

ACUTE AORTIC SYNDROMES

Definitions and Classification

Definitions

The term **acute aortic syndrome** includes the classic aortic dissection and conditions with a similar clinical profile such as intramural aortic haematoma and penetrating atherosclerotic aortic ulcer.^{1,2}

Aortic dissection (AoD) denotes disruption of the medial layer of the aorta with bleeding within and along the wall, and resulting in separation of the layers of the aorta (Figure 1). The outer part of the aortic media forms with the adventitia the false channel outside wall, whereas the rest of the media and the intima constitute the inner wall, ie the intimomedial flap (also called intimal flap). An intimal disruption is usually present (90% of cases) that results in tracking of the blood in a dissection plane within the media. This may rupture through the adventitia or back through the intima into the aortic lumen. Dissection may, and often does, occur without an aneurysm being present.

A classification of aortic dissection chronicity based on the 2020 SVS/STS Reporting Standards is as follows:¹

Hyperacute dissection is defined as occurring <24 h of onset of pain

Acute dissection within 1-14 days of onset of pain.

Subacute dissection between 15-90 days from onset of pain.

Chronic dissection more than 90 days from onset of pain.

Intramural haematoma (IMH) is characterised by the absence of an entrance tear, and may be found in 5-30% of patients presenting with a suspected acute aortic syndrome.¹ The false lumen is created by a haemorrhage into the aortic media, most likely after rehexis of the vasa vasorum that penetrates the outer half of the aortic media from the adventitia. An entrance tear is not visualised by the current imaging techniques, but is usually found at surgery or autopsy. IMH is difficult to distinguish from classic dissection, and carries a higher rate of complications.³ Involvement of the ascending aorta and aortic diameter ≥ 5 cm are the most important predictors of mortality. Focal intimal disruption has been described in >20% of type B intramural hematomas.⁴

Penetrating atherosclerotic ulcer (PAU) is ulceration of an aortic atherosclerotic lesion that penetrates the internal elastic lamina into the media. It is usually found in the descending aorta and may precipitate IMH or classic dissection. The natural history is not well defined. Asymptomatic PAU usually display minimal growth and, probably, infrequent complications including rupture.⁵ The incidence is estimated to account for 2-7% of all cases of acute aortic syndromes.¹

Incomplete dissection refers to that situation in which there is laceration of the intima and subjacent media (dissection tear) without significant intramural (separation of the medial layers) dissection. Patients are at high risk of aortic rupture. Limited intimal tears are an uncommon, but potentially lethal, variant of aortic dissection. High-quality computed tomography imaging can detect these subtle lesions pre-operatively.⁶

Classifications of Aortic Dissection

The **De Bakey Classification** is based on the on the origin of the tear and the extent of the dissection (Figure 2):

Type I: Dissection originates in the ascending aorta and propagates distally to include at least the aortic arch and typically the descending aorta (surgery usually recommended).

Type II: Dissection originates in and is confined to the ascending aorta (surgery usually recommended).

Type III: Dissection originates in the descending aorta and propagates most often distally (nonsurgical treatment usually recommended. In type IIIa the dissection limited to the descending thoracic aorta, and in type IIIb it is extending below the diaphragm.

In **Stanford classification** is based on the involvement of the ascending aorta vs the arch and/or descending aorta (Figure 2).

Type A: All dissections involving the ascending aorta regardless of the site of origin (surgery usually recommended)

Type B: All dissections that do not involve the ascending aorta (nonsurgical treatment usually recommended). Involvement of the aortic arch without involvement of the ascending aorta in the Stanford classification is labeled as Type B.

In 2020, the SVS and the STS proposed an entirely new classification scheme that defines the aortic dissection anatomy in more granular detail.⁷ Dissections are defined anatomically according to the location of intimal tears and the proximal and distal extent of the dissection process (Figure 3).

Epidemiology

The true incidence of AoD is difficult to define since it leads to immediate death in 40% of patients. Estimates range from 3 to 16 cases per 100,000 persons per year, being higher in men (>40 years old) than in women.^{8,9} The mortality from thoracic aortic aneurysm and aortic dissection has been found in decline the last decades.^{10 1,11}

Aetiology and pathophysiology

Mechanisms that weaken the media layers of the aorta lead to higher wall stress, can induce

aortic dilatation and aneurysm formation, eventually resulting in intramural hemorrhage, aortic dissection, or rupture. Acute aortic dissection requires a tear in the aortic intima that commonly is preceded by medial wall degeneration or cystic medial necrosis.^{12,13}

Risk factors for AoD include conditions that result in aortic medial degeneration or place extreme stress on the aortic wall. The main risk factor is uncontrolled **arterial hypertension**. Elevated systolic and diastolic blood pressure are associated with a high risk of aortic dissection.¹⁴ Hypertension, aortic aneurysms, and chronic obstructive pulmonary disease are the most prevalent comorbidities.¹⁵ **Traumatic rupture** of the aorta is seen in 20% of fatal motor vehicle accidents. **Nonaortic cardiac surgery** is associated with a 0.15% incidence of type A aortic dissection, and is mainly seen in patients with dilated (>4cm), and thin ascending aortas.¹⁶

Certain **genetic conditions** such as mutations in the gene FBN1 that encodes the extracellular matrix protein, fibrillin-1, and cause *Marfan syndrome*, also weaken the media layers of the aorta and predispose to dissection. Several other mutations predispose to aortic aneurysms and dissections. Long-term use of fluoroquinolones (at least two months) has been associated with increased risk of aortic dissection and aneurysms,^{17,18} but the evidence is rather controversial and shorter use of these drugs should not be discouraged if clinically indicated.² Although ascending aortic dilation is a well-established risk factor for acute dissection, 60% of the patients in the International Registry of Acute Aortic Dissection (IRAD) had maximum aortic diameters <5.5 cm, and 40% of patients had aortic diameters <5.0 cm.¹¹

The risk of aortic dissection or rupture is elevated during **pregnancy** and the **postpartum** period.¹⁹

Presentation

Patients with acute aortic syndromes often present in a similar fashion, regardless of whether the underlying condition is AoD, IMH, PAU, or contained aortic rupture (Table 1). **Acute chest pain** is the most common symptoms, but dissection may also occur without any symptoms or signs (6% of cases).¹¹ In contrast with the more gradual increasing intensity of pain due to coronary syndromes, pain from aortic dissection has a sudden onset with maximal intensity often at the time of onset. However, coronary compression by the false lumen or extension of the dissection flap into the coronary ostium may cause **coronary ischemia** in up to 19% of cases. The classic characteristics of pain are its tearing or ripping quality, and most of the time pain is described as sharp or stabbing, but the description can be highly variable. Chest pain irradiating to the neck, throat or jaw indicates that the aortic segment involved is the ascending aorta, whereas pain located in the back or the abdomen suggests that the

diseased segment is most probably the descending aorta (Table 2). **Fever** may be present. **Cerebrovascular accidents** occur in up to 40% of cases and may be transient but is suggestive of arch vessel involvement. **Syncope** (13%) is an ominous sign; it may be caused by severe AR or tamponade, impaired cerebral flow, false lumen rupture into the pleural space, and vasovagal responses. Approximately 6-13% of patients present with **cardiac failure** or **shock**. **Death** is usually due to rupture of the false channel.

Physical Findings

A complete search for arterial perfusion differentials in both upper and lower extremities, evidence of visceral ischemia, focal neurologic deficits, a murmur of aortic regurgitation, bruits, and findings compatible with possible cardiac tamponade is essential. Findings depend upon the extension of the dissection as previously described and the rapidity of the process. **Aortic regurgitation** (45-75% of cases) may be due to dilatation of the aortic root, disruption of the aortic valve by extension of the dissection or by the prolapsing flap. **Pulse differentials** may be present (30%). The majority of patients with acute type A acute aortic dissection present with **aortic diameters <5.5 cm**. Although up to 75% of patients may be hypertensive, on presentation >50% patients with type A dissection may be found **normotensive or hypotensive** (IRAD data).²⁰ **Ischemic lower extremities** (30%), and **tamponade** (5-10% in proximal dissections) may also be present. Paraplegia and other neurological signs may be present in 10-40% of patients, and in half of the cases may be transient.²¹

Diagnosis

AoD should be suspected in any patient presenting with acute chest pain, especially in the context of a wide mediastinum, and, usually, and usually without ECG evidence of STEMI. A patient with thickening of the aortic walls and pericardial effusion or AR should be evaluated for an acute aortic syndrome. Recommendation for the initial evaluation of patients are presented in Table 2 and Figure 4.

The **aortic dissection detection (ADD) risk score** is a useful clinical marker, especially in combination with D-dimers levels (see below). It calculated on the basis of the number of risk categories (high-risk predisposing conditions, high-risk pain features, high-risk examination features).²²

Chest X-ray has low sensitivity in excluding AoD in patients with widened mediastinum or abnormal aortic contour (especially in patients with chest trauma), but a completely normal chest X-ray lowers the likelihood of AoD.

Transesophageal echocardiography has an 80% sensitivity and 95% specificity for proximal AoD (Figure 4). For distal AoD sensitivity is lower and mid-portion ascending dissections

may be missed. Other disadvantage is the interference of reverberation artifacts that are produced by the echocardiography that may obscure the picture.

Multidetector helical CT with contrast (305 mL/s up to 150 mL) has excellent sensitivity and specificity (100 and 98%, respectively) in diagnosing AoD even in trauma patients.²³ ECG-gated tomographic images limit motion artifacts.

Magnetic resonance angiography with gadolinium is also sensitive and specific but CT is usually more expeditious.²³ MRI or CT should be combined with TEE for optimum results.

Angiography has lower sensitivity and specificity than the other techniques.

D-dimers elevation is very sensitive for aortic dissection but not specific since it may rise in several other conditions (see PE). A negative test (cut off value of 500 ng/mL) performed within the first 24 hours of symptoms excludes dissection,²⁴ especially in association with an ADD risk score ≤ 1 .²⁵ Other biomarkers such as ST2, an interleukin (IL)-1 receptor family member are also promising. ST2 tests with levels < 35 ng/mL have been found superior to D-dimers in ruling out acute aortic dissection in patients with suspicion of this disease if used within 24 hours after symptom onset.²⁶

Therapy

Initial management

Decreasing wall stress by lowering blood pressure and heart rate is the first step in hemodynamically stable patients (Table 3 and Figure 5). IV propranolol, metoprolol, esmolol, and especially labetalol (an alpha and beta blocker) are used.^{27,28} If beta blockers are not tolerated or contraindicated verapamil or diltiazem are a less well established alternative. Initial targets are a heart rate < 60 bpm and a systolic blood pressure between 100 and 120 mm Hg. Vasodilators such as sodium nitroprusside, nicardipine or nitroglycerine may be needed after initiation of beta blockade to avoid reflex tachycardia and increase in the force of left ventricular ejection leading to increased aortic wall stress. Pain control with opiates is essential.

Surgery and TEVAR

Complications in acute aortic syndromes are presented in Figure 6. Acute aortic dissection of the ascending aorta (**type A**) is highly lethal, with a mortality of 1% to 2% per hour after symptom onset during the first 48 hours. Urgent surgical consultation is vital (Table 4 and Figures 7-9), since without surgery, mortality exceeds 50% at 1 month. Analysis of the IRAD data indicates that in the last 20 years in-hospital mortality rate of patients presenting with type A aortic dissection dropped significantly from 31% to 22%, mainly because of a decline in surgical mortality from 25% to 18%.¹¹ Type A dissections are operated with aortic root repair or implantation of a composite graft in the ascending aorta with or without

reimplantation of coronary arteries. Aortic root replacement is preferred with aortic root intimal tear (the intimal flap extending into the sinuses of Valsalva), underlying connective tissue disease (with a high risk of subsequent aneurysm development), aortic root diameter ≥ 4.5 cm (with the risk of aneurysm progression), or unreparable aortic valve pathology (at least moderate aortic stenosis or regurgitation and an unreparable valve).²⁹ Resecting or closing any significant communication between true and false lumina in the distal ascending aorta or aortic arch is important,³⁰ and preservation of the native aortic valve offers better survival than a Bentall procedure.³¹ Because cardiac tamponade and hemodynamic instability are rare (1%), patients with previous cardiac surgery in stable condition should undergo preoperative native coronary and graft status assessment and appropriate operative planning, including aggressive revascularization and the use of deep hypothermic cardiac arrest.¹⁶ In inoperable patients with type A dissection, stent grafting may also be considered.¹³

Patients with uncomplicated aortic dissections confined to the descending aorta (**type B**) are mainly treated with medical therapy. Long-term treatment with beta-blockers has been shown to reduce the progression of aortic dilatation in patients with chronic type B dissection.²⁷ The overall in-hospital mortality rate of patients presenting with type B aortic dissection has not changed significantly (12% versus 14%) in the past 20 years. Overall mortality of open surgery according to IRAD data was 30% versus 10% when patients were treated with medical therapy alone.¹¹ Thoracic endovascular aortic repair (TEVAR) involves implantation of a membrane-covered stent graft across the lesion taking care to avoid vessels supplying the major spinal cord. Paraplegia and stroke range between 0.8-1.9% and 2.1-3.5%, respectively and appear to be lower than with open surgery. In the first randomized study on elective stent-graft placement in survivors of uncomplicated type B aortic dissection (INSTEAD trial), TEVAR failed to improve 2-year survival and adverse event rates. The pooled early mortality rate was approximately 6.4% with medical treatment, 10.2% with transthoracic endovascular repair (TEVAR), and 17.5% with open surgery.³² However, in a retrospective analysis of the International Registry of Acute Aortic Dissection,³³ as well as in other centres,³⁴ TEVAR was associated with lower mortality over a 5-year period compared to medical therapy alone. In survivors of type B dissection, thoracic endovascular aortic repair was also associated with improved 5-year aorta-specific survival and delayed disease progression, and should be considered to avoid late complications.³⁵ In a retrospective analysis of the Medicare database, open surgical repair was associated with increased odds of early postoperative mortality but reduced late hazard of death. Despite the late advantage of open repair, mean survival was superior for TEVAR. TEVAR, therefore, should be considered the first line for repair of intact descending thoracic aortic aneurysms.³⁶ Patients' age ≥ 90 years is not a contraindication.³⁷ TEVAR is considered appropriate in complicated type B dissection,³⁸ but not yet as a pre-emptive measure in uncomplicated dissections.¹³

However, most type B aortic dissections that are medically managed progress to aneurysms that increase the incidence of aorta related adverse events and mortality. Thus, early TEVAR has been proposed and is currently being studied.³⁹

Intramural hematomas and penetrating ulcers

Intramural hematomas and penetrating ulcers are treated in the same manner as classic dissection (Figures 10 and 11, and Table 5). The rate of aortic rupture is much higher for intramural hematoma (35%) and penetrating atheromatous ulcer (42%) in comparison with aortic dissection (type A 7.5%, type B 4.1%).⁴⁰ There has been some evidence that intramural hematoma type B may have a slightly more benign course than classic acute type B aortic dissection (ie with an intimal flap) in the acute setting, and can often be treated medically.⁴¹ The outlook is more favourable during the subacute or chronic phase, warranting medical management with periodic clinical and imaging surveillance.⁴ Asymptomatic penetrating aortic ulcers may be conservatively managed with serial imaging and risk factor modification.⁵

Aortic dissection with *persistent patent false lumen* carries a high risk of complications. Marfan syndrome, aorta diameter, and a large entry tear located in the proximal part of the dissection indicates a high risk of complications in this setting, particularly with type B dissections.³⁰

Traumatic Aortic Injury

Urgent surgical repair is recommended in *traumatic aortic rupture* (Figures 12-14, and Table 6). *Iatrogenic catheter dissection* of the aorta is a rare complication that usually carries an excellent short- and long-term prognosis with the adoption of a conservative approach. When a coronary artery is involved as an entry point, it usually can be sealed safely with a stent with good results.⁴²

Follow-up

Recommendations for follow-up after treatment of acute aortic syndrome are presented in Table 7 and Figure 15.

Pregnancy

Recommendations for pregnancy in patients with aortopathy in general are discussed in the Chapter on Aortic Anatomy and Disease.⁴³

References

1. Writing Committee M, et al. 2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. *Journal of the American College of Cardiology*. 2022. doi: 10.1016/j.jacc.2022.08.004
2. Mazzolai L, et al. 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases. *Eur Heart J*. 2024. doi: 10.1093/eurheartj/ehae179
3. Alomari IB, et al. Aortic intramural hematoma and its complications. *Circulation*. 2014;129:711-716. doi: 10.1161/CIRCULATIONAHA.113.001809
4. Moral S, et al. Clinical Implications of Focal Intimal Disruption in Patients With Type B Intramural Hematoma *Journal of the American College of Cardiology*. 2017:28-39.
5. DeCarlo C, et al. Prognostication of Asymptomatic Penetrating Aortic Ulcers: A Modern Approach. *Circulation*. 2021;144:1091-1101. doi: 10.1161/CIRCULATIONAHA.121.054710
6. Chin AS, et al. Acute Limited Intimal Tears of the Thoracic Aorta. *JACC*. 2018.
7. Lombardi JV, et al. Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) Reporting Standards for Type B Aortic Dissections. *Ann Thorac Surg*. 2020;109:959-981. doi: 10.1016/j.athoracsur.2019.10.005
8. Olsson C, et al. Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. *Circulation*. 2006;114:2611-2618. doi: CIRCULATIONAHA.106.630400 [pii]
10.1161/CIRCULATIONAHA.106.630400
9. Goldfinger JZ, et al. Thoracic aortic aneurysm and dissection. *Journal of the American College of Cardiology*. 2014;64:1725-1739. doi: 10.1016/j.jacc.2014.08.025
10. Sidloff D, et al. Mortality from thoracic aortic diseases and associations with cardiovascular risk factors. *Circulation*. 2014;130:2287-2294. doi: 10.1161/CIRCULATIONAHA.114.010890
11. Evangelista A, et al. Insights From the International Registry of Acute Aortic Dissection. A 20-Year Experience of Collaborative Clinical Research. *Circulation*. 2018. doi: 10.1161/CIRCULATIONAHA.117.031264
12. Peterss S, et al. Changing Pathology of the Thoracic Aorta From Acute to Chronic Dissection: Literature Review and Insights. *Journal of the American College of Cardiology*. 2016;68:1054-1065. doi: 10.1016/j.jacc.2016.05.091
13. Bossone E, et al. Acute aortic syndromes: diagnosis and management, an update. *Eur Heart J*. 2017. doi: 10.1093/eurheartj/ehx319
14. Hibino M, et al. Blood Pressure, Hypertension, and the Risk of Aortic Dissection Incidence and Mortality: Results From the J-SCH Study, the UK Biobank Study, and a Meta-Analysis of Cohort Studies. *Circulation*. 2022;145:633-644. doi: 10.1161/CIRCULATIONAHA.121.056546
15. Obel LM, et al. Clinical Characteristics, Incidences, and Mortality Rates for Type A and B Aortic Dissections: A Nationwide Danish Population-Based Cohort Study From 1996 to 2016. *Circulation*. 2022;146:1903-1917. doi: 10.1161/CIRCULATIONAHA.122.061065
16. Stanger O, et al. Type A aortic dissection after nonaortic cardiac surgery. *Circulation*. 2013;128:1602-1611. doi: 10.1161/CIRCULATIONAHA.113.002603
17. Lee CC, et al. Oral Fluoroquinolone and the Risk of Aortic Dissection. *Journal of the American College of Cardiology*. 2018;72:1369-1378. doi: 10.1016/j.jacc.2018.06.067
18. Chen S-W, et al. Effects of Fluoroquinolones on Outcomes of Patients With Aortic Dissection or Aneurysm. *JACC*. 2021.
19. Kamel H, et al. Pregnancy and the Risk of Aortic Dissection or Rupture: A Cohort-Crossover Analysis. *Circulation*. 2016;134:527-533. doi: 10.1161/CIRCULATIONAHA.116.021594
20. Pape LA, et al. Aortic diameter ≥ 5.5 cm is not a good predictor of type A aortic dissection: observations from the International Registry of Acute Aortic Dissection (IRAD). *Circulation*. 2007;116:1120-1127. doi: CIRCULATIONAHA.107.702720 [pii]
10.1161/CIRCULATIONAHA.107.702720
21. Coady MA, et al. Pathologic variants of thoracic aortic dissections. Penetrating atherosclerotic ulcers and intramural hematomas. *Cardiol Clin*. 1999;17:637-657.
22. Rogers AM, et al. Sensitivity of the aortic dissection detection risk score, a novel guideline-based tool for identification of acute aortic dissection at initial presentation: results from the international registry of acute aortic dissection. *Circulation*. 2011;123:2213-2218. doi: 10.1161/CIRCULATIONAHA.110.988568
23. Gulati M, et al. 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*. 2021:CIR0000000000001029. doi: 10.1161/CIR.0000000000001029
24. Suzuki T, et al. Diagnosis of acute aortic dissection by D-dimer: the International Registry of Acute Aortic Dissection Substudy on Biomarkers (IRAD-Bio) experience. *Circulation*. 2009;119:2702-2707. doi: 10.1161/CIRCULATIONAHA.108.833004
CIRCULATIONAHA.108.833004 [pii]

25. Nazerian P, et al. Diagnostic Accuracy of the Aortic Dissection Detection Risk Score Plus D-Dimer for Acute Aortic Syndromes: The ADVISED Prospective Multicenter Study. *Circulation*. 2017. doi: 10.1161/CIRCULATIONAHA.117.029457
26. Wang Y, et al. Magnitude of Soluble ST2 as a Novel Biomarker for Acute Aortic Dissection. *Circulation*. 2017. doi: 10.1161/CIRCULATIONAHA.117.030469
27. Genoni M, et al. Chronic beta-blocker therapy improves outcome and reduces treatment costs in chronic type B aortic dissection. *Eur J Cardiothorac Surg*. 2001;19:606-610. doi: S1010794001006625 [pii]
28. Whelton PK, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Journal of the American College of Cardiology*. 2017. doi: 10.1016/j.jacc.2017.11.006
29. Yang B, et al. Short- and long-term outcomes of aortic root repair and replacement in patients undergoing acute type A aortic dissection repair: Twenty-year experience. *J Thorac Cardiovasc Surg*. 2018. doi: 10.1016/j.jtcvs.2018.09.129
30. Evangelista A, et al. Long-term outcome of aortic dissection with patent false lumen: predictive role of entry tear size and location. *Circulation*. 2012;125:3133-3141. doi: 10.1161/CIRCULATIONAHA.111.090266
- CIRCULATIONAHA.111.090266 [pii]
31. Yang B, et al. Short-term outcomes of a simple and effective approach to aortic root and arch repair in acute type A aortic dissection. *J Thorac Cardiovasc Surg*. 2018;155:1360-1370 e1361. doi: 10.1016/j.jtcvs.2017.11.089
32. Nienaber CA, et al. Randomized comparison of strategies for type B aortic dissection: the INvestigation of STent Grafts in Aortic Dissection (INSTEAD) trial. *Circulation*. 2009;120:2519-2528. doi: 10.1161/CIRCULATIONAHA.109.886408
33. Fattori R, et al. Survival after endovascular therapy in patients with type B aortic dissection: a report from the International Registry of Acute Aortic Dissection (IRAD). *JACC Cardiovasc Interv*. 2013;6:876-882. doi: 10.1016/j.jcin.2013.05.003
34. Qin YL, et al. Endovascular Repair Compared With Medical Management of Patients With Uncomplicated Type B Acute Aortic Dissection. *Journal of the American College of Cardiology*. 2016;67:2835-2842. doi: 10.1016/j.jacc.2016.03.578
35. Nienaber CA, et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. *Circ Cardiovasc Interv*. 2013;6:407-416. doi: 10.1161/CIRCINTERVENTIONS.113.000463
36. Chiu P, et al. Endovascular Versus Open Repair of Intact Descending Thoracic Aortic Aneurysms. *Journal of the American College of Cardiology*. 2019;73:643-651. doi: 10.1016/j.jacc.2018.10.086
37. Prendes CF, et al. Endovascular Aortic Repair in Nonagenarian Patients. *JACC*. 2021. doi: 10.1016/j.jacc.2021.02.042
38. Grabenwoger M, et al. Thoracic Endovascular Aortic Repair (TEVAR) for the treatment of aortic diseases: a position statement from the European Association for Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI). *Eur Heart J*. 2012;33:1558-1563. doi: 10.1093/eurheartj/ehs074
- ehs074 [pii]
39. Tadros RO, et al. Optimal Treatment of Uncomplicated Type B Aortic Dissection. *JACC*. 2019.
40. Coady MA, et al. Surgical management of descending thoracic aortic disease: open and endovascular approaches: a scientific statement from the American Heart Association. *Circulation*. 2010;121:2780-2804. doi: 10.1161/CIR.0b013e3181e4d033
- CIR.0b013e3181e4d033 [pii]
41. Tolenaar JL, et al. The differences and similarities between intramural hematoma of the descending aorta and acute type B dissection. *J Vasc Surg*. 2013;58:1498-1504. doi: 10.1016/j.jvs.2013.05.099
42. Nunez-Gil IJ, et al. Incidence, Management, and Immediate- and Long-Term Outcomes After Iatrogenic Aortic Dissection During Diagnostic or Interventional Coronary Procedures. *Circulation*. 2015;131:2114-2119. doi: 10.1161/CIRCULATIONAHA.115.015334
43. De Backer J, et al. 2025 ESC Guidelines for the management of cardiovascular disease and pregnancy. *Eur Heart J*. 2025. doi: 10.1093/eurheartj/ehaf193