Perioperative Medical Management of Aortic Dissection

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KEYWORDS

• Aortic dissection • Perioperative medical management • Anti-impulse therapy
• Malperfusion syndrome • Type A versus type B dissection • Blood pressure and heart rate control

KEY POINTS

- Acute aortic dissection requires immediate medical stabilization alongside surgical or endovascular planning to prevent propagation and organ injury.
- Classification systems (Stanford, DeBakey, Penn, and chronicity) guide risk stratification, management strategy, and prognosis.
- Early diagnosis with computed tomography (CT) angiography or transesophageal echocardiography and vigilant monitoring for malperfusion and rupture are critical to survival.
- Anti-impulse therapy with strict heart rate and blood pressure control forms the cornerstone of management but must be individualized in malperfusion states.
- Optimal outcomes depend on multidisciplinary, protocol-driven perioperative care with timely intervention and structured long-term follow-up.

INTRODUCTION

Surgical or endovascular repair is the cornerstone of interventional management for acute ascending aortic dissection and complicated descending aortic dissection. However, perioperative medical therapy plays a decisive role in stabilizing patients, preventing propagation of dissection and mitigating end-organ injury. Perioperative clinicians must navigate competing priorities: reducing aortic shear stress while maintaining adequate perfusion, managing severe pain and sympathetic activation, addressing malperfusion syndromes, and preparing patients for urgent or emergent intervention. This article outlines a practical framework for perioperative medical management across the continuum of care.

Definition and Classification

Aortic dissection occurs due to a disruption of the aortic intimal layer, permitting blood to enter the medial layer, creating a true and false lumen. Classification systems have been created to describe overall risk and guide management. The Stanford system categorizes dissections into Type A, involving the ascending aorta, and Type B, confined to the descending aorta. The DeBakey system further subdivides dissections based on the site of origin and extent. Additional stratification based on ischemic pattern or pathophysiologic condition at presentation is addressed by the Penn classification, which subdivides patients into those with absence of malperfusion or circulatory collapse (Class A), presence of branch-vessel

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Abbreviations

ACE	angiotensin converting enzyme
ARBs	angiotensin receptor blockers
DOACs	direct oral anticoagulants
ECMO	extracorporal membrane oxygenation
IRAD	International Registry of Acute Aortic Dissection
MAP	mean arterial pressure
PCC	prothrombin complex concentrates
ROTEM	rotational thromboelastometry
SBP	systolic blood pressure
STS	Society of Thoracic Surgeons
SVS	Society for Vascular Surgery
TEE	transesophageal echocardiography
TEG	thromboelastography
TEVAR	Type B dissection randomized toendovascular repair
VA ECMO	veno-arterial extracorporal membrane oxygenation
VKA	Vitamin K antagonists

malperfusion (Class B), presence of circulatory collapse (Class C), and presence of both branch-vessel malperfusion and circulatory collapse (Class B + C).[1]

These classification systems are associated with clinical outcomes, as dissections involving the ascending aorta demonstrate improved survival with surgical repair, whereas dissections limited to isolated, uncomplicated descending dissection do well with medical management alone.[2]

Epidemiology and Incidence

The estimated incidence of aortic dissection ranges from 3 to 6 cases per 100,000 person-years, with a male predominance and peak incidence in the sixth to seventh decades of life. Hypertension remains the most prevalent risk factor, while connective tissue disorders, aortic aneurysm, bicuspid aortic valve, pregnancy, and prior cardiac surgery represent important predisposing conditions. Approximately two-thirds of dissections involve the ascending aorta with chest pain being the most common presenting feature.[3]

Pathophysiology and Natural History

Rather than occurring randomly, dissections are more common in winter months and early morning hours, when blood pressure is known to be highest, due to physiologic responses related to circadian and diurnal physiologic patterns.[2] The inciting event, therefore, is commonly related to a physiologic response in the setting of an *at risk* substrate resulting in a force exceeding the tensile limits of the aortic wall. The 3 most common sites for dissection tears to occur are approximately 2 to 2.5 cm above the aortic root (most common), just distal to the origin of the left subclavian artery, and within the aortic arch.[4]

Without intervention, acute Type A dissection carries a mortality approaching 1% to 2% per hour during the first 24 to 48 hours.[5] Complications include aortic rupture, coronary ischemia, cardiac tamponade, aortic regurgitation and end-organ malperfusion.[6]

Aortic rupture occurs less frequently in Type B than Type A dissections; however, rupture remains the primary cause of death in this cohort, with malperfusion being the second most common cause. Despite this, early survival for patients with Type B dissections is around 90%, and roughly two-third of patients can be managed with medical therapy alone.[2]

Importance of Timely Diagnosis and Management

Early diagnosis, rapid blood pressure and heart rate control, and timely surgical or endovascular intervention are critical determinants of survival. Optimal perioperative management—including early hemodynamic control and coordinated multidisciplinary care—has been shown to significantly improve operative outcomes and reduce complications in patients with acute aortic dissection.[7]

CLASSIFICATIONS AND IMPLICATIONS FOR MANAGEMENT

Stanford Type A versus Type B

Type A dissection is a surgical emergency requiring urgent surgical intervention due to the risk of catastrophic complications. Mortality of medical management is 2 to 3 fold higher than with surgical intervention, though surgical mortality still remains substantial at 15% to 30%. Type B dissection can typically be medically managed with aggressive blood pressure and heart rate control unless complications arise. Thirty-day mortality for medically managed type B dissections is approximately 10%.[5]

Non-A Non-B Aortic Dissection Management

Dissection involving the arch without ascending aortic involvement represents a heterogeneous group requiring individualized management, often incorporating hybrid surgical and endovascular approaches.

Complicated versus Uncomplicated Type B Dissection

Complicated Type B dissection is defined by malperfusion, rupture or impending rupture, continued growth or extension of the dissection, refractory pain, or recalcitrant hypertension. In-hospital mortality in these cases approaches 30% to 40% if not intervened upon. These cases require endovascular or surgical repair, with endovascular stent grafting being recommended over open surgical repair when anatomy is suitable.

Routine immediate intervention for uncomplicated Type B dissection is not recommended due to the higher risk of procedural complications compared with medical management. While endovascular repair performed after a stabilization period seems to ultimately provide benefit, optimal timing remains uncertain.

Acute versus Chronic Dissection

Chronicity classifications for aortic dissection were updated by the Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) in 2020.[8] These classifications now range from hyperacute (<24 hours since symptom onset) to chronic (more than 90 days since initial presentation) (**Table 1**).[8] Acute dissections (1–14 days from symptom onset) are associated with higher risk of rupture.

Increasing evidence supports selectively intervening in subacute or chronic Type B dissection in patients with high-risk features for late adverse aortic events (**Table 2**).

The INSTEAD-XL trial was a long-term follow-up of patients with stable uncomplicated Type B dissection randomized to endovascular repair (TEVAR) versus optimal medical therapy. This trial demonstrated lower aorta-specific mortality and reduced disease progression with TEVAR.[9] Similarly, the ADSORB trial which compared optimal medical management to medical management plus TEVAR, showed improved false lumen thrombosis and reduced aortic dilation with TEVAR, despite no early mortality difference.[10]

Table 1
Chronicity classification for aortic dissection

Chronicity Classification	Time From Symptoms Onset
Hyperacute	< 24 h
Acute	1–14 d
Subacute	15–90 d
Chronic	> 90 d

Table 2
Features of aortic dissection with high risk for late adverse aortic events

High Risk Feature	
• Aortic diameters > 40 mm	• Entry tear > 10 mm or lesser curve location
• Rapid aortic expansion	• Refractory pain
• False lumen > 22 mm	• Refractory hypertension
• Patent false lumen or partial thrombosis	• Bloody pleural or pericardial effusion

Penn Classification

The Penn classification predicts hospital mortality in both Type A and Type B dissections. In the European Registry of Type A Aortic Dissection, mortality rates were 12.4% for Penn A, 20.7% for Penn B, 28.9% for Penn C, and 31.8% for Penn B + C.[11] Beyond mortality prediction, this classification informs anesthetic complexity and perioperative resource utilization, such as bleeding and transfusion requirements.[12] As further data is added to our collective experience, it is becoming evident that a more nuanced management strategy is required in the setting of preoperative malperfusion, which incorporates the full ischemic profile. This is reflected in both the recent 2024 European and 2022 American guidelines for aortic diseases.[5,13]

CLINICAL PRESENTATION AND DIAGNOSIS

Common Symptoms and Physical Findings

The most common presenting symptom of acute aortic dissection is severe pain with abrupt onset.[14] Although a small percentage of patients (~10%) present without pain, symptoms typically correlate with the longitudinal extent of the dissection. However, many patients present atypically, making aortic dissection diagnostically challenging and earning it the nickname of *the great masquerader*. Accordingly, a high index of suspicion is needed in patients presenting with symptoms mimicking heart attack, stroke, acute abdominal conditions or limb ischemia when no alternative explanation is evident.

Physicians should have a heightened level of suspicion of aortic dissection in patients with Marfan syndrome or other connective tissue disease, family history of aortic disease, known aortic valve pathology, recent aortic manipulation, or known thoracic aortic aneurysm. Physical examination

features include pulse deficits, systolic blood pressure differential, focal neurologic deficits, new-onset aortic regurgitation murmur, and hypotension or shock state.[5] Additional physical examination findings may include muffled heart sounds, features of Horner syndrome (ptosis, miosis, and anhidrosis) due to involvement of carotid or subclavian arteries, or hoarseness from recurrent laryngeal nerve compression. Dyspnea and hemoptysis may occur secondary to rupture into the mediastinum.[4]

Hemodynamic Instability

Hypotension may reflect cardiac tamponade, aortic rupture, or severe acute aortic regurgitation. Conversely, hypertension —either due to poorly controlled baseline hypertension or sympathetic activation related to pain and stress—is also common and may accelerate propagation of the dissection.

Laboratory Evaluation

Initial laboratory assessment is not diagnostic but is a useful adjunct and can help assess clinical status and contribute to risk stratification. Elevated D-dimer levels greater than 500 ng/mL are highly sensitive for dissection and may help to rule out dissection in low-risk patients.[4] Elevated troponin levels may indicate coronary artery involvement or suggest demand ischemia due to shock state. Rising creatinine may indicate renal ischemia from malperfusion. Additional laboratory work should include complete blood count, comprehensive metabolic panel, coagulation studies and serum lactate.

Malperfusion Syndrome

Malperfusion syndrome occurs when the aortic dissection flap compromises blood flow to major branch vessels, resulting in impaired tissue perfusion. Preoperative malperfusion is present in approximately 25% to 30% of patients with acute Type A dissection and most commonly involves extremity, renal, and cerebral vascular beds. Its presence is associated with significantly worse outcomes, with the highest mortality observed among patients with coronary malperfusion.[15]

Malperfusion may arise from dynamic obstruction, in which pressurization of the false lumen compresses the true lumen or intermittently occludes branch vessel ostia, or from static obstruction, caused by extension of the dissection flap into branch vessels with fixed luminal compromise. These distinct mechanisms have important perioperative implications, as excessive hypotension may exacerbate ischemia, whereas uncontrolled hypertension increases the risk of aortic rupture.[7]

Organ malperfusion warrants prompt surgical or endovascular intervention. Fenestration of the intimal flap can restore distal flow by decompressing the false lumen; however, fenestration alone may be insufficient, with up to 75% of patients requiring adjunctive endovascular therapies, such as branch-vessel stenting, to achieve durable reperfusion.[16]

In select circumstances—particularly in patients with mesenteric malperfusion—endovascular fenestration of the intimal flap followed by reperfusion and delayed aortic repair is a reasonable approach. This decision may be guided by the severity of ischemia, duration of malperfusion, and degree of lactic acidosis.[17] Such interventions should generally be performed within 24 to 48 hours, before the dissection membrane becomes fixed or false lumen thrombus becomes organized.

Overall, malperfusion complicates up to 30% to 40% of acute dissections, most frequently affecting the extremities (~15%), kidneys (~10%) and brain (~10%).[11,15] Its impact on outcomes is substantial, nearly doubling operative mortality (26.8% vs 13.6%), especially if involving the coronary or mesenteric circulations.[15]

Diagnostic Imaging Modalities

Plain chest X-ray is nonspecific and nondiagnostic but may raise suspicion of dissection in the setting of mediastinal widening, disruption of the normally distinct contour of the aortic knob, separation of the intimal calcification from the aortic wall of greater than 5 mm (*calcium sign*), double density appearance within the aorta, tracheal deviation to the right, or deviation of a nasogastric tube to the right.[5]

Computed tomography (CT) angiography (CTA) is the first-line diagnostic imaging modality due to rapid acquisition and its high sensitivity and specificity. CTA reliably delineates the extent of dissection, identifies branch-vessel involvement, and may localize the primary entry tear. It also detects complications such as malperfusion, pericardial effusion, periaortic hematoma, and pleural effusion.[5]

Transesophageal echocardiography (TEE) is particularly valuable in patients with iodinated contrast allergy, in the intraoperative setting and in unstable patients. While transthoracic echo may provide rapid, noninvasive assessment, TEE is preferable due to improved imaging quality and higher sensitivity. TEE enables real-time assessment of valve and cardiac function, pericardial fluid, true lumen flow, and can also assess coronary and branch vessel perfusion.

MRI is a reasonable alternative for initial diagnostic imaging, particularly in patients with contrast allergy, and provides excellent spatial resolution, but is limited by availability and patient stability. As such, MRI is typically the third choice of imaging modality.[5]

Aortography is rarely used and not recommended given its invasiveness and lower sensitivity compared with other imaging modalities.

GOALS OF MEDICAL MANAGEMENT

All patients with acute aortic syndromes require immediate medical therapy while determining next steps in treatment and considering the need for interventional therapies. Preoperative care focuses on hemodynamic stabilization and comprehensive systemic monitoring to identify complications that may influence the timing, urgency, and strategy of surgical or endovascular repair (Fig. 1).

Blood Pressure and Heart Rate Reduction

The primary hemodynamic determinant of dissection propagation is aortic wall shear stress, which is proportional to both the rate of rise of systolic pressure (dP/dt) and heart rate. Sympathetic activation driven by pain and anxiety exacerbates hypertension and tachycardia, thereby accelerating false lumen expansion and increasing the risk of rupture or malperfusion.

Accordingly, the cornerstone of medical management involves aggressive control of heart rate and blood pressure, an approach that has been associated with reductions in both early and long-term adverse events. Heart rate targets fall in the range of 60 to 80 beats per minute.[18] Systolic blood pressure (SBP) goals should be individualized based on patient-specific factors, including end-organ perfusion and clinical tolerance. An initial SBP goal of less than 120 mm Hg is commonly pursued, though this may require liberalization or further restriction depending on clinical context (Fig. 2). Following thoracic endovascular aortic repair of Type B dissection, an SBP less than 130 mm Hg has been associated with lower risk of aortic related adverse events.[19]

Hemodynamic Management in Setting of Malperfusion

Hemodynamic management becomes substantially more complex in the presence of malperfusion, where the competing risks of aortic propagation and end-organ ischemia must be simultaneously balanced. While anti-impulse therapy with reduction of systolic blood pressure (<120 mm Hg) and heart rate (60–80 bpm) remains the default strategy in uncomplicated dissection, this paradigm requires modification in patients with dynamic malperfusion.

In patients with Type A dissection without malperfusion (Penn Class A), aggressive reduction in

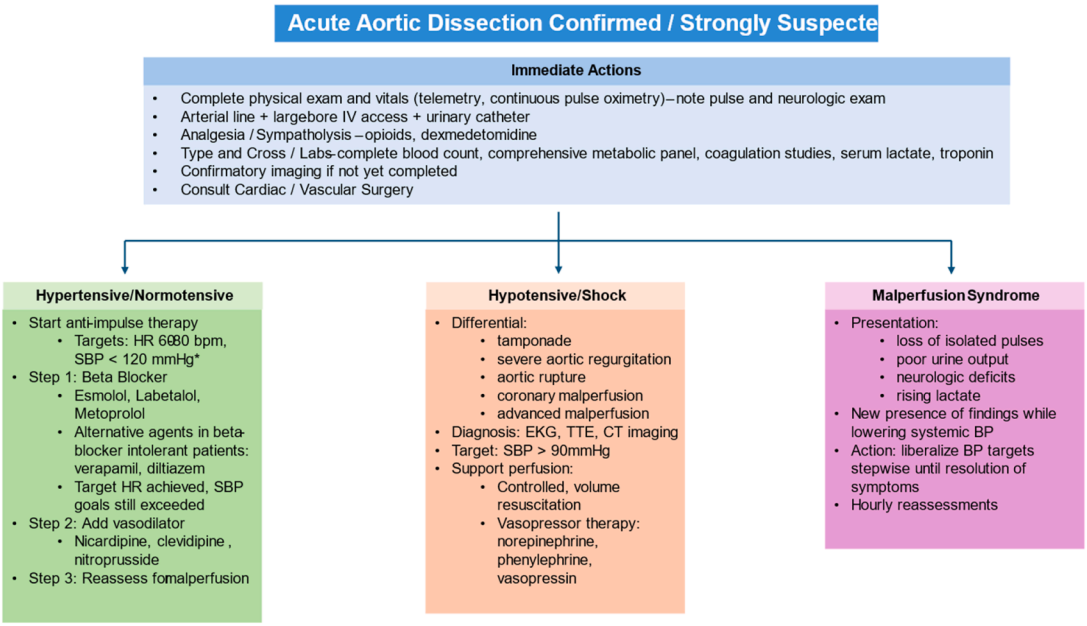

Fig. 1. Stepwise algorithm for initial medical management of aortic dissection. * Monitor for signs of malperfusion or clinical deterioration as target blood pressure is acheived

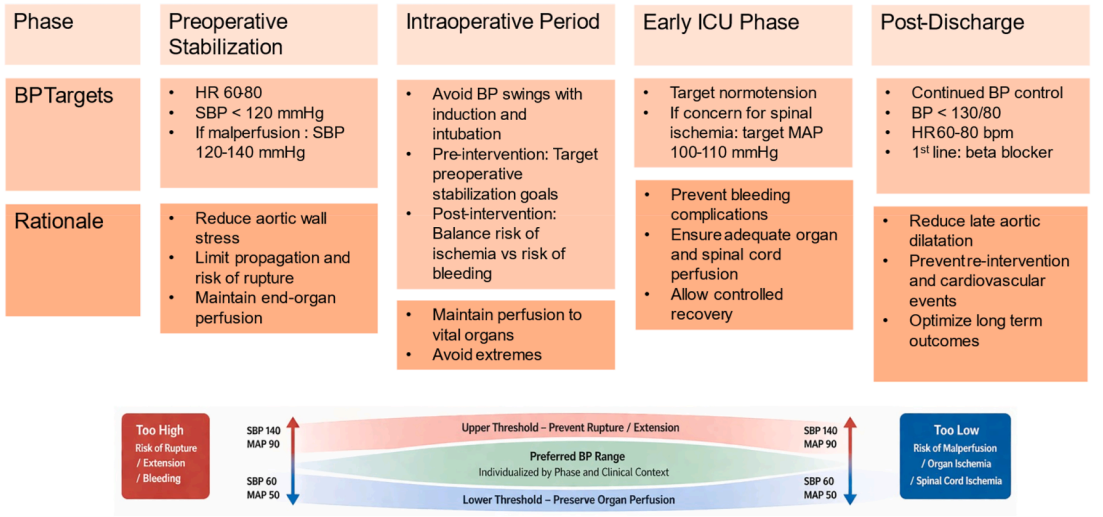

Fig. 2. Blood pressure management strategy across the perioperative continuum.

shear stress remains the primary objective. In contrast, in Type A dissection with dynamic malperfusion (Penn Class B or B + C), excessive blood pressure reduction may worsen true lumen collapse and exacerbate ischemia, particulary in vascular beds supplied by dynamic obstruction. In this context, a more permissive blood pressure strategy is often required, with systolic blood pressure targets individualized (often 120–140 mm Hg) to preserve end-organ perfusion while avoiding uncontrolled hypertension.

A practical framework is to prioritize *perfusion* over strict anti-impulse targets when there is clear evidence of ongoing ischemia (eg, rising lactate, oliguria, neurologic deficits), while maintaining heart rate control to limit shear stress. Serial reassessment is essential, as the hemodynamic profile may evolve rapidly. Hourly (or even more frequent) measurement of lactate levels, urine output, and physical neurovascular assessment can help guide hemodynamic targets. Definitive correction of malperfusion—via surgical repair or endovascular intervention—remains the cornerstone of management, and medical therapy should be viewed as a temporizing strategy rather than a definitive solution in this population.

Pain Control and Sympatholysis

Pain may function both as a cause and as a consequence of adverse hemodynamic responses. Uncontrolled pain can precipitate increases in heart rate and blood pressure, while effective blood pressure control may itself help to alleviate some degree of pain. Effective pain control therefore is a cornerstone of management, as untreated pain drives sympathetic activation, resulting in tachycardia, hypertension, and increased aortic wall stress. Intravenous opioids remain first-line therapy and should be titrated aggressively to achieve both analgesia and hemodynamic control.

Adjunctive use of dexmedetomidine may be particularly advantageous in this population, as it provides both sedation and sympatholysis, reduces catetcholamine surge, and may decrease opioid requirements while facilitating hemodynamic stability.

In contrast, ketamine should generally be avoided in the acute setting, given its sympathomimetic effects, which may increase heart rate, blood pressure, and myocardial oxygen demand—potentially exacerbating dissection propagation.

Nonsteroidal anti-inflammatory medications should likely be avoided due to the risks of platelet inhibition and bleeding, renal dysfunction, and hypertension. Overall, pain management should be viewed not simply as symptomatic treatment but as a critical component of anti-impulse therapy.

Preoperative Monitoring and Access

Advanced monitoring is essential for identifying end-organ malperfusion, cardiorespiratory compromise, and biochemical changes that are associated with adverse outcomes while informing perioperative risk stratification and optimization.

Continuous hemodynamic and respiratory monitoring is critical given the high incidence of preoperative instability. Invasive arterial blood pressure monitoring and continuous heart rate monitoring facilitates precise anti-impulse therapy and early detection of end-organ ischemia. Hypoxemia and respiratory compromise are common occurring in up to 50% of patients with acute Type A dissection—and are independently associated with worse

perioperative outcomes.[20] Preoperative evaluation and management of oxygenation and pulmonary complications should be incorporated into routine care.

Laboratory and biochemical markers increasingly contribute to perioperative risk stratification. Elevated lactate levels have been linked with mesenteric malperfusion and higher mortality. Serial biomarkers of end organ function should be trended to monitor for worsening function and offer insight into systemic inflammation and tissue hypoperfusion that may be dynamic or may not be readily apparent on imaging alone.

Large bore peripheral venous access and/or central venous access should be established early in anticipation of sudden hemodynamic deterioration and for procedural intervention. Preparation for the operating room typically includes central access for assessment of volume status and administration of vasoactive medications, and often incorporates pulmonary artery catheterization with cardiac output monitoring. Placement of a urinary catheter for hourly urine output measurement facilitates early detection of oliguria or anuria, which may indicate renal hypoperfusion or hypovolemia. Availability of blood products—including red blood cells, platelets, plasma, and cryoprecipitate—should be ensured before heading to the operating room.

Identification of Complications

Aortic dissection carries the potential for a wide spectrum of complications. Complications arise primarily through 2 mechanisms: extension of the dissection flap into branch vessels causing malperfusion, and rupture into adjacent structures leading to catastrophic bleeding. Early identification of these complications requires an integrated approach incorporating clinical assessment, advanced imaging and targeted laboratory assessment.

Clinical manifestations such as focal neurologic deficits, pulse deficits, limb ischemia, abdominal pain, anuria, syncope or shock may reflect underlying malperfusion or rupture. Frequent neurovascular checks—particularly during initiation and titration of blood pressure control—are essential for detecting dynamic changes in perfusion. Sudden hypotension or syncope should raise immediate concern for cardiac tamponade or aortic rupture and warrants urgent evaluation.

PHARMACOLOGIC THERAPY

No randomized controlled trials have evaluated specific medical therapies in acute dissection. Nonetheless, extensive clinical experience and observational data have informed contemporary pharmacologic management strategies. Intravenous medications with shorter half-lives that allow rapid titration are preferable to longer acting agents in the acute and perioperative setting.

Beta Blockers

Beta blockers are the most commonly used and recommended medications for initial treatment. These agents achieve impulse control by decreasing heart rate and systolic blood pressure, thereby decreasing aortic wall stress, and have been associated with improved survival in both operative and nonoperative patients.[21] No single beta blocker has demonstrated superiority over others. Commonly used agents include esmolol, labetalol and metoprolol. Beta blocker therapy should be used cautiously in patients with bradycardia, acute severe aortic regurgitation or high-grade atrioventricular block.

Adjunctive Agents

Intravenous vasodilators are often added to beta blocker therapy once heart rate goals are achieved but hypertension persists. Due to the risk of compensatory tachycardia with initiation of vasodilator medications, these should be avoided as initial treatment. In a small retrospective study, clevidipine and sodium nitroprusside showed similar efficacy as adjuncts to beta blocker therapy in reaching systolic blood pressure targets.[22] No specific vasodilator is preferred; commonly used agents include nicardipine, clevidipine and sodium nitroprusside.

Analysis of the International Registry of Acute Aortic Dissection (IRAD) demonstrated that calcium channel blocker use was associated with improved survival among medically managed patients with Type B dissection.[21] Intravenous nondihydropyridine calcium channel blockers may also be considered in patients intolerant of beta blockers, with verapamil or diltiazem being the most frequently used agents in this context.

Angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are useful in long term hypertension management but have little role in the acute management of patients in the perioperative period. In the IRAD database, ACE inhibitor use was not associated with reduced short-term mortality.[21] However, long-term follow-up data suggests that ACE inhibitors and ARB therapy is associated with lower all-cause mortality.[23]

Vasopressor Therapy in Hypotension and Shock

Although hypertension predominates in acute aortic dissection, a subset of patients—particularly those with cardiac tamponade, severe aortic

regurgitation, myocardial ischemia, or advanced malperfusion (Penn Class C or B + C)—present in shock. In these cases, restoration of adequate perfusion pressure takes precedence as hypotension is independently associated with increased in-hospital mortality and neurologic events.[6]

Initial fluid resuscitation should be titrated to blood pressure response, cardiac preload and cardiac output with a goal of ensuring adequate end-organ perfusion. Permissive hypotension, with a systolic pressure target of 90 mm Hg, has been recommended, as mortality increases lesser than this threshold.[6] However, vasopressor therapy should be initiated when volume status has been optimized and hypotension or malperfusion persists.

No definitive recommendations exist regarding optimal vasopressor choice. Phenylephrine and vasopressin are attractive options for increasing vascular tone without direct augmentation of cardiac contractility. Phenylephrine may also confer the additional benefit of reflex bradycardia, thereby reducing heart rate and minimizing aortic wall stress. However, both agents should be used cautiously in patients with impaired ventricular function or low cardiac output, as they may further reduce forward flow.

Norepinephrine may be preferred in many cases due to its balanced alpha- and beta-adrenergic effects, which support both vascular tone and cardiac output. Pure inotropes, such as epinephrine or dobutamine, should generally be avoided, as they can increase aortic wall stress and myocardial oxygen demand while providing less augmentation of systemic vascular resistance.

Management of Anticoagulants and Antiplatelet Medications

No specific interventions have been shown to reverse the coagulopathy associated with acute aortic dissection. However expediting surgery and limiting tissue factor exposure may help to limit dissection-specific coagulopathy. Low fibrinogen levels and thrombocytopenia at admission are associated with worse outcome. However, the prognostic value of viscoelastic testing is unclear despite relying heavily on platelet and fibrinogen measurements.[24] Preemptive administration of blood products or coagulation factors is not recommended.

Measurement of international normalized ratio is sufficient for patients on vitamin K antagonists (VKA). For urgent reversal, administration of 4-factor prothrombin complex concentrates (PCC) at a dose of 12.5 to 25 U/kg bodyweight is preferred.[25] Given the increased risk of thromboembolic complications, the lower dose of PCC is generally favored and can be considered after heparin reversal with protamine rather than before cardiopulmonary bypass.[24] Adjunctive vitamin K administration may facilitate faster postoperative recovery of vitamin K-dependent coagulation factors. Although fresh frozen plasma contains vitamin K dependent clotting factors, the volume required to reverse therapeutic VKA—approximately 8 units of plasma in a 70 kg patient—renders it impractical for rapid reversal.[18]

For patients on direct oral anticoagulants (DOACs), prolonged prothrombin time and activated partial thromboplastin time suggests elevated drug levels.[24] However, values in normal ranges do not exclude clinically relevant DOAC concentrations (greater than 30 ng/mL). Drug-specific assays are therefore preferred. For factor Xa inhibitors (such as rivaroxaban, apixaban, or edoxaban), a drug-specific calibrated anti-Xa test or a factor Xa assay calibrated for unfractionated heparin can reliably estimate the antifactor Xa activity.[24] Use of andexanet alfa is not recommended preoperatively as it interferes with heparinization for cardiopulmonary bypass.[26]

For patients on factor IIa inhibitors (dabigatran), use of diluted thrombin time is recommended to optimally estimate drug levels; a normal thrombin time effectively excludes clinically relevant drug levels.[24] Idarucizumab may be administered, given its approximately 350-fold greater affinity for dabigatran compared with thrombin. In surgical patients, normal hemostasis has been reported in 92% of cases following reversal, with a 6% incidence of postreversal thrombotic events.[18] If idarucizumab is unavailable, PCC may be considered.

Management of recent antiplatelet therapy other than aspirin, particularly potent P2Y12 inhibitors such as clopidogrel, prasugrel or ticagrelor, remains challenging. Routine use of point-of-care platelet function testing and preemptive platelet transfusion is generally not recommended.[24]

POSTOPERATIVE TRANSITION

Immediate Intensive Care Unit Care

Postoperatively, patients require management in a specialized intensive care unit setting with continuous hemodynamic monitoring to detect complications such as bleeding, renal failure, or stroke. Early recognition and intervention are critical for improving outcomes.

Surveillance for Complications

Surveillance for complications or ongoing ischemia begins in the operating room. Assessment for distal malperfusion following aortic repair,

often using vascular imaging, can guide interventions such as angioplasty, stenting, and open bypass.[12]

Renal dysfunction may be mitigated through careful blood pressure control, avoiding extremes of blood pressure, and prompt resuscitation for ongoing bleeding. Volume overload resulting from large volume resuscitation and venous hypertension may further impair renal perfusion; therefore, readiness for diuresis should be assessed on an ongoing basis.

Volume overload, pleural effusions or hemothorax, and the inflammatory insult of dissection and surgery may contribute to pulmonary compromise. Lung protective ventilation strategies should be maintained while closely monitoring for worsening oxygenation and development of acute respiratory distress syndrome. Serial chest radiography can monitor progression of pleural effusions, pulmonary infiltrates, or consolidation.

Intraoperative cerebral oxygenation monitoring can be continued postoperatively until normothermia is achieved and sedation can be safely weaned for neurologic assessment. Patients undergoing intraoperative circulatory arrest are at increased risk for cerebral ischemia due to hypoperfusion or embolic phenomena. Any intraoperative abnormalities in cerebral tissue oxygenation or neurologic monitoring (eg, EEG) should prompt consideration of CT angiography, potentially directly from the operating room if hemodynamics allow.

Spinal Cord Ischemia

Risk of spinal cord ischemia, one of the most feared complications of aortic repair, requires vigilant monitoring, particularly in patients undergoing endovascular or open repair of the descending thoracic and thoracoabdominal aorta. The classic presentation of spinal cord ischemia in this setting includes loss of lower extremity motor function with preserved sensation. Although most cases occur in the immediate postoperative period, spinal cord ischemia may present days to weeks later. Early recognition and treatment are time-sensitive and aggressive measures should be taken promptly after onset of neurologic deficits or suspected spinal cord ischemia.

Interventions should focus on optimizing spinal cord perfusion. Specific interventions including augmentation of blood pressure (often targeting a mean arterial pressure (MAP) of 100–110 mm Hg prior to spinal drain placement), transfusion to a hemoglobin target of 10 g/dL, and placement of a spinal drain to target a cerebrospinal fluid (CSF) pressure of 10 mm Hg.[27] Spinal cord perfusion pressure (difference between MAP and the higher of intracranial pressure or central venous pressure) target of greater than 70 mm Hg is broadly recommended and achieved through driving MAP to 85-100 mm Hg and draining CSF for a goal pressure of 10 mm Hg. Given the time sensitive nature of ischemia, spinal drain placement should occur as fast as the team can safely achieve, recognizing diminishing probability of meaningful neurologic recovery the longer deficits persist.

Risks of overly aggressive CSF drainage include intracranial hemorrhage and herniation. Limiting rate and volume of CSF drainage both hourly and daily can help to mitigate this risk. Suggested limits include capping CSF drainage at 150 mL over 24 hours and not exceeding 10 to 20 mL over 1 hour.[28]

Transfusing to a hemoglobin level greater than 10 g/dL is uniformly recommended as a rescue maneuver to maximize spinal cord oxygen delivery. However, this is based primarily on theoretic benefit of increasing oxygen carrying capacity. No randomized trials have evaluated optimal hemoglobin targets in this setting and the benefits of transfusion must be weighed against the risks of volume overload, increasing central venous pressure, increased blood viscosity with higher hemoglobin values, and the noted association of transfusion on mortality.

Long-Term and Discharge Transition

Longer-term management follows general critical care principles with early mobilization and de-escalation of invasive catheters as soon as safe and feasible. Even after successful operative treatment, patients remain at risk for progressive aortic enlargement, aneurysmal degeneration of residual native aorta, malperfusion syndromes, recurrent dissection, need for reintervention, and cardiovascular morbidity from poorly controlled hypertension or vascular disease. Therefore, longitudinal management should be structured, multidisciplinary, and focused on aggressive risk reduction, serial imaging and durable engagement with specialized follow-up care.

Beta blockers remain first-line therapy as a cornerstone of chronic management, often in combination with ACE inhibitors or ARBs, particularly in patients with connective tissue disorders. In patients with heritable thoracic aortic diseases, observational and syndrome specific data suggest a benefit to ARBs specifically in slowing aortic enlargement. Exact targets should be individualized, but generally aim for SBP less than 120 to

130 mm Hg and resting heart rate around 60 beats per minute. Patients should be educated and encouraged to measure home blood pressures.

Surveillance imaging with CT or MRI is typically performed at 1, 3, 6, and 12 months, and annually thereafter, although intervals may be individualized based on aortic stability. More frequent imaging may be warranted if rapid aortic enlargement, persistent patent false lumen or a large residual dissection flap is seen on imaging. Patients should be counseled to avoid heavy exertion and activities associated with abrupt increases in blood pressure. Referral for genetic counseling should be considered in patients with suspected heritable aorthopathies. Smoking cessation should be strongly emphasized during patient counseling, as well as medication compliance, weight optimization, glycemic control, treatment of obstructive sleep apnea, moderation of alcohol use and regular medical follow-up.

A structured transition of care—from intensivist and surgical teams to outpatient cardiology and vascular specialists—is essential to ensure continuity. Clear documentation of dissection anatomy, operative or endovascular procedures performed, residual disease burden, blood pressure goals, medications, imaging schedule, and contingency planning for symptoms or abnormal findings is imperative for reducing fragmented and incomplete care.

POSTOPERATIVE HEMOSTATIC MANAGEMENT

Postoperative bleeding is common following type A repair and frequently necessitates extensive blood product transfusion. Dissection-associated coagulopathy is linked to hospital mortality rates approaching 20%.[29] Multiple factors contribute to bleeding risk, including tissue factor release, consumption of coagulation factors, hyperfibrinolysis, hypothermia, hemodilution, heparin exposure, preoperative antiplatelet and anticoagulant therapy, and preexisting coagulopathy. The resulting hemostatic derangement resembles disseminated intravascular coagulopathy, with particular depletion of platelets and fibrinogen.[24]

No single transfusion strategy has demonstrated superiority over others. Management varies according to institutional protocols, patient specific factors, and multidisciplinary team preferences. Protocolized approaches based on clinical criteria may reduce practice variability, while algorithm-driven strategies using viscoelastic testing may limit unnecessary allogeneic blood product exposure.[24] Perioperative hemostatic goals include establishing *coagulation-friendly* conditions, such as correction of acidosis, maintenance of normothermia, and correction of hypocalcemia.[24,27] The 2017 European and 2021 American guidelines for patient blood management in cardiac surgery recommend viscoelastic testing to guide coagulation factor administration.[30,31] These functional assays such as rotational thromboelastometry (ROTEM) and thromboelastography (TEG) provide real-time assessment of coagulation status by measuring the initiation, propagation, strength, and breakdown of clot formation in whole blood and assist in tailoring therapy with fresh frozen plasma, PCC, fibrinogen concentrate and platelets. These assays should be interpreted alongside conventional coagulation testing (prothrombin time/international normalized ratio, partial thromboplastin time, and fibrinogen).[27]

Specific assay parameters can help identify the dominant hemostatic defect and guide targeted therapy. Prolonged clot initiation or propagation times—such as increased clotting time on ROTEM, or prolonged R time on TEG—may reflect deficiency of coagulation factors, residual heparin effect, or severe dilutional coagulopathy and may prompt administration of plasma, prothrombin complex concentrate, or heparin reversal depending on the clinical context. Reduced maximum clot firmness on ROTEM or maximum amplitude on TEG indicates impaired overall clot strength, frequently due to thrombocytopenia or platelet dysfunction, and may guide platelet transfusion or consideration of desmopressin. In this same context, low functional fibrinogen assays (ROTEM FIBTEM or FF TEG) may support administration of cryoprecipitate for low clot strength. A reduced α-angle or low amplitude on fibrin-based assays suggests hypofibrinogenemia and may support treatment with cryoprecipitate or fibrinogen concentrate. Excessive clot breakdown—such as high L130 on ROTEM or LY30 on TEG—may indicate hyperfibrinolysis and support administration of antifibrinolytic agents such as tranexamic acid or aminocaproic acid.

For patients on factors Xa inhibitors with life-threatening postoperative bleeding, administration of adexanet alfa can be considered, taking into account the time since last dose, as utility may be limited beyond 12 hours. Additionally, andexanet alfa may complicate subsequent heparinization if reoperation is required. As of December 2025, andexanet alfa was withdrawn from the US market due to concerns regarding thromboembolic complications and thrombosis-related mortality, though it remains available in other markets.

CONTROVERSIES AND EVOLVING STRATEGIES

Decision Making in Borderline Cases

Predictors for postoperative mortality include coma, shock secondary to pericardial tamponade, coronary or peripheral malperfusion, and stroke. In octogenarians, in-hospital mortality was lower after surgery than with conservative management, although this finding failed to reach statistical significance. Age alone should not be considered an exclusion criterion for surgical intervention but should be considered in the overall context of decision making.[7] Clinical judgment remains paramount in patients with substantial contraindications, including frailty, where the risk-benefit profile may favor medical management.[18]

The appropriateness of surgery in patients presenting with Type A dissection and neurologic deficits or coma remains debated. Although these findings predict poorer postoperative outcomes, neurologic recovery is possible when cerebral perfusion is restored expeditiously, particularly when the interval from symptom onset to surgery is less than 5 hours.[32] IRAD data supports improved survival with surgical intervention in patients presenting with cerebral malperfusion or acute stroke.[33] However, patients with complete occlusion of an internal carotid artery may be at particularly high risk of cerebral edema and herniation and may not benefit from surgical intervention.[34]

Use of Extracorporal Membrane Oxygenation in Aortic Dissection

Use of peripheral veno-arterial extracorporal membrane oxygenation (VA ECMO) in acute aortic dissection remains controversial, primarily given the potential to worsen aortic injury. Preoperatively, VA ECMO may temporize patients with profound malperfusion or facilitate transfer to specialized centers; however, the literature on safety and efficacy is sparse.[35] A recent narrative review concludes that evidence remains insufficient to guide use of extracorporal membrane oxygenation (ECMO) preoperatively.[35] Similarly, a systematic review from 2019 found no compelling evidence for or against the use of ECMO in aortic dissection.[36]

Postoperatively, ECMO following repair of acute Type A dissection is associated with high in-hospital mortality (approximately 63%) and substantial complication rates.[37] In a systematic review and meta-analysis, Sá and colleagues found no association between retrograde arterial flow from peripheral VA ECMO and in-hospital mortality despite previous authors noting this as a risk factor.[37] The same analysis questions recommendations to avoid intra-aortic balloon pump support, citing a lack of evidence for dissection propagation following placement after repair.[37] Factors predictive of postoperative ECMO support include preoperative hemodynamic instability, myocardial infarction, longer aortic cross clamp and cardiopulmonary bypass time, inadequate myocardial protection, and dissection propagation into the coronary arteries.[36] Overall, the literature is shifting to suggest that extracorporeal life support may be considered as a salvage strategy rather than being outright avoided.

In both the preoperative and postoperative setting, imaging assessment of the iliac and femoral arteries is essential before cannulation, with preference given to avoiding dissected vessels. When cannulation of a dissected artery is unavoidable, true lumen access must be confirmed with imaging guidance.

SUMMARY

Perioperative management of aortic dissection remains complex and dynamic, requiring continuous balance among competing physiologic priorities and rapid adaptation to evolving hemodynamic and perfusion states. Although surgical repair continues to be a cornerstone of treatment, advances in endovascular and hybrid techniques have revolutionized treatment paradigms, particularly for Type B dissection.

This article underscores that optimal care cannot rely on rigid algorithms alone. While foundational principles—anti-impulse therapy, timely intervention, vigilant monitoring, and aggressive treatment of complications—apply broadly, the most consequential decisions are often highly individualized. Patients presenting with branch vessel malperfusion, tamponade physiology, severe aortic regurgitation, refractory shock, or delayed neurologic injury frequently require deviation from standard pathways and nuanced prioritization of immediate threats. In these scenarios, expert care lies not only in knowing the guideline framework, but in recognizing when competing physiologic goals must be reweighted in real time.

The future of aortic dissection care is likely to be shaped by increasingly precise and personalized approaches. Advances in imaging and computational modeling may enable real-time estimation of aortic wall stress, false lumen pressurization, and regional flow dynamics, allowing clinicians to tailor blood pressure and heart rate targets to an

individual patient's anatomy rather than relying solely on generalized thresholds. Continuous multimodal monitoring that integrates arterial waveform analysis, echocardiography, near-infrared spectroscopy, spinal cord perfusion metrics, and organ-specific biomarkers may further enhance bedside decision-making during periods of instability. Expanding understanding of the genetic architecture of aortic disease is transforming risk stratification and may refine surveillance intervals, influence operative thresholds, guide family screening, and eventually support development of targeted molecular therapies. Ongoing innovation in pharmacologic therapies, surgical technique, perfusion management and perioperative monitoring also hold promise for advancing patient outcomes. Progress, however, depends not only on technology but also on systems of care. High-quality outcomes in aortic dissection rely on coordinated collaboration among emergency physicians, anesthesiologists, intensivists, cardiac and vascular surgeons, perfusionists, radiologists, nurses, rehabilitation teams, and outpatient clinicians. Standardized pathways, rapid transfer networks, simulation-based training, and protocolized rescue responses for complications remain essential components of modern aortic programs.

Ultimately, management of aortic dissection is evolving from a primarily reactive discipline to on that is increasingly predictive, personalized, and precision-based. Early diagnosis, meticulous perioperative care, multidisciplinary collaboration, and individualized treatment plans are essential for optimizing survival, preserving organ function, and minimizing both short and long-term complications.

CLINICS CARE POINTS

- Immediate goals of care should be to establish diagnosis and extent of dissection, stabilize hemodynamics, and minimize risk of extension or rupture balanced with risk of malperfusion.
- Secondary consideration is given to preparation for next steps in care, typically urgent or emergent surgical or endovascular repair.
- Patient-centered, multidisciplinary care helps to ensure decision-making concordant with patient values while improving outcomes.

DISCLOSURE

The authors have nothing to disclose.

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