late mortality.[30] The size and number of fenestrations may also predict aneurysmal dilation. Additionally, false lumen characteristics, such as partial thrombosis, or false lumen area size (over 70% of the total aortic area) are possible predictors for adverse outcomes. In contrast, complete thrombosis of the false lumen is associated with slower aortic growth.[31] Gadolinium enhanced MRI suggests inflammatory activity, and it is more common in patients with TBAD who require surgical intervention.[29] Although these markers may inform clinical decisions, they are based on small cohort studies and have not been validated in prospective trials.

Aneurysmal degeneration is the most common complication in TBAD, with an increased growth rate (0.1−0.74 mm/year), particularly affecting the proximal descending aorta. The false lumen may remain patent in patients undergoing TEVAR, hence delayed aortic expansion occurs regardless of initial treatment modality.[32,33] Results of the International Registry of Acute Aortic Dissection found that 73.3% of patients who were medically treated and 62.7% of patients who were treated by TEVAR developed aneurysmal degeneration at 5 years.[34] Rarely, progression of the initial dissection or a secondary aortic event might lead to intractable pain, rapid growth, visceral malperfusion, or aortic rupture in both medically treated, and TEVAR groups in the long term.[35,36]

2. **Management Strategies:** Maintaining target blood pressure through some form of multimodal antihypertensive therapy (e.g., alpha blockers, calcium channel blockers, diuretics, and angiotensin blockade) is crucial for CTBAD management. Adherence to this treatment strategy has been shown to reduce the risk of aortic-related complications and mortality. Specifically, long-term treatment with β-blockers is recommended since this has been shown to decrease progression, decrease the number or aortic events, and improve survival.[37] Cardiovascular risk management is equally critical due to high cardiovascular-related mortality rates in TBAD patients.

Intervention timing should be individualized, taking into account clinical and radiological findings alongside patient risk. For CTBAD, guidelines generally suggest intervention when aortic diameter exceeds 5.5−6.0 cm, with specific adjustments based on factors such as growth rate or symptoms, or presence of CTD (Table III).

Open surgery is recommended as the preferred treatment for CTBAD in low-risk patients across current guidelines. However, reliable prediction of operative risk remains challenging and outcomes highly depend on the expertise of the treating center and team. TEVAR has better perioperative mortality and morbidity outcomes, but there is no significant survival difference when compared to open aortic surgery at 1 and 3 years.[38,39] However, concerns about the long-term durability of TEVAR and the need for reintervention remain. This issue is related to changes in dissection flap characteristics, which can affect endograft expansion and aortic remodeling.

Currently, no specific recommendations exist for or against endovascular aortic repair (EVAR) in patients with CTD and CTBAD. However, the ACC/AHA and STS/AATS guidelines recommend open surgical reconstruction over endovascular techniques for CTD patients with thoracoabdominal aneurysms. While concerns remain about the durability of EVAR and the potential need for secondary reinterventions in this patient population, recent evidence suggests that EVAR offers lower perioperative mortality and comparable mid-term survival to open surgery. These findings may prompt future modifications to guideline recommendations, given the growing use of EVAR and its satisfactory mid-term outcomes.[40,41]

Guidelines universally recommend implementing spinal protective strategies to mitigate the risk of spinal cord ischemia. Current guidelines advocate for prophylactic CSF drainage in patients at high risk of spinal cord injury, including those with extensive thoracic aortic coverage, prior abdominal aortic surgery, severe thoracic aortic atherosclerosis, or occlusions of the left subclavian artery and/or hypogastric arteries. Additionally, the ESVS guidelines emphasize combining CSF drainage with left heart bypass and moderate hypothermia to reduce mortality and spinal cord injury during open CTBAD repair.

However, concerns about the risks associated with CSF drainage have led some centers to move away from prophylactic drains. Instead, there is increasing focus on alternative spinal protection strategies, such as avoiding perioperative hypotension and staging endovascular procedures in thoracoabdominal repairs. Whether these evolving

Table III. Summary of recommendations for imaging surveillance, indication for intervention, and repair modality

Guideline	Imaging surveillance	Indication for intervention	Repair modality
SVS	1, 3, 6 months, annually. (extend to 18−24 months if stable)		
ESVS	3, 6 months, annually (extend to 2−3 years if stable after 3 years)	5.6−5.9 cm—may be considered; ≥6 cm—should be considered; ≥6 cm if thoracoabdominal extension is present; Emergency repair: Malperfusion, rupture, progression of dissection	Open repair in dedicated centers, with adjunct techniques. TEVAR for patients with moderate to high surgical risk or contraindication.
ACC/AHA	1, 6, 12 months and annually if stable	Chronic residual aortic dissection and total aortic diameter of ≥5.5 cm	
STS/AATS		≥5.5−6 cm, or growth rate >10 mm/year; Symptomatic; acute redissection or rupture	Open repair unless comorbidities are prohibitive or anatomy not suitable for TEVAR; TEVAR for high-risk patients with suitable anatomy
EACTS/STS	6, 12 months, and annually for 5 years if stable	Chronic residual aortic dissection, and total aortic diameter ≥5.5 cm, without involvement of the ascending aorta; <5.5 cm in patients with HTAD; In patients with dSINE, treatment is recommended; >5 cm should be considered in chronic TBAD if multistep procedure (FET + TEVAR) is panned	

practices will influence future recommendations regarding prophylactic CSF drainage during TEVAR for chronic dissection remains uncertain.

Adjunct techniques to evoke false lumen thrombosis are linked to lower rate of stent graft−induced new reentry tear (SINE) and less frequent reintervention rates without any difference in overall aortic-related mortality.[42] Importantly, no head-to-head comparisons have evaluated the advantage of these different adjunctive false lumen management techniques. These include

i) Knickerbocker technique: Following TEVAR, the dissection membrane is ruptured with a compliant-balloon at the midportion of the endograft to efface the stent graft to the outer wall of the aorta. This aims to induce false lumen obliteration and prevent retrograde false lumen flow.

ii) Provisional extension to induce complete attachment technique: Combines a more proximal TEVAR covered stent graft with a more distal uncovered bare-metal stent component that extends through the visceral aortic segment (e.g., zone 6 to 8) to induce aortic remodeling, and augment true lumen expansion. The STABLE trial showed favorable results in the acute and subacute settings in treating true lumen compression and visceral malperfusion.[43] However, the benefit of employing this adjunct in the treatment of CTBAD remains unproven.

iii) Stent-assisted balloon-induced intimal disruption and relamination in AD repair): In addition to the provisional extension to induce complete attachment technique, a balloon is used to rupture the dissection membrane to induce false lumen obstruction and improve aortic remodeling. A meta-analysis which included patients with acute, subacute, and chronic TBAD reported a 6% in-hospital mortality rate, 4% rate of intraoperative aortic rupture, and a late reintervention rate of 11%.[44]

iv) False lumen embolization: In addition to the implantation of a thoracic endograft, a dedicated embolic material may be used to induce false lumen thrombosis. A multicenter study reported complete false lumen thrombosis 77% with remodeling achieved in 47% of the cases, while 30-day mortality was 3%.[45]

Importantly, F/BEVAR for postdissection aortic aneurysms has demonstrated acceptable perioperative and mid-term outcomes, though the rates of SCI and reintervention remain high. Notably, current guidelines do not provide general recommendations on this issue.

3. **Imaging Modalities and Surveillance:** Four-dimensional flow MRI can be used in patients with chronic TBAD to assess hemodynamics, flow patterns, and provide information on wall shear stress.[45] Although its image quality is inferior compared to conventional 3D MRI, it has been shown to improve risk prediction for aortic growth and disease progression.[46,47] Advanced imaging techniques are promising; however, more clinical experience and standardized methods are needed before they can be adopted for routine use.

Life-long imaging surveillance is essential, regardless of the treatment method. Guidelines generally recommend more frequent imaging during the first year, followed by annual surveillance for 3−5 years, which may be extended to 2−3 years if the aortic pathology is stable (Table III). The ESVS guideline recommends closer follow-up or considering intervention for patients with an aortic diameter >50 mm or an annual growth rate >0.5 mm. In patients with connective tissue disease, the threshold for operative intervention is typically reduced to an aortic diameter >45 mm and/or annual growth rate >5 mm.

Adequate cardiac rehabilitation and mental health screening are vital for patients undergoing aortic reconstruction to prevent deterioration in cognitive and social functions and overall quality of life. Evidence on physical exercise and long-term aortic-related risks is limited. Nonetheless, avoiding heavy lifting (especially activities involving Valsalva maneuvers) is recommended due to the risk of increased systolic blood pressure. Conversely, light weightlifting and low-intensity aerobic exercises are safe and can improve physical and mental health. Patients should be educated on the cardioprotective benefits of mild-to-moderate−intensity exercise.[48−51]

CONSENSUS AREAS AND BEST PRACTICES—FROM GUIDELINES TO CLINICAL PRACTICE AND WHAT DOES THE CURRENT EVIDENCE TELL US?

Notwithstanding the enormous progress that has been made over the past 2 decades, many critical questions related to prognostication, diagnosis, management, and follow-up of TBAD remain unanswered, leading to either heterogeneity in recommendations or silence in the existing guidelines. Indeed, despite these advancements, significant gaps persist in the understanding and treatment of AD. Evidence-based clinical practice guidelines for diagnosis and management of TBAD serve a critical role in summarizing state-of-the-art research and providing strategies for its integration into clinical care. However, guideline documents also provide an important mechanism for the identification of areas in the published reports for which data are inconsistent and associated recommendations may be limited or conflicting. Given the intrinsically multidisciplinary nature of TBAD care, harmonization of practice recommendations and research priorities across the various evidence-based clinical practice guidelines will ultimately represent a key step in reducing heterogeneity of care and improving outcomes on a patient and population level.

Notably, the various societal guidelines for managing acute and chronic AD reflect increasing alignment in clinical practice, driven by advancements in diagnostic imaging, therapeutic interventions, and multidisciplinary care. All guidelines emphasize early diagnosis using advanced imaging, particularly CT angiography, which is widely regarded as the gold standard.

For acute TBAD, there is strong consensus that endovascular repair is the preferred treatment for complicated cases involving clinically evident malperfusion or rupture. UTBADs are uniformly managed with medical therapy focused on strict blood pressure and heart rate control, alongside long-term imaging surveillance to monitor disease progression. However, recommendations diverge on the management of "high-risk" uTBAD, with varying thresholds for early TEVAR across different guidelines.

In the chronic phase, guidelines differentiate between stable and progressive dissection. Stable cases are often managed conservatively, while progressive cases—especially those with aneurysmal degeneration or persistent symptoms—may require intervention. Complex endovascular techniques are increasingly supported for treating late aneurysmal degeneration in chronic TBAD, though their long-term outcomes warrant further study.

Another shared theme across all guidelines is the endorsement of multidisciplinary, team-based approaches, involving cardiothoracic surgeons, vascular surgeons, radiologists, and anesthesiologists. Additionally, the SVS/STS reporting standards

emphasize the importance of standardized terminology and dissection characterization, enabling better cross-comparison of clinical trials and the development of a stronger evidence base for future guideline updates.

GAPS IN KNOWLEDGE AND FUTURE DIRECTIONS—FROM TODAY TO TOMORROW: WHAT FUTURE EVIDENCE IS NEEDED?

Acute aortic syndromes remain rare and challenging pathologies, where early suspicion and accurate diagnosis are critical for timely treatment. The variability in patient symptoms often delays disease recognition, emphasizing the need for dedicated multidisciplinary ''aortic teams.'' These teams, widely endorsed by most guidelines, are essential for selecting the optimal care pathway based on careful evaluation of guideline recommendations tailored to individual cases.

A key challenge is the absence of reliable biomarkers or genetic markers to predict dissection risk and progression, limiting the potential for personalized treatment. Another contentious issue is the optimal timing for intervention in uTBAD. Current evidence largely stems from retrospective studies or small observational cohorts, leaving this question unresolved. Similarly, long-term comparisons between endovascular repair and continued medical management require further investigation to clarify durability and outcomes.

Emerging technologies offer hope for addressing these gaps. Advanced imaging modalities, such as PET/MRI, may enhance diagnostic precision and monitoring. Patient-specific stent grafts tailored to individual anatomy and computational tools like artificial intelligence for diagnosis and risk stratification hold promise but need rigorous clinical validation. Innovations in integrating clinical, imaging, and laboratory biomarkers could enable more accurate prognostication, identifying subgroups likely to benefit most from specific interventions in terms of timing and modality.

The role of social determinants of health, along with sex and ethnoracial differences, also demands further exploration. Distinguishing biologically distinct pathways from disparities attributable to healthcare access will be crucial for developing population-level mitigation strategies. Prospective, multicenter, randomized studies remain essential for resolving uncertainties in treatment timing, particularly for chronic dissections. Expanding registries to capture more diverse populations and rare complications will further enrich the evidence base for future guidelines.

Notably, sex-based analyses have revealed significant disparities in outcomes that remain poorly understood. Targeted research is needed to address these differences and ensure equity in care. Furthermore, studies investigating the optimal duration and intensity of medical therapy and imaging surveillance for patients with TBAD—both conservatively managed and postintervention—will be highly informative. Finally, the pace of technological advancement poses challenges for guideline updates to remain relevant. Endovascular procedures, fusion imaging technologies, surgical staging strategies, and genetic testing are reshaping clinical practice. However, gaps persist in understanding how these advancements impact long-term outcomes. Ongoing randomized trials, such as the Scandinavian Trial of Uncomplicated Dissection Therapy, the IMPROVE-AD trial, and the Early Aortic Repair in Patients Needing Endovascular/Open Surgery for Type B Aortic Dissection, aim to address these critical questions, potentially informing the next generation of clinical practice.

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