Eur J Vasc Endovasc Surg (2026) 71, 172—270

CLINICAL PRACTICE GUIDELINE DOCUMENT

European Society for Vascular Surgery (ESVS) 2026 Clinical Practice Guidelines on the Management of Descending Thoracic and Thoraco-Abdominal Aortic Diseases – Editor's Choice

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Objective: The European Society for Vascular Surgery (ESVS) has developed clinical practice guidelines for the care of patients with descending thoracic and thoraco-abdominal aortic pathologies, in succession to the 2017 version, with the aim of assisting physicians and patients in selecting the best management strategy.

Methods: The guidelines are based on scientific evidence complemented with expert opinion on the matter. By summarising and evaluating the best available evidence, recommendations for the evaluation and treatment of patients have been formulated. The recommendations are graded according to the ESVS grading system, where the strength (class) of each recommendation is graded from I to III and the level of evidence from A to C.

Results: One hundred and twenty-nine recommendations have been issued across the following main topics: (1) acute thoracic aortic syndrome; (2) chronic type B aortic dissection; (3) descending thoracic and thoraco-abdominal aortic aneurysms; (4) ruptured descending thoracic and thoraco-abdominal aortic aneurysms; and (5) blunt thoracic aortic injury. Additional topics include genetic aortopathy, floating thrombus and shaggy aorta, inflammatory aortitis, mycotic aortic aneurysms, coarctation of the aorta, aberrant subclavian artery, and service standards such as surgical volume, imaging, risk assessment, and optimisation. Special considerations include pregnancy, left subclavian artery revascularisation, spinal cord ischaemia, stroke prevention, vascular access, and the patient's perspective. A final chapter addresses unresolved issues.

Conclusion: These clinical practice guidelines provide comprehensive, up to date advice to clinicians and patients on the management of descending thoracic and thoraco-abdominal aortic pathologies.

Keywords: Aneurysm, Aortic, Descending thoracic aorta, Dissection, Guideline, Thoraco-abdominal aorta
Available online 23 December 2025

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https://doi.org/10.1016/j.ejvs.2025.12.050

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ABBREVIATIONS

4D flow MRI	Four dimensional flow magnetic resonance imaging
AAA	Abdominal aortic aneurysm
AAS	Acute aortic syndrome
ACE	Angiotensin converting enzyme
ACR	American College of Rheumatology
ACT	Activated clotting time
AD	Aortic dissection
AGREE II	Appraisal of Guidelines for Research and Evaluation
AHI	Aortic height index
ARA	Accessory renal artery
ARB	Angiotensin receptor blocker
ASA	American Society of Anesthesiologists
ASI	Aortic size index
ATBAD	Acute type B aortic dissection
BMI	Body mass index
BMT	Best medical treatment
BTAI	Blunt thoracic aortic injury
CA	Coeliac artery
CAD	Coronary artery disease
CEUS	Contrast enhanced ultrasound
CFA	Common femoral artery
CI	Confidence interval
CMD	Custom made device
CoA	Coarctation of the aorta
COPD	Chronic obstructive pulmonary disease
CSFD	Cerebrospinal fluid drainage
CT	Computed tomography
CTA	Computed tomography angiography
CTBAD	Chronic type B aortic dissection
CTD	Connective tissue disorder
DAPT	Dual antiplatelet therapy
DASI	Duke Activity Status Index
DMARD	Disease modifying antirheumatic drug
dSINE	Distal stent graft induced new entry
DTA	Descending thoracic aorta
DTAA	Descending thoracic aortic aneurysm
DUS	Duplex ultrasound
EACTS	European Association for Cardio-Thoracic Surgery
ECG	Electrocardiogram
eGFR	Estimated glomerular filtration rate
EJVES	European Journal of Vascular and Endovascular Surgery
ESVS	European Society for Vascular Surgery

EULAR	European League Against Rheumatism
EVAR	Endovascular aortic repair
FBEVAR	Fenestrated and branched endovascular aortic repair
^{18}F-FDG	Fluorine-18 fluorodeoxyglucose
FL	False lumen
FTAD	Familial thoracic aortic disease
GCA	Giant cell arteritis
GSC	Guideline Steering Committee
GWC	Guideline Writing Committee
HR	Hazard ratio
HTAD	Heritable thoracic aortic disease
HTK	Histidine—tryptophan—ketoglutarate
ICU	Intensive care unit
IL	Interleukin
IMH	Intramural haematoma
IRAD	International Registry of Acute Aortic Dissection
ISS	Injury Severity Score
KD	Kommerell's diverticulum
LDS	Loeys—Dietz syndrome
LHB	Left heart bypass
LIMA	Left internal mammary artery
LSA	Left subclavian artery
LZ	Landing zone
MEP	Motor evoked potential
MET	Metabolic equivalent
MFS	Marfan syndrome
MI	Myocardial infarction
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
NT-proBNP	N terminal pro-brain natriuretic peptide
OR	Odds ratio
OSR	Open surgical repair
PAU	Penetrating aortic ulcer
PET	Positron emission tomography
PETTICOAT	Provisional extension to induce complete attachment
PG	Parallel graft
PMEG	Physician modified endograft
QoL	Quality of life
RCT	Randomised controlled trial
ROC	Receiver operating characteristic
ROTEM	Rotational thromboelastometry
RR	Risk ratio
RTAAD	Retrograde type A aortic dissection
SBP	Systolic blood pressure
SCI	Spinal cord ischaemia
SDM	Shared decision making
SINE	Stent graft induced new entry
SLE	Systemic lupus erythematosus
SSEP	Somatosensory evoked potential
STABILISE	Stent assisted balloon induced intimal disruption and relamination in aortic dissection repair
STS	Society of Thoracic Surgeons
SVS	Society for Vascular Surgery
TA	Takayasu arteritis
TAA	Thoracic aortic aneurysm
TAbdAo	Thoraco-abdominal aorta
TAAA	Thoraco-abdominal aortic aneurysm
TAAD	Type A aortic dissection
TAVR	Transcatheter aortic valve replacement
TBAD	Type B aortic dissection
TEG	Thrombo-elastography
TES	Transcatheter electrosurgical septotomy
TEVAR	Thoracic endovascular aortic repair
ToE	Table of Evidence
TOE	Transoesophageal echocardiography
TL	True lumen
TTE	Transthoracic echocardiography
uTBAD	Uncomplicated type B aortic dissection
VATS	Video assisted thoracoscopic surgery
vEDS	Vascular Ehlers—Danlos syndrome
VLFDC	Vascular Low Frequency Disease Consortium
VQI	Vascular Quality Initiative

STUDY ACRONYMS

ADSORB	Acute Dissection Stentgraft OR Best Medical Treatment
BBEST	Beta Blockers in Ehlers—Danlos Syndrome Treatment
COAST	Coarctation of the Aorta Stent Trial
EARNEST	Early Aortic Repair in patients Needing Endovascular or open Surgery for Type B Aortic Dissection
ETTAA	Effective Treatments for Thoracic Aortic Aneurysms
EVICTUS	Endovascular Aortic Intervention in Patients with Connective Tissue Disease
IMPROVE-AD	IMPRoving Outcomes in Vascular DisEase — Aortic Dissection
INSTEAD	INvestigation of STEnt Grafts in Aortic Dissection
INTERCEPTevar	Carbon Dioxide Flushing versus Saline Flushing of Thoracic Aortic Stents
SUNDAY	Scandinavian Trial of Uncomplicated Aortic Dissection Therapy

WHAT IS NEW COMPARED WITH THE EUROPEAN SOCIETY FOR VASCULAR SURGERY (ESVS) 2017 DESCENDING THORACIC AORTA DISEASE GUIDELINES?

The European Society for Vascular Surgery (ESVS) 2026 clinical practice guidelines on the management of descending thoracic aorta (DTA) and thoraco-abdominal aorta (TAbdAo) diseases represent a comprehensive and fully updated revision of the previous 2017 edition.[1] Every section has been carefully revised or rewritten to reflect the rapid technological and clinical advances in the field over the past decade.

A total of 129 recommendations have been issued; 42 graded Class I, 57 Class IIa, 26 Class IIb, two Class IIIa, and two Class IIIb. Compared with the previous 2017 edition, 83 recommendations are new (21 Class I, 39 Class IIa, 19 Class IIb, two Class IIIa, two Class IIIb) and 33 recommendations (17 Class I, 11 Class IIa, five Class IIb) have been regraded or significantly rephrased, altering meaning to some extent. Only 13 recommendations remain unchanged (four Class I, seven Class IIa, two Class IIb). This reflects both the growing body of knowledge in DTA/TAbdAo disease management and the broadened scope of the document, highlighting the urgent need for updated guidance to keep pace with rapidly evolving technology, particularly in the field of complex endovascular repair.

The 2026 guidelines integrate 458 new references published between 2018 and 2025, including nine primary or secondary analyses from randomised controlled trials (RCTs), 131 literature reviews and meta-analysis, and 75 studies based on vascular registries or quality initiative programmes. Nevertheless, the overall strength of evidence remains weak, with only three of 129 (2.3%) recommendations supported by Level A evidence, five by Level B evidence, and a striking 121 (93.8%) recommendations rely on Level C evidence or expert consensus. This highlights the persistent need for robust prospective studies to strengthen the evidence base for DTA/TAbdAo disease management.

Structurally, the guidelines introduce several new chapters, expand existing content, and place greater emphasis on multidisciplinary decision making, patient centred care, and quality assurance. A new chapter on service standards addresses resource requirements, institutional capabilities, minimum surgical and endovascular case volumes, registry participation, and quality control measures. Another major update consolidates recommendations on imaging, risk assessment, and peri-operative considerations. Here, upgraded guidance is provided on computed tomography angiography for diagnosis and follow up, while new sections introduce intra-operative fusion imaging, magnetic resonance imaging, transoesophageal echocardiography, intravascular ultrasound, and fluorodeoxyglucose positron emission tomography/computed tomography (FDG-PET/CT) for detecting infection or inflammation. The same chapter introduces the concept of multidisciplinary pre-operative risk assessment by dedicated aortic teams, expands recommendations on medical optimisation, and provides new advice on managing the “shaggy” aorta. It also emphasises comprehensive patient information and shared decision making (SDM) to ensure individualised care. Special attention is given to pregnancy related DTA pathology and dissections, with a strong recommendation to refer such cases to high volume aortic centres for individualised multidisciplinary management by cardiovascular—obstetric teams. Several new recommendations address left subclavian artery revascularisation techniques, reflecting the rapid development of endovascular approaches. Spinal cord ischaemia prevention and management have been further protocolised, with a stronger focus on staged repair, reactive cerebrospinal fluid drainage, preservation of collateral networks, and early clinical neurological evaluation. A new recommendation suggests pre-flushing of the thoracic endograft delivery system during thoracic endovascular aortic repair (TEVAR) to reduce the risk of cerebral air embolism. In addition, a newly added section on vascular access recommends percutaneous femoral access with ultrasound guidance and provides updated strategies to minimise access related complications.

The chapter on acute thoracic aortic syndromes has been comprehensively updated. Conservative management remains the standard of care for uncomplicated acute type B aortic dissection (ATBAD), with anti-impulse therapy and structured imaging surveillance forming the cornerstone of treatment. A new negative Class IIIa recommendation explicitly advises against routine TEVAR in these cases. In recent years, much attention has been paid to identifying high risk features for complications in ATBAD, with invasive treatment increasingly considered. However, the Guideline Writing Committee notes that among the proposed markers, only early aortic expansion with a diameter greater than 4.0 cm has shown consistent predictive value for aneurysmal degeneration. Other factors, such as false lumen diameter, primary entry tear size or location, and false lumen thrombosis, remain less well validated. Pending the results of ongoing RCTs, early TEVAR should therefore be considered only in carefully selected patients with uncomplicated ATBAD who show early aortic expansion or a large initial diameter greater than 40 mm, preferably during the subacute phase. Several new and revised recommendations address endovascular treatment of complicated type B dissections, incorporating updated technical strategies.

In chronic type B aortic dissection, the principal threshold for elective repair remains unchanged at 60 mm. This distinction is important, as it differs from some other contemporary guidelines and reflects the absence of robust evidence supporting any benefit from lowering the threshold for intervention. However, numerous new recommendations reflect advances in fenestrated and branched endovascular aortic repair (FBEVAR) as well as adjunctive endovascular techniques. The indication for open surgical repair is more restricted, further emphasising an endovascular first strategy when anatomically feasible. The same paradigm applies to descending thoracic aortic aneurysm (DTAA) and thoraco-abdominal aortic aneurysm (TAAA), where the 6.0 cm threshold for elective repair is

retained, but complex endovascular repair is prioritised over open surgery whenever possible. This chapter also introduces new sections on peri-operative anticoagulation, antithrombotic therapy, and considerations for accessory renal artery preservation during repair.

The chapter on ruptured DTAA and TAAA issues multiple new recommendations on decision pathways for emergency endovascular *vs.* open repair. The management of blunt thoracic aortic injury has been updated in line with the newly adopted ESVS classification, harmonising recommendations with the 2025 ESVS vascular trauma guidelines.[2] Non-operative management is now the preferred strategy for ESVS grade 1 injuries, while ESVS grade 2 injuries are stratified into high and low risk subgroups, guiding the decision between urgent or delayed TEVAR.

Further new chapters expand the scope of the guidelines. An entirely new section covers the classification, detection, and management of endoleaks and distal stent induced new entry, with updated imaging protocols and tailored treatment recommendations. Genetic aortopathies now receive expanded detailed guidance on genetic testing, surveillance, and surgical decision making, with carefully selected patients offered endovascular repair as an alternative option. Additional recommendations address special conditions such as floating thrombus, aberrant subclavian artery (arteria lusoria) and Kommerell's diverticulum, aortic coarctation, and inflammatory aortic diseases such as Takayasu arteritis and giant cell arteritis. Mycotic aortic aneurysms are now recognised as treatable by either open surgery or endovascular repair, depending on anatomic suitability.

Taken together, the 2026 guidelines strongly emphasise an endovascular first paradigm wherever anatomy permits, reflecting the central role of fenestrated and branched techniques in contemporary practice. It also underlines the importance of multidisciplinary care, promoting centralised treatment in high volume aortic centres and structured team based decision making. Quality assurance is reinforced through recommendations on registry participation, standardised outcome reporting, and continuous quality improvement. A greater focus is placed on patient centred care, with an emphasis on incorporating patient perspectives, SDM, and tailoring interventions to life expectancy and functional status. Finally, the guidelines acknowledge the persistent evidence gaps in many areas of aortic disease management and call for robust prospective trials and registry based studies to address these limitations in the future.

Key themes across the 2026 update:

- Endovascular first paradigm — complex endovascular repair (FBEVAR) is prioritised whenever anatomically feasible.
- Multidisciplinary care — stronger emphasis on centralised treatment in high volume centres and team based decision making.
- Quality assurance — reinforced guidance on participation in registries, structured reporting, and continuous quality improvement.
- Patient centred approach — expanded focus on patient perspectives, SDM, and tailoring interventions to life expectancy and functional status.
- Acknowledgment of evidence gaps — clear recognition of the limited high quality evidence in many areas, with a call for future robust prospective studies.

Table 1. New, changed, and unchanged recommendations included in the European Society for Vascular Surgery (ESVS) 2026 clinical practice guidelines on the management of descending thoracic and thoraco-abdominal aortic pathologies in comparison with the previous 2017 guidelines. Numbers correspond to the numbers of the recommendations in the guideline document.

New Class I recommendations	
1.	Management of descending thoracic aortic disease is recommended to be organised in defined networks with established referral pathways.
4.	A fast track vascular surgical care pathway is recommended for patients with a descending thoracic aortic pathology who meet the size criteria for elective repair.
5.	Aortic centres performing descending thoracic aortic repair are recommended to enrol cases in validated prospective quality registries to enable systematic monitoring of practice and clinical outcomes.
16.	Assessment and optimisation of cardiovascular risk factors, best medical therapy, and a healthy lifestyle including smoking cessation are recommended for patients with descending thoracic aortic disease.
18.	Patients with thoracic aortic disease are recommended to be fully informed about all treatment options (medical management, endovascular repair, or open surgery), as well as their short and long term risks, including their potential impact on function, independence, and quality of life, to support shared decision making.
20.	Pregnant women with descending thoracic aortic aneurysm or dissection are recommended to be referred to experienced aortic centres for individualised multidisciplinary management by a cardiovascular–obstetric team.
29.	Early clinical neurological evaluation is recommended after open or endovascular thoracic or thoraco-abdominal aortic repair to enable prompt detection and treatment of spinal cord ischaemia.
32.	Pre-operative assessment of the iliofemoral access vessels is recommended before thoracic endovascular aortic repair.
35.	Ultrasound guidance is recommended for percutaneous common femoral artery access in thoracic endovascular aortic repair.

Continued

Table 1-*continued*

42. Patients with residual malperfusion following thoracic endovascular aortic repair for complicated acute type B aortic dissection are recommended to undergo immediate selective endovascular revascularisation of the affected target vessels.
60. Patients with a small descending thoracic or thoraco-abdominal aortic aneurysm who are deemed fit for repair are recommended for surveillance with serial cross sectional imaging to monitor growth.
74. Intra-operative administration of intravenous heparin (50 − 100 IU/kg) is recommended during elective endovascular repair of descending thoracic and thoraco-abdominal aortic aneurysms to prevent thromboembolic complications.
81. Thoracic endovascular aortic repair is recommended as the preferred treatment modality for ruptured descending thoracic aortic aneurysm.
88. Non-operative management, with blood pressure control and follow up imaging to assess lesion stability, is recommended for ESVS grade 1 blunt thoracic aortic injury without concomitant traumatic brain injury.
96. Continued medical therapy with anti-impulse treatment and serial imaging until aortic remodelling is recommended for patients with blunt thoracic aortic injuries managed non-operatively.
97. After endovascular stent graft repair of blunt thoracic aortic injury, follow up imaging is recommended at one month, one year, and annually thereafter for a minimum of five years, followed by continued imaging follow up every five to ten years for life.
103. Genetic counselling and testing are recommended in patients with thoracic aortic disease < 60 years of age, a family history of thoracic aortic disease, concomitant arterial aneurysms or dissections, or syndromic features.
104. Patients with suspected or confirmed genetic aortopathies are recommended for multidisciplinary management at highly specialised aortic centres.
114. Multidisciplinary team management is recommended for active inflammatory disease of the aorta and or its branches.
118. Long term post-operative surveillance is recommended for patients treated for inflammatory aortitis, given the high risk of delayed complications and disease progression.
119. Multidisciplinary management at highly specialised aortic centres is recommended for patients with mycotic thoracic and thoraco-abdominal aortic aneurysms.

New Class IIa recommendations

2. Patients with acute descending thoracic aortic disease should be considered for prompt transfer to aortic centres for comprehensive evaluation and management.
8. To reduce radiation exposure, magnetic resonance imaging should be considered as an alternative imaging modality for diagnosis, surveillance, and post-operative follow up of descending thoracic aortic pathologies, especially in younger patients.
9. Flourine-18 fluorodeoxyglucose positron emission tomography integrated with computed tomography (18F-FDG-PET/CT) should be considered an adjunctive imaging modality in the diagnostic evaluation of infected and non-infected aortitis of the descending thoracic aorta.
10. Intra-operative image fusion should be considered during endovascular thoracic and thoraco-abdominal aortic repair to reduce radiation exposure, contrast volume, and operating time.
15. Multidisciplinary assessment of patient frailty should be considered for candidates for elective descending thoracic aortic repair.
22. The choice between open and endovascular left subclavian artery revascularisation during thoracic endovascular aortic repair should be considered on a case by case basis, taking into account urgency, patient physiology, anatomy, underlying pathology, and patient preferences.
24. Staged repair should be considered for patients undergoing extensive thoracic and thoraco-abdominal endovascular aortic repair.
28. Preservation of flow to the internal iliac arteries should be considered during endovascular thoraco-abdominal aortic repair to reduce the risk of spinal cord ischaemia.
33. To enable device delivery, endovascular or open surgical optimisation should be considered for compromised access due to diseased and or small calibre iliofemoral arteries.
34. Percutaneous access should be considered the preferred method for common femoral artery access in patients undergoing thoracic endovascular aortic repair.
38. Patients with uncomplicated acute type B aortic dissection should be considered for continued conservative management, with anti-impulse therapy and surveillance with serial cross sectional imaging.
41. Distal extension with an appropriately sized endograft should be considered to ensure a distal seal in ruptured acute type B aortic dissection.
43. Proximal landing of the thoracic endograft in non-dissected aorta should be considered for patients undergoing treatment for acute type B aortic dissection.
51. Patients with type B aortic dissection should be considered for long term statin therapy.
55. Proximal landing of the thoracic endograft in non-dissected aorta or surgical graft should be considered in patients treated for chronic type B aortic dissection.

Continued

Table 1-*continued*

56.	Endovascular repair with fenestrated and branched technologies should be considered first line therapy in patients with chronic type B aortic dissection and aneurysm formation involving the thoraco-abdominal aorta.
67.	If coverage of the coeliac artery is required to achieve an adequate seal during endovascular repair for descending thoracic aortic repair, revascularisation should be considered if collateral flow from the superior mesenteric artery is insufficient, preferably by endovascular techniques.
69.	Balloon expandable bridging covered stent design should be considered as first choice for fenestrations.
72.	Renal perfusion using cold crystalloid solutions, such as histidine–tryptophan–ketoglutarate (HTK) solution, should be considered during open surgical repair of thoracic and thoraco-abdominal aortic aneurysms to minimise ischaemia–reperfusion injury.
73.	Viscoelastic testing to assess real time clot formation and stability should be considered during open surgical repair of thoracic and thoraco-abdominal aortic aneurysms to guide haemostatic management.
75.	Activated clotting time monitoring should be considered during open and endovascular repair of thoracic and thoraco-abdominal aortic aneurysms to assess heparin efficacy and guide additional dosing.
77.	Preservation of large accessory renal arteries ($\geq$ 4 mm) or those that supply a significant portion of the kidney ($\geq$ one third) should be considered during thoraco-abdominal aortic aneurysm repair to minimise the risk of post-operative renal dysfunction.
80.	A policy of permissive hypotension during the pre-operative period should be considered in patients with a ruptured thoracic or thoraco-abdominal aortic aneurysm, provided adequate end organ perfusion is maintained.
82.	Increased endograft oversizing should be considered in emergency endovascular repair of ruptured descending thoracic aortic aneurysm when pre-operative imaging was obtained during hypotension.
83.	Endovascular repair using off the shelf branched stent grafts, physician modified endografts, or *in situ* fenestration techniques should be considered the preferred treatment modality for ruptured thoraco-abdominal aortic aneurysms.
87.	Video assisted thoracoscopic decompression of haemothorax should be considered for organised haematoma or inadequate percutaneous drainage cases after endovascular ruptured descending thoracic aorta repair.
94.	Systemic heparinisation during thoracic endovascular aortic repair for blunt traumatic aortic injury should be considered individualised, taking into account the risk of bleeding, thromboembolic complications, and the severity of any associated traumatic brain injury.
95.	Moderate stent graft oversizing should be considered in emergency endovascular repair of blunt thoracic aortic injury when pre-operative imaging was obtained during hypotension.
98.	Magnetic resonance angiography should be considered the preferred imaging modality for long term surveillance after blunt thoracic aortic injury, whenever feasible.
102.	Distal stent graft induced new entry should be considered for re-intervention with distal endograft extension to seal the defect.
105.	Patients with genetic aortopathies should be considered for serial imaging surveillance of the aorta and its major arterial branches with computed tomography or magnetic resonance angiography.
112.	Antithrombotic therapy should be considered for symptomatic thrombus or asymptomatic “floating” thrombus in the descending aorta.
117.	Elective endovascular repair should be considered as the first line surgical approach for patients with inflammatory arteritis of the thoraco-abdominal aorta.
120.	Urgent surgical repair of mycotic thoracic and thoraco-abdominal aortic aneurysms should be considered irrespective of aneurysm size.
121.	An individualised treatment strategy (open surgical repair, endovascular repair, conservative, or palliative management) for mycotic thoracic and thoraco-abdominal aortic aneurysms should be considered, based on urgency, anatomic considerations, surgical risk, causative pathogens, and patient preferences.
122.	Individualised antibiotic therapy for mycotic thoracic and thoraco-abdominal aortic aneurysms should be considered, with treatment duration ranging from four to six weeks to lifelong, depending on microbiology, surgical approach, and immune status.
125.	Patients with an aberrant subclavian artery and or Kommerell’s diverticulum should be considered for evaluation of associated structural abnormalities.
127.	Surgical treatment should be considered in patients with an aberrant subclavian artery and or Kommerell’s diverticulum who present with symptoms attributable to compression of adjacent structures, such as the oesophagus or trachea.
129.	The choice of surgical repair technique (open, hybrid, or endovascular repair) of an aberrant subclavian artery and or Kommerell’s diverticulum should be considered, individualised based on the treatment indication, anatomy, surgical risk, underlying pathology, and patient preferences.
New Class IIb recommendations	
11.	Transoesophageal echocardiography may be considered for use as an adjunctive intra-operative imaging modality during open or endovascular thoracic aortic repair.
12.	Intravascular ultrasound may be considered for use as an adjunctive intra-operative imaging modality for guidance during thoracic endovascular aortic repair.

Continued

Table 1-*continued*

17.	Extensive thrombosis "shaggy aorta" may be considered for intensified medical therapy with statins to stabilise the thrombotic lesions before aortic repair.
31.	Pre-flushing of the delivery system of the thoracic endograft may be considered in patients undergoing thoracic endovascular aortic repair to reduce the risk of cerebral air embolism.
57.	Distal false lumen occlusion may be considered as an adjunct to thoracic endovascular aortic repair in patients with chronic type B aortic dissection and aneurysm formation.
63.	Non-infected saccular descending thoracic aortic aneurysms may be considered for early surgical treatment with a lower diameter threshold for elective repair than for standard fusiform aneurysms.
68.	In endovascular thoraco-abdominal aortic repair, fenestrations may be considered preferred over branches for the renal arteries when the endograft is adjacent to the aortic wall at the level of the renal arteries.
70.	Balloon expandable, self expanding, and combinations of bridging covered stent designs may be considered for branches during thoraco-abdominal aortic repair, taking into account the bridging distance, target vessel anatomy, and properties of the bridging stent.
76.	For patients without an increased bleeding risk, temporary dual antiplatelet therapy may be considered after endovascular thoraco-abdominal aortic aneurysm repair with fenestrated or branched endografts to reduce thrombotic complications and improve target vessel patency.
78.	Pre-emptive embolisation of accessory renal arteries may be considered in selected cases before endovascular thoraco-abdominal aortic aneurysm repair.
84.	Compartmentalisation of a ruptured post-dissection thoraco-abdominal aortic aneurysm, by false lumen occlusion techniques or focal, controlled rupture of the dissection lamella, may be considered to enable endovascular exclusion of an isolated thoracic rupture.
85.	Open surgical repair may be considered in selected patients with a ruptured descending thoracic or thoraco-abdominal aortic aneurysm when endovascular repair is not feasible.
86.	Haemothorax drainage may be considered after endovascular repair of a ruptured descending thoracic aorta once the patient is haemodynamically stable.
89.	Thoracic endovascular aortic repair may be considered for ESVS grade 1 blunt thoracic aortic injury and presence of concomitant traumatic brain injury to permit higher cerebral perfusion pressures when strict blood pressure control is not feasible.
100.	Compromised or inadequate landing zones without visible endoleak after thoracic or thoraco-abdominal endovascular repair may be considered for re-intervention to improve seal.
101.	Type II endoleak after thoracic or thoraco-abdominal endovascular aortic repair may be considered for re-intervention in the presence of significant ($\geq$ 1.0 cm) aneurysm sac growth, after ruling out type I and III endoleaks.
113.	Endovascular intervention may be considered for symptomatic thrombus or asymptomatic "floating" thrombus in the descending aorta when medical management is unsuccessful.
126.	An incidentally detected, non-aneurysmal, asymptomatic aberrant subclavian artery may be considered for serial imaging surveillance at five year intervals, beginning at age 40 years.
128.	Surgical treatment for asymptomatic aberrant subclavian artery and or Kommerell's diverticulum may be considered when the transverse vessel diameter is $\geq$ 3.0 cm or when the combined distance between the edges of the diverticulum and the adjacent aorta is $\geq$ 5.5 cm.
New Class IIIa recommendations	
37.	Routine thoracic endovascular aortic repair is not indicated for uncomplicated acute type B aortic dissections outside clinical trials.
111.	Escalation of antithrombotic therapy is not indicated for asymptomatic mural thrombus in the descending thoracic aorta.
New Class IIIb recommendations	
19.	Endovascular or open surgical repair, whether elective or for ruptured thoracic or thoraco-abdominal aortic aneurysm, is not recommended in patients deemed unfit for repair or unlikely to benefit due to severe comorbidities, frailty, limited life expectancy, or those who decline intervention after shared decision making.
44.	Balloon moulding and or excessive oversizing of the thoracic endograft is not recommended in patients with acute type B aortic dissection as it increases the risk of proximal or distal stent graft induced new entry.
Changed Class I recommendations	
3.	Aortic centres treating descending thoracic aortic disease are recommended to have a yearly caseload of $\geq$ 20 repairs *(class upgraded from IIb)*
6.	Thin slice multiphase computed tomography angiography is recommended as the first line imaging modality for diagnosis, surveillance, pre-operative planning, and post-operative follow up of descending thoracic aortic pathologies *(class upgraded from IIa)*
7.	It is recommended that aortic imaging encompasses the entire aorta, from the supra-aortic vessels to the common femoral arteries *(class upgraded from IIa)*

Continued

Table 1-*continued*

23.	Protocolised strategies for the prevention, early detection, and management of spinal cord ischaemia are recommended for patients undergoing descending thoracic and thoraco-abdominal aortic repair *(class upgraded from IIa and rephrased)*
25.	Prophylactic cerebrospinal fluid drainage is recommended for patients undergoing open thoracic or thoraco-abdominal aortic repair *(class upgraded from IIa)*
36.	Prompt medical therapy, with pain control and anti-impulse therapy, and invasive haemodynamic monitoring are recommended as initial treatment for all patients with acute type B aortic dissection *(rephrased)*
40.	Patients with complicated acute type B aortic dissection are recommended for immediate thoracic endovascular aortic repair to cover the primary entry tear *(rephrased)*
46.	Individualised long term clinical follow up and serial cross sectional imaging is recommended after endovascular and open surgical treatment of acute type B aortic dissection, taking into account extent of disease and repair, imaging findings, patient fitness, and life expectancy *(rephrased)*
49.	Patients with chronic type B aortic dissection are recommended for life long antihypertensive therapy, with target blood pressure $<130/80$ mmHg, and surveillance with serial cross sectional imaging after six and 12 months and thereafter individualised as appropriate *(rephrased)*
50.	Beta blockers, angiotensin converting enzyme inhibitors, angiotensin receptor blockers, and or calcium channel blockers are recommended for long term antihypertensive medical treatment in patients with chronic type B aortic dissection *(rephrased)*
59.	Individualised long term clinical follow up and serial cross sectional imaging are recommended after endovascular and open surgical treatment of chronic type B aortic dissection, taking into account the extent of disease, repair, imaging findings, fitness, and life expectancy *(class upgraded from IIa and rephrased)*
64.	Thoracic endovascular aortic repair is recommended as the first line surgical treatment option in patients with descending thoracic aortic aneurysms *(class upgraded from IIa and rephrased)*
71.	Distal aortic perfusion is recommended during open surgical repair for thoracic and thoraco-abdominal aortic aneurysms to maintain end organ perfusion and reduce the risk of ischaemic complications *(class upgraded to from IIa)*
79.	Individualised long term clinical follow up and serial cross sectional imaging are recommended after thoracic and thoraco-abdominal aortic aneurysm repair, taking into account extent of disease, repair, imaging findings, fitness, and life expectancy *(class upgraded from IIa and rephrased)*
91.	Urgent thoracic endovascular aortic repair (<24 hours) is recommended for ESVS grade 2 blunt traumatic aortic injury presenting with high risk features *(rephrased)*
115.	High dose glucocorticoids combined with a disease modifying antirheumatic drug are recommended as first line treatment for active inflammation of the aorta and or its branches *(rephrased)*
123.	Surgical or endovascular treatment of aortic coarctation is recommended in adults with upper limb hypertension or left ventricular hypertrophy when a systolic pressure gradient ≥ 20 mmHg is present *(class upgraded from IIa and LoE upgraded from C to B)*
Changed Class IIa recommendations	
30.	Peri-operative monitoring of motor evoked potentials should be considered during open thoraco-abdominal aortic repair for early detection of spinal cord ischaemia *(class upgraded from IIb)*
54.	Thoracic endovascular aortic repair should be considered as the primary treatment option for patients with chronic type B aortic dissection and aneurysm formation confined to the thoracic aorta *(rephrased)*
65.	Endovascular repair with fenestrated and branched technologies should be considered as the first line surgical treatment option in patients with thoraco-abdominal aortic aneurysms *(rephrased)*
90.	Delayed thoracic endovascular aortic repair (>24 hours) should be considered for ESVS grade 2 blunt traumatic aortic injury without high risk features *(rephrased)*
99.	Direct (type I and III) endoleak after thoracic or thoraco-abdominal endovascular repair should be considered for re-intervention *(class downgraded from I)*
106.	Marfan syndrome patients with an aortic diameter ≥ 5.0 cm should be considered for descending thoracic aortic aneurysm repair *(rephrased)*
107.	Patients with Loeys–Dietz syndrome and an aortic diameter $>4.5-5.0$ cm should be considered for descending thoracic aortic aneurysm repair, taking into consideration diameter progression rate, age, family history, and pathogenic variants *(rephrased)*
108.	Patients with vascular Ehlers–Danlos syndrome with descending thoracic aortic pathologies should be considered for individualised surgical treatment decision making *(rephrased)*
109.	For preference, open surgical repair should be considered for elective treatment of thoracic aortic disease in patients with genetic aortopathies *(rephrased)*
110.	Endovascular aortic repair should be considered in patients with genetic aortopathies when open surgery is contraindicated, in high risk surgical candidates, in acute settings, or for redo procedures, preferably with landing zones in pre-existing surgical grafts *(class upgraded from IIb and rephrased)*

Continued

Table 1-*continued*
124. Endovascular treatment should be considered as first line therapy for adult patients with aortic coarctation, with stent choice (balloon expandable *vs.* self expanding, bare metal *vs.* covered stents) based on individual anatomic and clinical factors *(rephrased)*
Changed Class IIb recommendations
14. Pre-operative revascularisation of symptomatic or significant asymptomatic coronary artery disease may be considered in selected cases, particular before extensive thoracic and thoraco-abdominal aortic repair *(class downgraded from IIa and rephrased)*
26. A reactive (rescue) cerebrospinal fluid drainage strategy may be considered preferable to routine prophylactic drainage in patients undergoing extensive thoracic endovascular aortic repair *(class downgraded from IIa and rephrased)*
39. Selected patients with uncomplicated acute type B aortic dissection and suitable anatomy with early aortic expansion or large initial diameter (>4.0 cm) may be considered for thoracic endovascular aortic repair, preferably during the subacute phase *(class downgraded from IIa)*
58. Open surgical repair may be considered in selected patients at low surgical risk with chronic type B aortic dissection and thoraco-abdominal aortic aneurysm formation *(class downgraded from IIa)*
66. Open surgical repair may be considered in selected patients at low surgical risk with descending thoracic or thoraco-abdominal aortic aneurysms *(class downgraded from IIa and rephrased)*
Unchanged Class I recommendations
13. Pre-operative assessment of cardiac, pulmonary, and renal function is recommended before elective open or complex endovascular repair involving the descending thoracic aorta to allow for informed decision making and pre-operative optimisation.
47. Patients with uncomplicated penetrating aortic ulcer or intramural haematoma of the descending thoracic aorta are recommended for conservative management, including best medical therapy and surveillance with serial cross sectional imaging.
92. Immediate thoracic endovascular aortic repair is recommended for ESVS grade 3 blunt thoracic aortic injury.
93. Thoracic endovascular aortic repair is recommended as the first line treatment for blunt thoracic aortic injury.
Unchanged Class IIa recommendations
21. Left subclavian artery revascularisation should be considered in patients undergoing elective thoracic endovascular aortic repair with intentional left subclavian artery coverage, taking into account the risk of end organ ischaemia and urgency of the procedure.
27. Re-implantation of major intercostal arteries should be considered during open thoraco-abdominal aortic repair to reduce the risk of spinal cord ischaemia.
45. Open surgical repair of complicated acute type B aortic dissection should be considered in selected patients unsuitable for endovascular repair.
48. Patients with complicated penetrating aortic ulcer or intramural haematoma of the thoracic aorta should be considered for thoracic endovascular aortic repair.
52. Patients with chronic type B aortic dissection and an aortic diameter ≥ 6.0 cm should be considered for repair, taking into account fitness, aneurysm anatomy, and patient preferences.
61. Patients with descending thoracic or thoraco-abdominal aortic aneurysms and an aortic diameter ≥ 6.0 cm should be considered for repair, taking into account fitness, aneurysm anatomy, and patient preferences.
116. Surgical treatment should be considered in selected patients with inflammatory arteritis of the thoraco-abdominal aorta and its branches, taking into consideration aneurysm size, evidence of end organ ischaemia, and inflammatory disease activity.
Unchanged Class IIb recommendations
53. A lower aortic diameter threshold (≥ 5.5 cm) for repair may be considered in selected patients with chronic type B aortic dissection.
62. A lower aortic diameter threshold (≥ 5.5 cm) for repair may be considered in selected patients with descending thoracic or thoraco-abdominal aortic aneurysms.

1. METHODOLOGY

1.1. Purpose of the guidelines

The European Society for Vascular Surgery (ESVS) has developed clinical practice guidelines for the care of patients with diseases of the descending thoracic aorta (DTA) and thoraco-abdominal aorta (TAbdAo), in succession to the 2017 version,[1] with the aim of assisting physicians in selecting the best management strategy.[3]

Potential users of these guidelines include any physician involved in the management of patients with DTA and TAbdAo diseases, such as vascular surgeons, vascular physicians and angiologists, primary care doctors, cardiologists, cardiovascular surgeons, interventional radiologists, and other healthcare professionals involved in the care of these patients, as well as health policy makers and industry. Furthermore, the guidelines aim to serve as an important source of unbiased information for the patient and their

relatives to optimise shared decision making (SDM) (see Chapter 11).

Guidelines promote standards of care, but are not a legal standard of care. They are a "guiding principle", and the care delivered depends on patient presentation, choice, comorbidities, and setting (techniques available, local expertise).

These guidelines are based on scientific evidence complemented by expert opinion on the matter. By summarising and evaluating the best available evidence, recommendations for the evaluation and treatment of patients have been formulated. The recommendations represent the general knowledge at the time of writing these guidelines, but technology and disease knowledge in this field may change rapidly; therefore, recommendations can become outdated. The ESVS aims to update the guidelines when important new insights into the management of diseases of the DTA and TAbdAo become available.

The ESVS 2026 clinical practice guidelines on the management of DTA and TAbdAo diseases are published in the *European Journal of Vascular and Endovascular Surgery* (EJVES) as an online open access publication, are perpetually freely available, as well as being free to access via the ESVS website. They are also available on a dedicated ESVS Guideline App (https://esvs.org/guidelines/).

1.2. Compliance with AGREE II standards

The Appraisal of Guidelines for Research and Evaluation (AGREE II) reporting standards for assessing the quality and reporting of practice guidelines[4] were adopted during preparation of the 2026 guidelines; a checklist is available as supplementary material in Appendix A.

1.3. Guideline Writing Committee

Guideline Writing Committee (GWC) members were selected by the GWC chairs and the ESVS Guideline Steering Committee (GSC) to represent clinicians involved in the management of patients with DTA and TAbdAo diseases. The GWC comprised 20 vascular and cardiovascular surgeons from 11 European countries.

The members of the GWC have provided disclosure statements of all relationships that might be perceived as real or potential sources of conflicts of interest. These disclosure forms are kept on file at the ESVS headquarters. GWC members received no financial support from any pharmaceutical, device, or industry body to develop the guidelines.

The ESVS GSC was responsible for the endorsement process of this guideline. All experts involved in the GWC have approved the final document. The guideline document underwent a formal external expert review process and was reviewed and approved by the ESVS GSC and by the EJVES. This document has been reviewed by 18 reviewers from 11 countries, including 11 members of the GSC and seven external reviewers.

1.4. Methodology

1.4.1. Strategy. The work on this guideline started in October 2020 but was delayed due to the COVID-19 (coronavirus disease 2019) pandemic and the subsequent restructuring of the GWC. The GWC had regular online meetings and a face to face meeting in Uppsala, Sweden, in August 2024 to review the wording and grading of each recommendation. If there was no unanimous agreement, discussions were held to decide how to reach a consensus. If this failed, then the wording, grade, and level of evidence was secured via a majority vote of the GWC members. After several online follow up meetings, the GWC was able to agree on a final set of recommendations in August 2025. The document underwent four external review rounds. The final version of the guideline was submitted in September 2025.

1.4.2. Literature search and selection. A systematic literature search for relevant papers published up to June 2025 was performed in PubMed (MEDLINE), Embase, and the Cochrane Library. No language constraints were applied. Reference checking and hand search by the GWC members added other relevant literature.

The members of the GWC performed the literature selection based on information provided in the title and abstract of the retrieved studies. Only peer reviewed publications were included, following the pyramid of evidence principle. Multiple randomised controlled trials (RCTs) or meta-analyses of multiple RCTs were at the top, then single RCTs or large non-randomised studies (including meta-analyses of large non-RCTs), followed by meta-analyses of small non-RCTs, observational studies, case series, and large prospective audits. Expert opinion was at the bottom of the pyramid, while case reports and abstracts were excluded.

1.4.3. Recommendations. The ESVS clinical practice guidelines recommendation grading system[5] was used for grading the strength (class) of each recommendation, graded from I to III (Table 2), and the level of evidence, graded from A to C (Table 3).

Table 2. European Society for Vascular Surgery (ESVS) clinical practice guidelines recommendation grading system: class of recommendations.[5]

Class	Definition	Wording
I	Evidence and or general agreement that a given treatment or procedure is beneficial, useful, effective	is or are recommended
II	Conflicting evidence and or divergence of opinion about the usefulness or effectiveness of the given treatment or procedure	
IIa	Weight of evidence or opinion is in favour of usefulness or effectiveness	should be considered
IIb	Usefulness or effectiveness is less well established by evidence or opinion	may be considered
III	Evidence or general agreement that a given treatment or procedure is not useful or effective, and in some cases may be harmful	
IIIa	The given treatment or procedure is not necessarily useful or effective	is or are not indicated
IIIb	The given treatment or procedure may be dangerous or harmful to patients	is or are not recommended

Table 3. European Society for Vascular Surgery (ESVS) clinical practice guidelines recommendation grading system: levels of evidence.[5]

Level of Evidence A	Data derived from multiple randomised trials or meta-analyses of randomised trials
Level of Evidence B	Data derived from a single randomised trial, high quality* non-randomised studies, or meta-analysis of such studies
Level of Evidence C	Consensus opinion of experts, data from low quality† studies, or meta-analysis of such studies

* Large prospective, population based, observational, or registry studies.
† Small, retrospective studies or case series.

The supporting text aims to provide a summary basis for the need for and classification of recommendations. Described differences, effects, etc., are always significant unless otherwise stated, although confidence intervals or *p* values are not always stated. For more details, the readers are referred to the Table of Evidence (ToE) (Appendix A) or the cited reference.

1.4.4. Limitations. These guidelines have important limitations affecting generalisability. The general lack of robust evidence from prospective studies and RCTs with appropriate follow up observations is an unfortunate reality, for both open and endovascular DTA and TAbdAo repair. The rapid technological development in the field makes it difficult to obtain the necessary long term data for different devices and methods. The lion's share of the available evidence originates from highly developed socio-economic societies, while conditions in low and medium income countries are not covered. Other conditions that may require adaptation are large geographic referral area, inaccessibility of certain products, devices, and apparatus, social deprivation, and poverty. These factors must be kept in mind when managing other target groups or when operating in other settings and environments.

Aortic disease occurs more frequently in men, and current guidelines, while well intentioned, may not fully apply to women, who remain under represented in most trials and studies. Similarly, participants are often predominantly white males, limiting the generalisability of findings across different racial and ethnic groups.

There was no formal evaluation of facilitators and barriers, and the guidelines did not have the scope to go into detail regarding health economics, largely because individual countries have different processes for determining cost acceptability and different insurance and healthcare provider structures, pricing levels, and economic incentives, which makes costs largely incomparable.

As in the previous version of this guideline,[1] pathologies affecting the ascending aorta and the aortic arch are not discussed, with reference to other recently published dedicated guidelines and ESVS consensus document.[6,7]

The chapter on blunt thoracic aortic injury (BTAI) focuses specifically on aspects relevant to vascular surgical treatment of the DTA. For a more comprehensive overview of vascular trauma management, readers are referred to the ESVS 2024 clinical practice guidelines on the management of vascular trauma.[2]

For guidance on aortic graft and endograft infections, the reader is referred to the ESVS 2024 clinical practice guidelines on the management of abdominal aorto-iliac artery aneurysms[8] as well as the ESVS 2020 clinical practice guidelines on the management of vascular graft and endograft infections[9] and its pending 2026 update.

The chapter on genetic aortopathy focuses on the vascular surgical management of DTA/TAbdAo pathologies. For broader clinically relevant information on genetic and internal medicine aspects, the reader is referred to other sources.[10,11]

1.4.5. The patients' perspective. A key aim of these guideline is to optimise SDM. This requires access to unbiased evidence based information regarding all available treatment options, together with a balanced discussion of risks, benefits, and potential consequences in a manner the patient understands and that takes his or her preferences, needs, and values into account.

In order to improve accessibility and interpretability for patients and the public, a plain English summary of these guidelines was subjected to a lay review process. Men and women with a history of DTA or TAbdAo disease were provided with the drafted plain language summary of the guideline document, which they were advised to read before attending an educational event. At that event, the diagnostics, management, and outcomes of treatments for common DTA diseases were discussed. They were then invited to attend a focus group meeting, where the guideline development process was explained and specific aspects of the guideline were discussed, such as focus on patient orientated outcomes and methods to enhance accessibility and interpretability. Issues such as clarity, consistency, and simplicity in the presentation and recommendations that have been raised previously in earlier ESVS guideline developments were also addressed. Information gathered was used to inform the guideline development process and or the formation of the recommendations.

2. SERVICE STANDARDS

DTA diseases are relatively rare.[12] While the presentations of DTA pathologies may involve various specialties (e.g., acute care medicine, cardiology, surgery), most physicians are likely to manage few patients with these very rare pathologies over their career. On the other hand, for centres focusing on aortic disease, management of DTA diseases is an integral part of practice. Another challenge in the management of DTA pathologies is the relative scarcity of high level evidence in this field. These aspects may have implications for the organisation of services provided for management of DTA pathology, as discussed in this chapter.

2.1. Resources, availability, and surgical volume

DTA pathology may range from incidental findings with no immediate clinical relevance to life threatening emergencies requiring urgent surgical intervention. These cases are often first detected at centres with limited experience in managing aortic disease, where timely action is critical. Well established referral networks and protocols for rapid transfer to specialised aortic centres can improve outcomes in acute cases. The UK's centralised "hub and spoke" model for aortic surgery, which has led to improved results in abdominal aortic aneurysm (AAA) repair, illustrates this approach.[13] These networks also help to ensure that incidental findings and follow up decisions are managed by experienced specialists.

Although no universal definition exists, an "aortic centre" in the context of these guidelines refers to a facility with round the clock vascular surgical expertise capable of providing both medical, endovascular, and open surgical care of DTA pathology. Additional essential competencies include advanced cardiovascular imaging, cardiology and cardiothoracic surgery for cardiac and proximal aortic disease, vascular anaesthetics and intensive care, clinical genetics for genetic aortopathy, and specialised angiology and hypertension management.

Not all patients with acute DTA pathology require immediate surgery, e.g., those with uncomplicated intramural haematoma (IMH) or type B dissections (see Chapter 4). In such cases, the need for transfer to an aortic centre is often debated. While data are limited, one study suggests improved survival outcomes for aortic dissection (AD) managed at high volume centres.[14] Given the potential for rapid deterioration even in initially uncomplicated cases, access to urgent surgical expertise may be beneficial. The decision to transfer should be guided by clinical judgment and SDM with the patient.

A clear volume–outcome relationship has been demonstrated across numerous surgical procedures, including those for DTA disease.[15–18] This association is particularly evident in thoraco-abdominal and open repairs, whereas the data are less robust for standard thoracic endovascular aortic repair (TEVAR). Several national studies have demonstrated improved outcomes for endovascular thoraco-abdominal aortic aneurysm (TAAA) repair at high volume centres, despite treating older patients with more complex anatomy. US,[19] German,[20] and Australian[17] registry studies all show lower morbidity and mortality rates in high volume hospitals and among high volume surgeons. Furthermore, a population based study from Canada, including 664 patients undergoing TAAA repair, reported improved long term outcomes in high volume institutions.[21] Although no precise threshold can be defined, centres performing fewer than 20 DTA repairs annually appear to have inferior outcomes; the GWC therefore considers this a reasonable minimum surgical volume threshold for now, including DTA, TAbdAo, and arch repair.

The optimal timing for intervention in DTA pathology has not been well studied. However, by analogy with AAA and other high risk conditions (e.g., malignancies), an upper limit of eight weeks from decision to treatment is considered reasonable for standard elective DTA repair.[8] For high rupture risk cases, such as large aneurysms (>7 cm), a shorter timeframe is warranted (for more information on high risk dissection and aneurysms, see Chapters 5.2 and 6.2). Conversely, in patients with significant comorbidities or complex anatomy, delayed repair may be appropriate to allow for work up and medical optimisation and planning, including custom graft preparation.[22]

Recommendation 1 — New

Management of descending thoracic aortic disease is recommended to be organised in defined networks with established referral pathways.

Class	Level	Reference
I	C	Consensus

Recommendation 2 — New

Patients with acute descending thoracic aortic disease should be considered for prompt transfer to aortic centres for comprehensive evaluation and management.

Class	Level	Reference
IIa	C	Consensus

Recommendation 3 — Changed

Aortic centres treating descending thoracic aortic disease are recommended to have a yearly caseload of ≥20 repairs.

Class	Level	Reference
I	C	Consensus

Recommendation 4 — New

A fast track vascular surgical care pathway* is recommended for patients with a descending thoracic aortic pathology who meet the size criteria for elective repair.

Class	Level	Reference
I	C	Consensus

* An eight week pathway is a reasonable upper limit from referral to elective treatment, while a shorter timeframe should be considered for high risk disease (e.g., >7.0 cm aneurysms), and a lengthier planning or work up time may be justified for more complex pathology or comorbid patients.

2.2. Registries and quality control

Much of the evidence guiding the management of DTA disease is derived from registry based cohort studies. Device specific registries, such as the Global Registry for Endovascular Aortic Treatment (GREAT) (W.L. Gore Medical, Phoenix, AZ, USA) and the Medtronic Thoracic

Endovascular Registry (MOTHER) (Medtronic, Santa Rosa, CA, USA), have been instrumental in evaluating TEVAR practice.[23] Multicentre disease specific registries such as the International Registry of Acute Aortic Dissection (IRAD) also provide valuable data for DTA management.[24] National and regional quality registries increasingly include DTA, and collaborative efforts such as VASCUNET and the International Consortium of Vascular Registries have established TEVAR outcome benchmarks and proposed a core dataset to harmonise quality measures (Table 4).[25] These registries support outcome monitoring and quality improvement, benefiting both patients and clinicians. However, a standardised core outcome set and validated patient reported outcome measures for DTA repair are lacking and represent key priorities for future research.[29]

Recommendation 5		New
Aortic centres performing descending thoracic aortic repair are recommended to enrol cases in validated prospective quality registries to enable systematic monitoring of practice and clinical outcomes.		
Class	**Level**	**Reference**
I	C	Consensus

3. GENERAL CONSIDERATIONS

3.1. Imaging

3.1.1. Computed tomography angiography. Computed tomography angiography (CTA) is the first line imaging modality for diagnosis, surveillance, pre-operative planning, and post-operative follow up of aortic pathologies. It offers rapid and detailed visualisation of the entire aorta and surrounding structures and is widely accessible.[30–34]

An aortic CTA examination should extend from the supra-aortic vessels to the common femoral arteries[35] using thin slice (typically ≤ 1 mm) acquisition for multiplanar and 3D image reconstruction.[36] In cases with supra-aortic vessel involvement, imaging should include the circle of Willis to assess stroke risk and to guide potential pre- or intra-operative revascularisation strategies.[37]

CTA provides a detailed dataset of the TAbdAo and access vessels, which can be reconstructed into 3D models using dedicated post-processing software. These models can be registered and overlaid with real time fluoroscopy to enable image fusion, offering 3D guidance during complex endovascular procedures. Image fusion has been shown to significantly reduce contrast volume, fluoroscopy time, and total procedure time in complex endovascular aortic repair (EVAR).[38] A meta-analysis reported that image fusion reduced contrast use by 79 mL, fluoroscopy time by 14 minutes, and overall procedure time by 52 minutes in complex EVAR cases.[39] Image fusion is recommended in the ESVS 2023 clinical practice guidelines on radiation safety to reduce radiation exposure during aortic endovascular procedures.[40]

3.1.2. Magnetic resonance imaging. Magnetic resonance imaging (MRI) allows for high quality, multiplanar evaluation of the entire aorta, with similar diagnostic capabilities as CTA but without ionising radiation or, in some cases, contrast agents. Given the cumulative radiation exposure and risks associated with iodinated contrast, MRI should be considered an alternative imaging modality for long term surveillance, particularly in younger patients.[41]

Gadolinium based contrast agents carry a very low risk ($< 0.1\%$) of nephrogenic systemic fibrosis, even in patients with renal impairment.[42] When contrast is contraindicated, non-contrast magnetic resonance angiography (MRA) techniques are preferred, especially in pregnant or renally impaired patients.[36]

The presence of metal implants may render MRI unsafe or significantly reduce image quality due to artefact formation. Additional limitations include longer acquisition times, contraindications in claustrophobic patients, and limited availability, particularly in emergency settings.[36]

Four dimensional flow MRI (4D flow MRI) is a non-invasive technique for assessing aortic haemodynamics. It enables detailed analysis of jet angles, wall shear stress, and volumetric blood flow throughout the vessel, including post-TEVAR.[43] However, its clinical role remains to be defined.

3.1.3. Ultrasound. Transthoracic echocardiography (TTE) allows evaluation of the aortic root and proximal ascending aorta but is not useful for assessing DTA pathology.[44]

Transoesophageal echocardiography (TOE) provides better visualisation of the thoracic aorta and can be useful intra-operatively, particularly during open or endovascular repair of ADs.[44–47]

Intravascular ultrasound offers 360° visualisation of the aorta and its branches and may assist during TEVAR to confirm true lumen (TL) wire passage, assess false lumen (FL) depressurisation, evaluate TL expansion, and guide real time aortic sizing.[48,49]

3.1.4. Positron emission tomography. Fluorine-18 fluorodeoxyglucose positron emission tomography combined with computed tomography (^{18}F-FDG-PET/CT) detects increased metabolic activity by measuring uptake of ^{18}F-FDG, a glucose analogue. It is a valuable tool for identifying increased metabolic activity associated with vascular graft infection and aortic infection and inflammation.[50,51]

For detailed guidance on aortic graft and endograft infections, refer to the ESVS 2024 clinical practice guidelines on the management of abdominal aorto-iliac artery aneurysms[8] as well as the ESVS 2020 clinical practice guidelines on the management of vascular graft and endograft infections,[9] with an updated version expected in 2026.

Recommendation 6		Changed
Thin slice multiphase computed tomography angiography is recommended as the first line imaging modality for diagnosis, surveillance, pre-operative planning, and post-operative follow up of descending thoracic aortic pathologies.		
Class	**Level**	**Reference**
I	C	Consensus

Table 4. Proposed core dataset for standardised thoracic endovascular aortic repair (TEVAR) data collection (adapted from Hellgren *et al.*[25] and de Borst *et al.*[26])

Parameter	Data and units
Demographics and risk factors	
Country	Country name
Age	0–120 years
Sex	Number (%) men/women
Mode of admission	Elective/emergency
Diabetes mellitus	Yes/no
Cardiac disease	Yes/no
Lung disease	Yes/no
Stroke or TIA	Yes/no
Creatinine level (eGFR)	μmol/L (mL/min)
ASA grade	1–5
Indication for TEVAR	Dissection, aneurysm, trauma, or other
Dissection	
Time from onset of symptoms	Acute (1–14 days)/subacute (15–90 days)/chronic (>90 days)
Date of onset of symptoms	YYYY-MM-DD
Time from dissection to treatment	Number of days
Anatomic classification*	Type A(D)/type B(P, D)/type I(D)
Indication for TEVAR	Visceral ischaemia/renal ischaemia/rupture/dilatation/refractory pain/extremity ischaemia/other
Maximum aortic diameter	mm
Aneurysm	
Aortic pathology	Degenerative/mycotic/aortic ulcer/other
Intact or rupture	Intact/rupture
Maximum aortic diameter	mm
Trauma	
Time from trauma to treatment	Number of days
Trauma severity grading†	Grade 1–3
Operative data	
Operation date	YYYY-MM-DD
Stent grafts used	Manufacturer + device name + product number + unique device identifier number (for each stent graft)
Proximal landing zone‡	0–5
Distal landing zone‡	0–10
Non-revascularised covered branches	BA/LCC/LSA/CA/SMA/LRA/RRA
Revascularised covered branches	BA/LCC/LSA/CA/SMA/LRA/RRA
Prophylactic CSF drain	Yes/no
Post-operative (30 day) data	
TIA/stroke	None/non-disabling/disabling
Paraplegia	None/transient/permanent
Renal failure (requiring RRT)	Yes/no
Respiratory failure (requiring assisted ventilation)	Yes/no
Post-operative coronary event (myocardial infarction, dysrhythmia, cardiac failure)	Yes/no
Haemorrhage (requiring return to operating room)	Yes/no
Infection	Yes/no
Ischaemic bowel (requiring laparotomy)	Yes/no
Other complication(s)	Free text
Return to operating room	Yes/no
Death at 30 days	Alive/dead
In hospital death	Alive/dead
Death at 90 days	Alive/dead
Time from treatment to discharge	Number of days

TIA = transient ischaemic attack; eGFR = estimated glomerular filtration rate; ASA = American Society of Anesthesiologists; TEVAR = thoracic endovascular aortic repair; BA = brachiocephalic artery; LCC = left common carotid artery; LSA = left subclavian artery; CA = coeliac artery; SMA = superior mesenteric artery; LRA = left renal artery; RRA = right renal artery; CSF = cerebrospinal fluid; RRT = renal replacement therapy.

* Anatomic classification system proposed by Lombardi *et al.*[27]

† European Society for Vascular Surgery (ESVS) vascular trauma score.[2]

‡ Landing zones according to Fillinger *et al.*[28]

Recommendation 7	Changed
It is recommended that aortic imaging encompasses the entire aorta, from the supra-aortic vessels* to the common femoral arteries.	
Class	**Level**	**Reference**
I	C	Consensus

* In case of supra-aortic vessel involvement in the repair, the circle of Willis is to be included in imaging.

Recommendation 8	New
To reduce radiation exposure, magnetic resonance imaging should be considered as an alternative imaging modality for diagnosis, surveillance, and post-operative follow up of descending thoracic aortic pathologies, especially in younger patients.	
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 9	New
Flourine-18 fluorodeoxyglucose positron emission tomography integrated with computed tomography (^{18}F-FDG-PET/CT) should be considered an adjunctive imaging modality in the diagnostic evaluation of infected and non-infected aortitis of the descending thoracic aorta.	
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 10	New
Intra-operative image fusion should be considered during endovascular thoracic and thoraco-abdominal aortic repair to reduce radiation exposure, contrast volume, and operating time.	
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 11	New
Transoesophageal echocardiography may be considered for use as an adjunctive intra-operative imaging modality during open and endovascular thoracic aortic repair.	
Class	**Level**	**Reference**
IIb	C	Consensus

Recommendation 12	New
Intravascular ultrasound may be considered for use as an adjunctive intra-operative imaging modality for guidance during thoracic endovascular aortic repair.	
Class	**Level**	**Reference**
IIb	C	Consensus

3.2. Risk assessment and optimisation

Aortic surgery is considered a high risk procedure, with major adverse cardiovascular events occurring in over 5% of cases.[52,53] Cardiovascular morbidity and mortality following DTA repair are influenced both by patient and procedure related factors, including centre experience (see Chapter 2), anaesthetic strategy, surgical approach (endovascular *vs.* open), and intervention timing (Table 5).[21,52,54] In a US Aortic Research Consortium study of 2 099 patients undergoing elective fenestrated and branched endovascular aortic repair (FBEVAR) (709 complex AAA, 777 TAAA I—III, 580 TAAA IV—V), independent predictors of one year death included age >75 years, current smoking, chronic obstructive pulmonary disease (COPD), heart failure, creatinine >1.7 mg/dL, haematocrit <36%, TAAA extent I—III, and maximum aortic diameter >7 cm.[55]

Patients with DTA pathology frequently have significant comorbidities. A thorough evaluation, including medical and family history, cardiovascular risk factors (hypertension, dyslipidaemia, diabetes, obesity, smoking), symptom assessment, and physical examination, is essential.[52] Baseline laboratory testing should include full blood count, electrolytes, renal and liver function. Pre-operative

Table 5. Definitions of timing of surgery.[52,53]

Timing	Definition
Immediate	Immediate threat to life, limb, or organ without surgical intervention (<2 hours)
Urgent	Repair should be performed without unnecessary delay to save life, limb, or organ function, but there may be time for pre-operative clinical evaluation to allow interventions that could reduce MACEs or other post-operative complications (>2 to <24 hours)
Time sensitive	Repair should be performed as soon as possible as there is a time dependent risk of losing life, limb, or organ function. Time sensitivity varies depending on the underlying disease but allows pre-operative evaluation and management without negatively impacting outcomes (<3 months)
Elective	Repair can be performed electively without significant risk of losing life, limb, or organ function, and permits a complete pre-operative evaluation and appropriate management.

MACE = major adverse cardiovascular event.

assessment of cardiac, pulmonary, and renal function is recommended to guide patient selection and reduce peri-operative risk.

3.2.1. Timing of repair. Early death is significantly more common in urgent or emergency TAAA repair than for elective procedures. In a single centre, retrospective analysis of 3 309 patients undergoing open surgical repair (OSR) for Crawford extent I — IV TAAA, the operative mortality rate was 6.2% following elective repair *vs.* 21.8% for ruptured cases.[56] Similarly, in a series of 209 patients with extent I — III TAAA treated with FBEVAR using the Zenith platform (Cook Medical, Bloomingdale, IN, USA), the 30 day mortality rate was 4.6% after elective repair *vs.* 28.5% following urgent repair. A multicentre study of 100 patients with ruptured TAAA treated endovascularly also reported a 24% early mortality rate.[57] These data underscore the importance of timely elective intervention to avoid the high mortality rate associated with emergency repair (see Chapter 7).

3.2.2. Extent of aortic aneurysmal disease. The extent of TAAA strongly influences outcomes. In a large retrospective analysis, early death after OSR varied by Crawford extent: 5.9% for type I, 9.5% for type II, 8.8% for type III, and 5.4% for type IV.[56] Similarly, a pooled analysis from eight prospective, non-randomised, physician sponsored investigational device exemption studies conducted between 2005 and 2020 demonstrated that patients with an extensive TAAA had a higher risk of late aortic related death (defined as any early or late death due to rupture, dissection, malperfusion, complications, re-interventions, or other aortic related causes) compared with those with extent IV disease.[58]

3.2.3. Underlying aortic pathology. Patients undergoing OSR for post-dissection aneurysms, whether acute or chronic, tend to have more favourable outcomes than those with degenerative aneurysms, probably reflecting younger age, fewer comorbidities, and a higher prevalence of heritable aortic disease.[59] In contrast, patients with degenerative aneurysms are generally older and more burdened by risk factors such as hypertension, dyslipidaemia, diabetes, renal impairment, coronary artery disease (CAD), and pulmonary dysfunction, contributing to higher peri-operative morbidity and mortality rates.[55,60] Analysis of the Vascular Quality Initiative (VQI) database (2014 — 2021) found that patients treated with complex endovascular repair for post-dissection aneurysms ($n = 123$) were on average a decade younger, more often Afro-American, more frequently symptomatic, and had higher rates of hypertension, anaemia, and chronic kidney disease than those treated for degenerative aneurysms ($n = 3\,635$). Despite the greater extent of disease and more frequent previous aortic interventions, the 30 day mortality rate was similar (7.3%) between groups, probably due to the differing baseline risk profiles.[61]

3.2.4. Shaggy aorta. The shaggy aorta is characterised by "extensive atheromatous disease with diffuse ulcers associated with soft, loosely held debris, and a paucity of actual thrombus within the aorta" (Fig. 1).[62] Shaggy aorta is a recognised independent risk factor for diffuse embolisation as well as increased post-operative morbidity and mortality following TAAA repair, regardless of the treatment approach.[63—65] In a multicentre, retrospective, observational cohort study including 255 patients undergoing TEVAR, shaggy aorta was strongly associated with ischaemic organ complications (odds ratio [OR] 12.1) and by extension increased 30 day death (OR 3.6).[66] In a single centre study of 497 patients undergoing OSR for TAAA, shaggy aorta was associated with a higher risk of post-operative embolic complications (28.1% *vs.* 8.8%), renal failure (21.1% *vs.* 5.3%), and in hospital death (14% *vs.* 3.5%).[64]

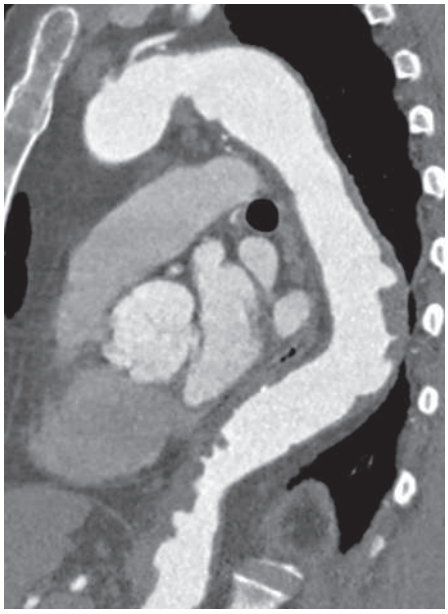


Figure 1. Shaggy aorta. Sagittal computed tomography angiography (CTA) reconstruction demonstrating extensive aortic wall irregularity with multifocal intramural thrombus deposits along the thoracic and abdominal aorta, characteristic of a "shaggy" aorta appearance (artist and copyright Carlota F. Prendes, Uppsala, Sweden).

Several scoring systems based on pre-operative CTA have been proposed to quantify shaggy aorta. One method evaluates axial slices every 5 mm (excluding the aneurysmal segment), assigning one point per slice with (1) ulcer like thrombus, (2) thrombus ≥ 5 mm thick, and (3) involvement of $> 2/3$ of the aortic circumference. The total "shaggy score" correlates with embolic risk.[67] Another system scores five thrombus characteristics: (1) location, (2) type, (3) thickness, (4) surface area, and (5) circumferential extent. Each parameter is scored from 0 to 2, yielding a total score maximum score of 10. In 212 patients undergoing FBEVAR, adverse events occurred in 53% of those with moderate or severe thrombus *vs.* 35% with mild disease.[63]

In patients with shaggy aorta, repair should be approached with caution, and intensified medical therapy is advised to stabilise the thrombus pre-operatively. There is, however, little evidence regarding the medical management of shaggy aorta. A small retrospective analysis of 27 patients

found a significant reduction of shaggy aorta after a mean 771 days of statin administration.[68] In a retrospective study of 519 patients with shaggy aorta, statins reduced the stroke rate by up to 70%, being superior to antiplatelet drugs, which did not show a protective effect on the incidence of stroke and other embolic events.[69] More evidence on surgical decision making and medical management of patients with shaggy aorta is needed.

3.2.5. Comorbidities. Chronic kidney disease is a major predictor of adverse outcomes after both open and endovascular TAAA repair. In patients with chronic kidney disease undergoing OSR for type II TAAA, higher rates of adverse events (27% *vs.* 18.6%), dialysis (14.1% *vs.* 8.7%), cardiac complications (42.8% *vs.* 33.4%), and life altering events (18.1% *vs.* 9.6%) were reported compared with matched controls.[70] Similarly, TEVAR registry data ($n = 2\,995$) showed a stepwise increase in post-operative acute kidney injury with worsening renal function: estimated glomerular filtration rate (eGFR) > 90 mL/min, 3%; $60 - 90$ mL/min, 10%; $15 - 30$ mL/min, 23%; < 15 mL/min, 25%.[71] Medicare data also linked chronic kidney disease stage III – V and end stage renal disease with higher 30 day mortality and serious complication rates.[72] Pre-operative hydration and preserving renal perfusion are essential preventive strategies.

COPD and smoking increase the risk of complications after elective TAAA OSR. Two large retrospective studies reported higher rates of respiratory complications (38.4% *vs.* 30%), operative mortality (7.9% *vs.* 3.8%), and adverse events (14.9% *vs.* 9.8%) in patients with COPD.[73,74] Data for endovascular repair are limited. Pulmonary function testing is essential to assess respiratory reserve; a forced expiratory volume in 1 second (FEV_1) $\leq 50\%$ is associated with a nearly sevenfold increased risk of major adverse events after OSR. Global Initiative for Chronic Obstructive Lung Disease (GOLD) staging may guide pre-operative risk assessment.[74] Pulmonary optimisation, including smoking cessation, treatment of bronchitis, weight loss, and prehabilitation, should begin one to three months before surgery, particularly in patients with severe obstruction. Data on the significance of COPD for endovascular repair are limited.

CAD is common in patients with TAAA, with the prevalence increasing by Crawford extent, from 28% in Crawford extent I to 49.5% in extent IV,[59] and is more frequent in complex endovascular repair than OSR (43.7% *vs.* 31.3%).[54] Comprehensive pre-operative cardiac evaluation is essential and should include electrocardiogram (ECG), echocardiography (to detect reduced ejection fraction, valvular regurgitation, and pulmonary hypertension, conditions poorly tolerated during aortic cross clamping), and cardiac biomarkers (e.g., high sensitivity cardiac troponin, brain natriuretic peptide, or N terminal pro-brain natriuretic peptide [NT-proBNP] to quantify myocardial injury and haemodynamic stress).[75,76] In selected patients, pre-operative revascularisation of symptomatic or significant asymptomatic CAD may reduce the risk of peri-operative myocardial infarction (MI) and improve outcomes, particularly for extensive surgery.[77] A single centre study showed coronary angiography and percutaneous coronary intervention with bare metal stents can be performed safely before TAAA repair without increasing the risk of bleeding, aneurysm rupture, stent thrombosis, MI, or death.[78]

Patients with unstable dysrhythmias, decompensated heart failure, severe valvular disease, pulmonary hypertension, or implantable cardiac devices should be evaluated by a multidisciplinary team to determine the need for further cardiac workup.[52,53,79]

Routine carotid screening before non-cardiac surgery is not recommended per current ESVS carotid guidelines.[80] However, screening is recommended in patients with a recent stroke or transient ischaemic attack (with six months). Carotid intervention is recommended before non-cardiac surgery in patients with symptomatic $50 - 99\%$ carotid stenosis, while prophylactic carotid intervention is not recommended in patients with asymptomatic carotid stenosis. These recommendations are based on cardiac surgery data, with limited evidence specific to DTA and TAbdAo repair.[80] In cases involving supra-aortic arterial reconstruction, cerebral circulation should be thoroughly assessed and any coexisting carotid disease managed individually.

3.2.6. Age. Many patients with degenerative atherosclerotic TAAA are of advanced age. In a single centre study, the operative mortality rate after OSR was markedly higher in octogenarians (19.2%) compared with patients aged ≤ 50 years (3.2%).[56] VQI data showed that patients aged ≥ 80 years undergoing FBEVAR were less often treated for extent II and more often for extent IV disease but experienced higher complication rates, more frequent discharge to rehabilitation or nursing care, and increased aortic specific death.[81] Chronological age alone, however, should not preclude intervention. Treatment decisions should be individualised based on aneurysm extent, patient comorbidities, and functional status, recognising that biological age and physiological reserve vary widely.

3.2.7. Functional capacity and frailty. Functional capacity, often measured in metabolic equivalents (METs), reflects a patient's ability to tolerate physiological stress. A threshold of < 4 METs suggests poor capacity and is commonly assessed by stair climbing ability or the Duke Activity Status Index (DASI; https://www.mdcalc.com/calc/3910/duke-activity-status-index-dasi#evidence). However, subjective METs have limited predictive value. In a multicentre study of 1 401 patients undergoing major non-cardiac surgery, only objective measures (DASI, NT-proBNP, and peak oxygen consumption [VO_2]) predicted 30 day death and myocardial injury.[22,82] Objective tools such as DASI and NT-proBNP may therefore be preferred for pre-operative risk stratification.

Frailty is a multidimensional decline in physiological reserve, which increases vulnerability to surgical stress.[52,83] Indicators include age > 70 years, low body mass index (BMI), anaemia, and poor nutritional status (e.g., albumin < 3.5 g/dL, $> 5 - 10\%$ recent weight loss, BMI < 22 kg/m^2).[84] In 1 089 patients undergoing FBEVAR, severe hypoalbuminaemia predicted higher 30 day (OR 5.0) and two year mortality

rates.[85] Impaired functional status, including daily and instrumental activities (e.g., bathing, shopping, medication management), strongly predicts poor outcomes.[60] Sarcopenia, assessed by lean psoas muscle area on pre-operative CTA, is a validated frailty marker in endovascular repair,[86,87] although its role in OSR remains uncertain.[88] The modified Frailty Index (mFI-11) effectively stratifies risk: in 592 FBEVAR patients, the 90 day mortality rate was 13% in high risk *vs.* 3% in low risk groups.[89] Prehabilitation may improve functional capacity, but evidence on the effect in DTA and TAbdAo repair is limited.[22]

Risk scoring based on patient and procedural factors aids patient selection and counselling.[90,91] However, SDM should also reflect patient values, including discharge goals and quality of life (QoL). This requires objective information, adequate time for discussion, and comprehensive pre-operative assessment.[6,92] No universal definition of surgical unfitness exists. In elective DTA cases, deferring repair due to limited life expectancy or frailty should be a multidisciplinary decision. A life expectancy of less than two to three years is a reasonable threshold.[8]

3.2.8. Lifestyle and medical management. Patients with DTA disease should adopt a healthy lifestyle, including a Mediterranean diet, regular exercise, and smoking cessation.[73,74,93] Exercise based cardiac rehabilitation is under evaluation.[93] Medical therapy should target cardiovascular risk factors, especially hypertension, statins, and antiplatelets in degenerative aneurysms due to coexisting atherosclerosis.[53,94,95] Statins may stabilise shaggy aorta lesions, although effects may require at least three months.[96] Targeted therapies apply in genetic aortopathy (see Chapter 10.1). Medical management is appropriate for asymptomatic patients below repair thresholds, unfit for intervention, or those opting for conservative care.[72]

Recommendation 13 — Unchanged

Pre-operative assessment of cardiac, pulmonary, and renal function is recommended before elective open or complex endovascular repair involving the descending thoracic aorta to allow for informed decision making and pre-operative optimisation.

Class	Level	References	ToE
I	C	Coselli *et al.* (2016),[59] Coselli *et al.* (2018),[70] Brown *et al.* (2020),[72] Girardi *et al.* (2017),[73] Orozco-Sevilla *et al.* (2024),[74] Girardi *et al.* (2014)[78]	

Recommendation 14 — Changed

Pre-operative revascularisation of symptomatic or significant asymptomatic coronary artery disease may be considered in selected cases, particularly before extensive thoracic and thoraco-abdominal aortic repair.

Class	Level	Reference
IIb	C	Consensus

Recommendation 15 — New

Multidisciplinary assessment of patient frailty should be considered for candidates for elective descending thoracic aortic repair.

Class	Level	References	ToE
IIa	C	Flanagan *et al.* (2021),[60] Paajanen *et al.* (2022),[89] Kärkkäinen *et al.* (2021)[97]	

Recommendation 16 — New

Assessment and optimisation of cardiovascular risk factors, best medical therapy, and a healthy lifestyle including smoking cessation are recommended for patients with descending thoracic aortic disease.

Class	Level	Reference
I	C	Consensus

Recommendation 17 — New

Extensive thrombosis "shaggy aorta" may be considered for intensified medical therapy with statins to stabilise the thrombotic lesions before aortic repair.

Class	Level	Reference
IIb	C	Consensus

Recommendation 18 — New

Patients with thoracic aortic disease are recommended to be fully informed about all treatment options (medical management, endovascular repair, or open surgery), as well as their short and long term risks, including their potential impact on function, independence, and quality of life, to support shared decision making.

Class	Level	Reference
I	C	Consensus

Recommendation 19 — New

Endovascular or open surgical repair, whether elective or for ruptured thoracic or thoraco-abdominal aortic aneurysm, is not recommended in patients deemed unfit for repair or unlikely to benefit due to severe comorbidities, frailty, limited life expectancy*, or those who decline intervention after shared decision making.

Class	Level	Reference
IIIb	C	Consensus

* A life expectancy of two to three years may be regarded a reasonable threshold.

3.3. Pregnancy and descending thoracic aorta disease

Pre-conception counselling by a multidisciplinary cardiovascular—obstetric team is recommended for women with DTA pathologies to assess risk and discontinue teratogenic drugs (e.g., angiotensin converting enzyme [ACE] inhibitors or angiotensin receptor blockers [ARBs]). ECG

gated CTA or MRA of the entire aorta should be performed before pregnancy. The risk of AD and aneurysm growth or rupture during pregnancy depends on aetiology and aortic size.[98,99] Elective repair may be advised before pregnancy in women with genetic aortopathy, given the high risk of pregnancy related complications (see Chapter 10.1). In an international prospective global registry including 170 women with known genetic aortopathy under specialised surveillance, the pregnancy related AD rate was 3.5%.[100] AD is associated with high maternal and foetal mortality rates (8.6% and 50%, respectively).[101]

Continuous monitoring by a multidisciplinary cardio-obstetric team (including vascular and cardiac surgery, anaesthesia, intensive care, and maternal—fetal medicine) is essential during pregnancy. Beta blockers are recommended for aortic dilation, and hypertension should be treated promptly. TTE is advised each trimester; if inadequate, non-contrast MRA may be used. AD typically occurs in the third trimester or postpartum. In the IRAD registry, half of pregnancy related AD cases had a known aortopathy, mainly Marfan syndrome (MFS).[102] Management of AD during pregnancy should follow standard protocols. Delivery planning should consider aortic size and haemodynamic risks, with earlier delivery indicated in cases with rapid aortic growth, valve disease, or hypertensive complications. Caesarean section is recommended for aortic dilation $>4.5-5.0$ cm or for untreated dissection; vaginal delivery may be considered for aorta $<4.0-4.5$ cm, based on underlying genetic pathology and SDM.[98,101,103] There appears to be no association between breastfeeding and postpartum complications.[100]

Postpartum imaging surveillance with TTE, CTA, or MRA is important, as the risk of dissection remains elevated for weeks after delivery. Surveillance frequency should be guided by aortic size, growth rate, and underlying aetiology.[98]

Recommendation 20		New
Pregnant women with descending thoracic aortic aneurysm or dissection are recommended to be referred to experienced aortic centres* for individualised multidisciplinary management by a cardiovascular—obstetric team.		
Class	**Level**	**Reference**
I	C	Consensus

* See Chapter 2.1 for definition of aortic centres.

3.4. Left subclavian artery revascularisation

Intentional left subclavian artery (LSA) coverage is often necessary during TEVAR to ensure an adequate proximal seal.[104] However, this may compromise perfusion to the brain, spinal cord, left arm, or myocardium (in patients with left internal mammary artery [LIMA] grafts), increasing the risk of stroke, spinal cord ischaemia (SCI), limb ischaemia, and MI. Pre-emptive LSA revascularisation can mitigate these risks but carries procedural risks such as stroke, nerve injury, and infection.[105] No RCTs have compared routine *vs.* selective *vs.* no LSA revascularisation during TEVAR. Meta-analyses of observational studies suggest that revascularisation reduces rates of stroke (OR 0.67) and SCI (OR 0.75) but not paraplegia or death.[106—110] This supports that patients undergoing elective TEVAR with intentional LSA coverage should be considered for LSA revascularisation. While LSA revascularisation may also be considered in the acute setting, the decision making is typically more complex and less straightforward. Current consensus supports revascularisation in high risk scenarios, such as dominant left vertebral artery, compromised right vertebral artery, incomplete circle of Willis, underdeveloped left vertebral artery ending in the posterior inferior cerebellar artery or isolated vertebral artery, LIMA graft, upper limb dialysis access, extensive aortic coverage, or aberrant right subclavian artery where both subclavian origins would be covered.

Revascularisation techniques include open surgery (carotid—subclavian or carotid—axillary bypass, subclavian transposition) or endovascular methods (branched and fenestrated endografts, chimney and periscope grafts, and *in situ* fenestration). Surgical methods offer durable patency but carry risks of local complications.[111—115] Endovascular options are less invasive but may increase endoleak risk, especially with parallel grafts (PGs).[116,117]

No head to head trials have compared surgical and endovascular LSA revascularisation during TEVAR. A meta-analysis of 14 retrospective studies (1 695 patients) found no significant difference in rates of stroke, SCI or upper limb ischaemia, or death between open surgical ($n=1\,204$) and endovascular ($n=491$) techniques, although PGs were associated with higher rates of type I endoleak.[111] Another meta-analysis of 28 studies (2 759 patients) reported similar patency across approaches, but higher rates of re-stenosis with endovascular repair. Subgroup analysis showed increased stroke, 30 day mortality, and endoleak with PGs compared with branched or fenestrated devices.[116]

In conclusion, the choice between routine and selective revascularisation, and between surgical and endovascular techniques, should be individualised based on patient anatomy, urgency, and overall risk profile. For elective endovascular LSA revascularisation, custom made or off the shelf fenestrated or branched devices are generally preferred if the anatomy is suitable, with alternatives including *in situ* laser or needle assisted fenestrations or physician modified endografts (PMEGs). In urgent settings, endovascular options include off the shelf fenestrated and branched devices, suitable custom made devices (CMDs) from other patients, *in situ* fenestrations, PMEGs, or PGs.

Recommendation 21		Unchanged
Left subclavian artery revascularisation should be considered in patients undergoing elective thoracic endovascular aortic repair with intentional left subclavian artery coverage, taking into account the risk of end organ ischaemia* and urgency of the procedure.		
Class	**Level**	**Reference**
IIa	B	Consensus

* Dominant left vertebral artery, compromised right vertebral artery, incomplete circle of Willis, left vertebral artery ending in the posterior inferior cerebellar artery, left arm dialysis access, patent pedicled left internal mammary artery bypass graft, and extensive aortic stent graft coverage or previous open or endovascular aorto-iliac surgery compromising the collateral supply to the spinal cord.

Recommendation 22		New
The choice between open and endovascular left subclavian artery revascularisation during thoracic endovascular aortic repair should be considered on a case by case basis, taking into account urgency, patient physiology, anatomy, underlying pathology, and patient preferences.		
Class	**Level**	**Reference**
IIa	C	Consensus

3.5. Spinal cord ischaemia

Spinal cord perfusion relies on a complex collateral network involving segmental, subclavian, iliac, and paravertebral vessels, which connect via the internal thoracic, epigastric, intercostal, and lumbar arteries (Fig. 2).[118] During aortic repair, this network may be disrupted by cross clamping, ligation, or stent graft coverage, increasing the risk of SCI.[120] Resulting hypoperfusion can trigger inflammation and neuronal death within 48 hours.[121]

SCI is a major complication after open and endovascular TAAA repair[122–124] with an incidence ranging from 2–20%, although rates have declined.[125,126] A systematic review reported SCI in 13.5% after endovascular repair and 7.4% after OSR. Risk factors include extensive aortic coverage, aortic cross clamping, prolonged surgery, blood loss, and hypotension.[124,127,128]

Intra- and post-operative measures, applied when appropriate, such as maintaining perfusion pressure and haemoglobin levels, preservation of collaterals, staged repair, cerebrospinal fluid drainage (CSFD), and minimising coverage, are supported by growing evidence; these strategies are being incorporated into structured SCI prevention protocols.[126,128–130] In a recent study of 562 patients undergoing FBEVAR, implementation of a dedicated SCI prevention protocol significantly reduced SCI rates, particularly among high risk patients,[124,128] where the incidence decreased from 23.2% to 5.0% ($p < .001$).[129]

3.5.1. Permissive hypertension and optimised haematocrit levels. Peri-operative blood loss and sustained hypotension are consistently associated with increased SCI risk.[124,127,128,131,132]

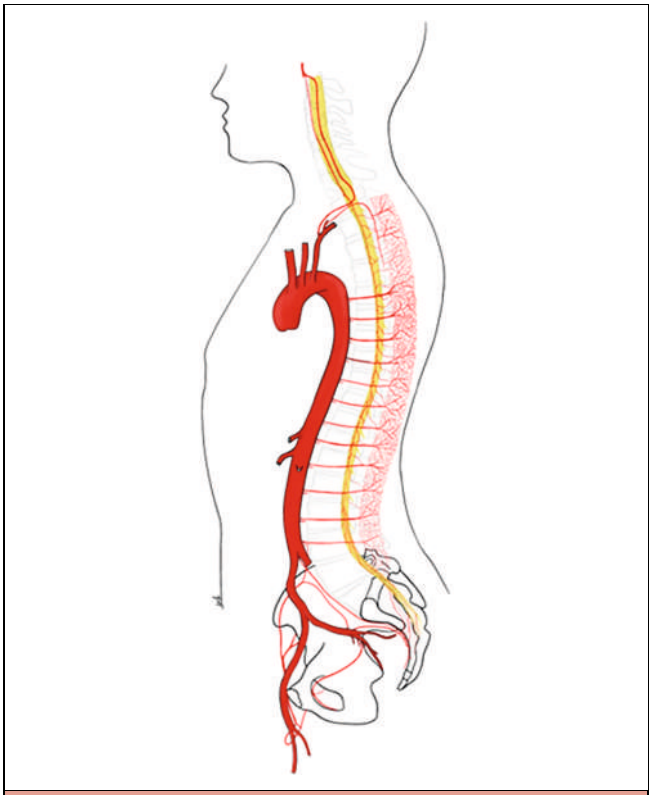


Figure 2. Spinal cord collateral circulation, involving the subclavian, hypogastric, intercostal and lumbar arteries.[119]

Maintaining adequate systemic blood pressure is critical, with suggested targets of systolic blood pressure (SBP) $\geq$ 120 mmHg and mean arterial pressure $\geq$ 90 mmHg peri-operatively, followed by permissive hypertension up to an SBP 150 mmHg for one month post-operatively.[129] Adjustment of antihypertensive therapy should be considered to minimise the risk of peri- and post-operative hypotension, specifically by withholding ACE inhibitors or ARBs for at least 48 hours before surgery and throughout the peri- and early post-operative period.[129]

Early blood transfusion to maintain a haemoglobin $\geq$ 10 g/dL during the first 48 hours may further reduce SCI risk after endovascular repair.[129]

3.5.2. Preservation of collaterals. To preserve spinal cord perfusion during open TAAA repair, selective re-implantation of intercostal arteries is frequently performed, guided by surgical judgement or intra-operative neuromonitoring (e.g., motor or somatosensory evoked potentials).

In endovascular repair, maintaining collateral flow via the LSA (see Recommendation #21 and #22 in Chapter 3.4) and pelvic vessels is crucial.[130] Revascularisation of major intercostal arteries ($\geq$ 4 mm) with stenting has been proposed for extensive TEVAR, although limited experience currently restricts its routine use. Early reperfusion of the pelvis and lower limbs can be achieved by withdrawing large sheaths from the iliac arteries immediately after deployment of the central device and before cannulation and extension to the visceral branches.[133] In selected cases

with inadequate iliofemoral access, temporary conduits may facilitate reperfusion by allowing retraction of the aortic sheath into the conduit.[126]

Staged intercostal artery occlusion may enhance collateralisation via the paraspinal network, according to the "spinal cord network concept".[120] The MIS^{2}ACE technique (minimally invasive staged segmental artery coil embolisation) has been proposed to reduce SCI risk but remains investigational pending trial results.[134]

3.5.3. Staged repair. A staged approach (deliberately delaying full aortic exclusion to allow temporary sac perfusion) has become a key strategy to reduce SCI during endovascular TAAA repair.[135] By promoting collateral network development between stages, this technique decreases SCI risk, as supported by experimental models.[136,137] The staged approach also reduces overall surgical trauma and thereby the risk of peri-operative hypotension, a major contributor to SCI. Additionally, final stage procedures can often be performed under local anaesthesia, further minimising haemodynamic stress.[138]

In a large multicentre study of 1 947 patients treated with FBEVAR for extent I – III TAAA, a multistage strategy was associated with lower 30 day or in hospital mortality or permanent paraplegia (OR 0.47) and improved one and three year survival.[139] Permanent paraplegia occurred in 4.3% of multistage *vs.* 10% of single stage repairs ($p < .001$). However, staging delays full exclusion and may increase rupture risk. The optimal interval remains undefined and must be balanced against urgency.

3.5.4. Cerebrospinal fluid drainage. Cerebrospinal fluid pressure rises during aortic clamping, and lowering it improves spinal cord perfusion. In an RCT of 145 patients undergoing extent I – II TAAA open repair, prophylactic CSFD reduced permanent SCI from 13% to 3%, an 80% relative risk reduction.[125] This led to widespread adoption of prophylactic CSFD in open TAAA repair, supported by subsequent meta-analyses.[140,141]

Prophylactic CSFD has also been widely adopted in endovascular TAAA repair, despite limited supporting evidence.[142] Furthermore, the incidence of drain related complication ranges from 7 – 26%.[143,144] Consequently, many aortic centres have now discontinued the routine use of prophylactic CSFD during endovascular TAAA repair, citing the high rates of drain related complications and equipoise among patients treated without prophylactic drainage.[145] In a recent study of 541 patients undergoing extent I – III FBEVAR without prophylactic CSFD, the overall SCI rate was 8%, with rescue CSFD and permissive hypertension improving symptoms in 73%; permanent paraplegia occurred in only 2%.[131] Current evidence supports selective, reactive use of CSFD over routine prophylaxis during extensive TEVAR to reduce SCI risk while avoiding drain related complications. However, higher quality evidence is needed, and a recent pilot study demonstrated the feasibility of conducting an RCT to evaluate prophylactic *vs.* therapeutic CSFD for SCI prevention in endovascular TAAA repair.[146]

3.5.5. Neuromonitoring. Neuromonitoring with motor evoked potentials (MEPs) and somatosensory evoked potentials (SSEPs) provides real time assessment of spinal cord integrity during TAAA repair. MEPs monitor descending motor pathways and are more sensitive to early ischaemia, while SSEPs assess ascending sensory pathways and help differentiate SCI from limb ischaemia. Anaesthetic techniques should be tailored to preserve signal quality.[147] Loss of MEPs often signals reversible ischaemia; SSEP loss suggests more advanced, potentially irreversible injury. Signal changes warrant immediate interventions to restore spinal perfusion, including intercostal artery reattachment (T8 – T12), pelvic reperfusion, CSFD (< 10 mmHg), blood pressure elevation, haematocrit optimisation, and ensuring a distal perfusion pressure > 80 mmHg.[148]

While standard in open TAAA repair, neuromonitoring is less established in endovascular repair. Staged TEVAR under local anaesthesia allows clinical assessment, but in extensive single stage procedures under general anaesthesia, neuromonitoring may be useful. In a prospective study of 170 FBEVAR patients, MEP and SSEP changes occurred in 55%, prompting interventions and signal recovery in 90%; SCI occurred in 10% of patients with signal loss *vs.* 1% with normal signals.[126,149] A systematic review of 11 studies (1 069 patients) reported neuromonitoring sensitivity and specificity of 93% and 96%, respectively, for SCI detection during TEVAR and or FBEVAR.[149]

Early clinical neurological evaluation, either continuous in awake patients or via immediate wake up tests, is recommended after TAAA repair for prompt SCI detection.

Near infrared spectroscopy may offer a non-invasive alternative for spinal perfusion monitoring but remains investigational owing to limited validation.[150,151] Biomarker based detection of spinal ischaemia is also under investigation, but no clinically validated markers are available.[152,153]

Recommendation 23			**Changed**
Protocolised strategies* for the prevention, early detection, and management of spinal cord ischaemia are recommended for patients undergoing descending thoracic and thoracoabdominal aortic repair.			
Class	**Level**	**References**	**ToE**
I	C	Tenorio *et al.* (2022),[126] Dijkstra *et al.* (2018),[128] Aucoin *et al.* (2021),[130] Sickels *et al.* (2025)[154]	

* Staging the procedure, maintaining a high blood pressure and haemoglobin ≥ 10 g/dL, preservation or restoration of collaterals, cerebrospinal fluid drainage, and neuromonitoring, as appropriate.

Recommendation 24			New
Staged repair should be considered for patients undergoing extensive thoracic and thoraco-abdominal endovascular aortic repair.			
Class	**Level**	**References**	**ToE**
IIa	C	Kasprzak *et al.* (2014),[135] Dias-Neto *et al.* (2023),[139] Juszczak *et al.* (2019)[145]	

Recommendation 25			Changed
Prophylactic cerebrospinal fluid drainage is recommended for patients undergoing open thoracic or thoraco-abdominal aortic repair.			
Class	**Level**	**References**	**ToE**
I	A	Coselli *et al.* (2002),[125] Khan *et al.* (2012)[140]	

Recommendation 26		Changed
A reactive (rescue) cerebrospinal fluid drainage strategy may be considered preferable to routine prophylactic drainage in patients undergoing extensive thoracic endovascular aortic repair.		
Class	**Level**	**Reference**
IIb	C	Consensus

Recommendation 27		Unchanged
Re-implantation of major intercostal arteries should be considered during open thoraco-abdominal aortic repair to reduce the risk of spinal cord ischaemia.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 28		New
Preservation of flow to the internal iliac arteries should be considered during endovascular thoraco-abdominal aortic repair to reduce the risk of spinal cord ischaemia.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 29		New
Early* clinical neurological evaluation is recommended after open or endovascular thoracic or thoraco-abdominal aortic repair to enable prompt detection and treatment of spinal cord ischaemia.		
Class	**Level**	**Reference**
I	C	Consensus

* Continuously during the peri-operative period in the awake patient undergoing repair under local anaesthesia, or by immediately post-repair wake up test in the sedated patient.

Recommendation 30		Changed
Peri-operative monitoring of motor evoked potentials should be considered during open thoraco-abdominal aortic repair for early detection of spinal cord ischaemia.		
Class	**Level**	**Reference**
IIa	C	Consensus

3.6. Stroke

The risk of stroke during TEVAR is estimated at 3 — 5% for standard procedures and 10 — 20% for FBEVAR.[155—157] A meta-analysis confirmed a 4% stroke rate after endovascular descending thoracic aortic aneurysm (DTAA) repair, with a significantly lower risk when the LSA is preserved or revascularised. Stroke occurred in 12% of patients with LSA coverage without revascularisation, three times higher than in those with LSA preservation or revascularisation.[155] Additional risk factors include proximal landing in zones above Ishimaru zone 3 and native aortic landing zones (LZs).[158,159]

Stroke pathophysiology in TEVAR is multifactorial, involving embolism, haemodynamic compromise, and haemorrhage. Emboli can even originate distal to the LSA owing to retrograde diastolic flow into the vertebral and carotid arteries.[160] While solid emboli from atheroma and thrombus have long been implicated, growing evidence highlights the importance of air embolism. Air bubbles released from delivery systems can impair perfusion and trigger neuro-inflammation.[161] Transcranial Doppler studies show that > 90% of high intensity transient signals during TEVAR are gaseous and correlate with silent brain infarctions on diffusion weighted MRI.[162]

Preventive strategies include careful patient selection, minimising catheter and wire manipulation in the arch, and avoiding unnecessary LSA coverage. Cerebral protection devices (filters, clamps, balloons) have been explored[162—165] but may disrupt cerebral perfusion and increase complexity.

Flushing endografts with carbon dioxide (CO_2) followed by saline (rather than saline alone) significantly reduces air release from the delivery system in simulated settings. This method is compatible with current sheath constrained devices equipped with a flushing port[166,167] and may be considered during TEVAR. Data from the Carbon Dioxide Flushing versus Saline Flushing of Thoracic Aortic Stents (INTERCEPTevar) RCT are eagerly awaited.[168] For sleeve constrained thoracic endografts, air is removed from the device by allowing back bleeding during insertion (GORE TAG Conformable Thoracic Stent Graft instructions for use; W.L. Gore & Associates, Inc., Flagstaff, AZ, USA).

Recommendation 31		New
Pre-flushing* of the delivery system of the thoracic endograft may be considered in patients undergoing thoracic endovascular aortic repair to reduce the risk of cerebral air embolism.		
Class	**Level**	**Reference**
IIb	C	Consensus

* With carbon dioxide followed by flushing with saline solution for sheath constrained thoracic endografts, or with back bleeding for sleeve constrained thoracic endografts.

3.7. Vascular access

Endovascular repair of DTA and TAbdAo pathologies is typically performed via the common femoral artery (CFA) for retrograde device delivery. The advent of CMDs and off the shelf low profile FBEVAR devices has expanded the feasibility of complex EVAR in patients with hostile femoral or iliac access.[169] However, in patients with very small, calcified, or tortuous iliofemoral arteries, alternative approaches, such as direct iliac or aortic puncture, or surgical conduit creation may still be necessary. For complex repairs involving visceral branches, upper limb access via the brachial, axillary, or subclavian artery allows for antegrade branch cannulation. Rare access routes (e.g., carotid, transapical, ascending aorta, transcaval) have been described in arch pathologies but are beyond the scope of this chapter.[170]

Surgical CFA access is achieved via longitudinal or transverse groin incisions. A meta-analysis of five observational studies and two RCTs (5 922 incisions) found longitudinal incisions associated with higher risks of wound infection and dehiscence (risk ratio [RR] 2.9), with no difference in lymphatic or haematoma complications.[171] Similarly, a Cochrane review of two RCTs (283 incisions) showed a lower infection risk with transverse incisions (RR 0.25), but no difference in lymphatic complications.[172]

Surgical conduits are typically created via retroperitoneal access to the iliac artery or distal aorta using a 10 mm prosthetic graft tunnelled to the groin. A review of 14 studies (16 855 cases) reported 94% technical success with surgical conduits, but higher complication rates and longer hospital stays.[173] Endoconduits, combining stenting and balloon dilation, are an alternative and can be created concurrently with TEVAR or as a staged procedure. Limited data suggest that endoconduits may offer reduced morbidity and shorter hospitalisation compared with surgical conduits.[173] There is increasing interest in using intravascular lithotripsy for vessel preparation to facilitate large bore access in heavily calcified arteries.[174] Current evidence is, however, mainly derived from the transcatheter aortic valve replacement (TAVR) literature, and further studies are needed to establish its clinical efficacy in other settings.

Percutaneous CFA access, guided by ultrasound, fluoroscopy, and or landmarks, has become an established less invasive alternative to surgical access for endovascular aortic procedures. Ultrasound guidance improves first pass success and reduces haematomas and major vascular complications. While evidence mainly derives from coronary interventions, findings also support ultrasound guidance over traditional methods for aortic intervention. A meta-analysis of nine RCTs (2 361 patients) showed higher first pass success (OR 3.1) and lower haematoma risk (OR 0.41) with ultrasound *vs.* anatomic landmark technique, fluoroscopic guidance, or a combination thereof.[175] An individual participant data meta-analysis of four RCTs (2 441 patients) confirmed lower rates of major bleeding and vascular complications with ultrasound guidance.[176] A recent RCT in 544 patients undergoing large bore access for complex coronary procedures also showed improved first pass success with ultrasound (*vs.* fluoroscopy guidance), but no significant difference in major complications.[177]

Various vascular closure devices are used for percutaneous femoral arteriotomy closure and are broadly classified into (1) suture based, (2) collagen or plug based, and (3) membrane based systems. Suture based devices are most commonly used for large bore access. The Prostar XL (Abbott Vascular Inc., Santa Clara, CA, USA) is approved for 8.5 — 10 F access but is used off label up to 24 F. The Perclose ProGlide (Abbott Vascular Inc., Santa Clara, CA, USA), the most widely used for up to 26 F closure, typically requires two devices. Both allow suture mediated closure over a wire, enabling placement of an additional device if haemostasis is inadequate. The MANTA (Teleflex Inc., Wayne, PA, USA) collagen based device uses an intra-arterial bioresorbable polymer and an extravascular bovine collagen plug, available in 14 F and 18 F versions for closing arteriotomies between 10 — 14 F and 14 — 22 F, respectively. PerQseal (Vivasure Medical, Galway, Ireland) and InSeal (InSeal Medical, Waltham, MA, USA) are membrane based devices CE marked for closure of 14 — 24 F arteriotomies.

Comparative data on vascular closure devices largely stem from studies in TAVR, with limited evidence specific to endovascular repair of the DTA and TAbdAo. A systematic review and meta-analysis of two RCTs and eight observational studies (3 113 TAVR patients: MANTA, $n = 1\,358$; ProGlide/Prostar XL, $n = 1\,755$) found no significant difference in major access site complications, although collagen and plug based devices showed a non-significant trend toward more unplanned vascular interventions.[178] A network meta-analysis of two RCTs and 15 observational studies (11 344 TAVR patients) found MANTA and ProGlide had similar rates of major access complications and bleeding, while ProGlide was superior to Prostar in both outcomes.[179] RCTs and cohort studies have shown that the combination of suture—collagen plug vascular closure devices (one Perclose ProGlide + one Angio-Seal) is superior to suture only devices (dual Perclose ProGlide). A recent systematic review and meta-analysis of > 2 000 patients undergoing TAVR from six studies (two RCTs) found a significant reduction of vascular closure device failure (RR 0.14; $p < .0001$), bleeding complications (RR 0.35; $p < .0001$), major vascular complications (RR 0.42; $p < .0001$), and minor vascular complications (RR 0.54; $p < .0001$) in the combination group, while no difference was seen in all cause mortality (RR 0.54; $p = .0709$).[180] A similar finding was reported from a network meta-analysis of > 21 000 patients undergoing TAVR from 28 studies.[181] Thus, current evidence suggests that combining small bore collagen plug based and suture mediated closure devices may be the safest and most effective strategy for TAVR related vascular access. However, its applicability in the DTA and TAbdAo settings remains to be validated.

Data directly comparing percutaneous access with surgical femoral exposure in TEVAR or complex EVAR, procedures requiring large bore arteriotomies, remain limited. A meta-analysis of four RCTs and 13 observational studies

with a total of 7 889 access sites (ten studies reporting EVAR, one reporting TEVAR, three reporting both EVAR and TEVAR, and another three reporting TAVR) found percutaneous femoral access was associated with lower risk of seroma (OR 0.15), wound dehiscence (OR 0.14), and surgical site infection (OR 0.38), but a higher risk of pseudoaneurysm (OR 3.83).[182] Another meta-analysis of four RCTs involving 368 participants (530 access sites) compared surgical cutdown with total percutaneous access for elective EVAR. No significant differences in access site complications, infections, bleeding, arterial injury, femoral occlusion, pseudoaneurysm, hospital stay, or peri-operative mortality were found. However, seroma and lymphorrhoea were significantly less frequent with percutaneous access (0% *vs.* 3%) and the procedure time was shorter by an average of 12 minutes.[183] Thus, available evidence suggests a modest advantage for percutaneous access, supporting its preferential use.

Upper limb access may facilitate cannulation of downward facing branches and enable through and through wire support, particularly when femoral access is limited by iliac disease or tortuosity. The left brachial or axillary artery is traditionally preferred to avoid crossing the aortic arch and great vessels, thereby minimising cerebrovascular risk. However, right sided access may offer ergonomic advantages and shorter delivery times, with studies showing no significant difference in stroke risk or access related complications compared with left sided access.[184,185] Ultimately, the choice of side should be guided by patient anatomy and procedural complexity. In a registry of 331 patients, percutaneous transaxillary access using suture mediated closure had an 85% success rate, with 1.5% requiring open conversion and 13.6% adjunctive procedures.[186] Another multicentre registry comparing surgical and percutaneous upper limb access using the Perclose ProGlide vascular closure device in 1 098 patients found similar complication rates after matching, although adjunctive procedures were more frequent with percutaneous access.[187] Current evidence remains insufficient to support a definitive recommendation on upper limb access for complex TAbdAo repair.

Recommendation 32 — New

Pre-operative assessment of the iliofemoral access vessels is recommended before thoracic endovascular aortic repair.

Class	Level	Reference
I	C	Consensus

Recommendation 33 — New

To enable device delivery, endovascular or open surgical optimisation should be considered for compromised access due to diseased and or small calibre iliofemoral arteries.

Class	Level	Reference
IIa	C	Consensus

Recommendation 34 — New

Percutaneous access should be considered the preferred method for common femoral artery access in patients undergoing thoracic endovascular aortic repair.

Class	Level	References	ToE
IIa	A	Vierhout *et al.* (2017),[182] Antoniou *et al.* (2021)[183]	

Recommendation 35 — New

Ultrasound guidance is recommended for percutaneous common femoral artery access in thoracic endovascular aortic repair.

Class	Level	References	ToE
I	A	Li *et al.* (2023),[175] d'Entremont *et al.* (2024),[176] Meijers *et al.* (2024),[177]	

4. ACUTE THORACIC AORTIC SYNDROME

The term acute aortic syndrome (AAS) encompasses aortic dissection (AD), intramural haematoma (IMH), and penetrating aortic ulcer (PAU). AD is generally attributed to an intimal tear, whereas IMH is thought to result from rupture of the vasa vasorum without intimal disruption, although its pathogenesis remains debated. Some view IMH as a thrombosed AD, while others consider it a distinct medial haemorrhage. PAU refers to a focal atherosclerotic lesion penetrating into the media.[27] Modern imaging suggests that AD, IMH, and PAU may reflect a spectrum of the same disease process.

Other acute aortic conditions, such as BTAI and ruptured DTAA, are discussed separately. For AAS and pregnancy, refer to Chapters 3.3 and 10.1.

4.1. Acute type B aortic dissection

Acute AD is the most common form of AAS and results from an intimal tear (entry tear) that allows blood to enter the medial layer of the aortic wall, creating a FL alongside the TL. The dissection may extend antegradely or retrogradely along the aorta and into branch vessels, potentially impairing organ perfusion. ADs involving the ascending aorta are cardiothoracic emergencies and are not addressed here. This section focuses on acute type B aortic dissection (ATBAD), which does not involve the ascending aorta and accounts for 30 — 40% of all AD cases.[36,188,189]

Hypertension, often poorly controlled, is the most common risk factor associated with ATBAD, present in over 70% of patients.[190—192] Other associated factors include increasing age, atherosclerosis, congenital aortic valve abnormalities (e.g., bicuspid or single commissure valves), stimulant use (cocaine, amphetamines), pregnancy, intense physical exertion, and emotional stress.[193—195] A family history of aortic disease is reported in 13 — 22% of cases.[196] Connective tissue disorders (CTDs) such as Marfan

Table 6. Overview of the different anatomic classification systems for acute aortic dissection, three purely based on anatomic characteristics, and three combining anatomic and pathophysiological attributes.

	Anatomic			Anatomic and pathophysiological		
	DeBakey	**Stanford**	**SVS/STS**	**Penn**	**DISSECT**	**TEM**
Year	1965	1970	2020	2012	2013	2020
Definition	Involvement of the ascending and or descending aorta	Involvement of the ascending aorta (yes/no)	Location of the primary entry tear	Stanford + circulatory and branch vessel perfusion assessment	Mnemonic	Stanford + type + entry + malperfusion
Categories	Type I: ascending + descending	Type A: ascending	Type A: entry tear in zone 0	Class A: uncomplicated	Duration (D): acute, subacute, chronic	Stanford type A and B, plus type non-A non-B
	Type II: only ascending	Type B: ascending aorta not involved	Type B: entry tear beyond zone 0	Class B: branch vessel malperfusion	Intimal tear (I): ascending, arch, descending, abdominal, unknown	Entry: E0, E1, E2, E3, which should be added to the type
	Type III: only descending	–	Type U: unidentified entry tear	Class C: circulatory compromise	Size (S): maximum aortic diameter	Malperfusion: M0–M3, plus symptoms (+/–)
	–	–	–	Class B-C: branch vessel malperfusion + circulatory compromise	Segmental extent (SE): from the ascending to the iliacs	–
	–	–	–	–	Complications (C): aortic regurgitation, rupture, malperfusion, progression, uncontrolled hypertension or enlargement	–
	–	–	–	–	Thrombosis of the FL (T): patent, partial, complete	

SVS = Society for Vascular Surgery; STS = Society of Thoracic Surgeons; Penn = University of Pennsylvania; DISSECT = duration, intimal tear, size, segmental extent, clinical complications, thrombosis of the aortic false lumen; TEM = type, entry, malperfusion; FL = false lumen.

syndrome (MFS), Loeys–Dietz syndrome (LDS), and vascular Ehlers–Danlos syndrome (vEDS) are also established risk factors (see Chapter 10.1).

Acute AD can be classified based on three key criteria: (1) anatomic extent; (2) dissection chronicity; and (3) presence or absence of acute complications (Table 6).

There are three major classification systems based exclusively on the anatomic extent of the AD (Fig. 3).

DeBakey: proposed in 1965, the DeBakey classification defines ADs according to the involvement of the ascending and or descending aorta: type I, involvement of both the ascending and descending aorta; type II, exclusive involvement of the ascending aorta; and type III, exclusive involvement of the descending aorta.

Stanford: introduced in 1970 and based on whether the dissection involves the ascending aorta:[197] type A aortic dissection (TAAD), involvement of the ascending aorta; and type B aortic dissection (TBAD), does not involve the ascending aorta. The most common site for the proximal entry tear is just distal to the origin of the LSA, with 90% of patients presenting with multiple re-entry tears along the intima, allowing blood to re-enter the TL.[188]

Society of Vascular Surgery/Society of Thoracic Surgeons: proposed by the North American Society for Vascular Surgery (SVS) and the Society of Thoracic Surgeons (STS) in 2020.[27] This system categorises dissections based on the location of the primary entry tear, rather than focusing on ascending and or descending aortic involvement: type A, location of the primary entry tear in zone 0; type B, location of the primary entry tear in zone 1 or beyond; and type U, cases in which the primary entry tear is not identifiable.

In addition to anatomic classifications, three additional systems incorporate pathophysiological characteristics, providing a more comprehensive framework for acute AD assessment (Fig. 4).

Penn: developed at the University of Pennsylvania and published in 2012,[198] this classification integrates the Stanford system (TAAD or TBAD) with four clinical presentation classes based on branch vessel malperfusion and circulatory compromise: class A, uncomplicated, haemodynamically stable patients without malperfusion; class B, branch vessel malperfusion; class C, circulatory compromise, further divided into type I: aortic rupture with haemorrhage outside the aortic wall; and type II: threatened aortic rupture, typically indicated by refractory pain and or hypertension; and class B-C, branch vessel malperfusion combined with circulatory compromise.

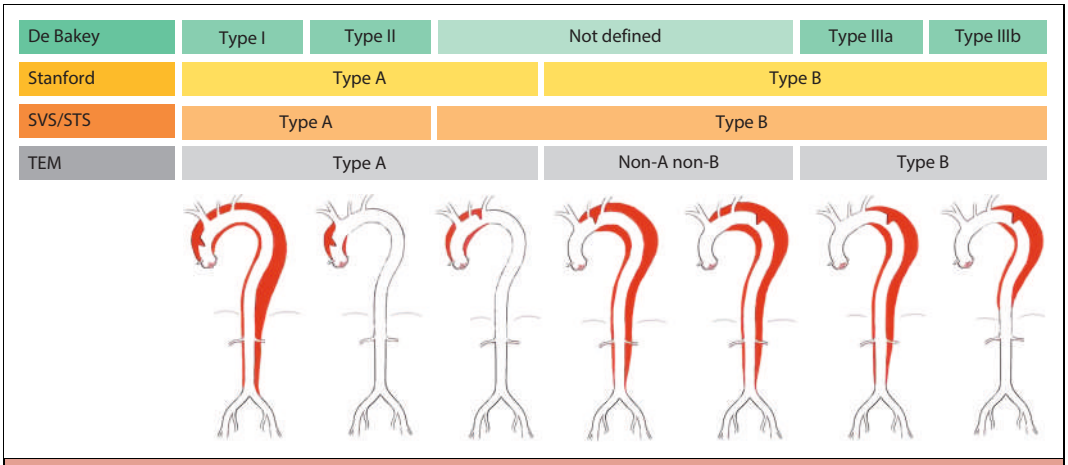
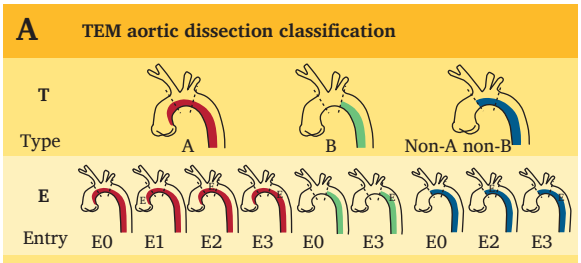


Figure 3. Anatomic classifications of aortic dissections. SVS = Society of Vascular Surgery; STS = Society of Thoracic Surgeons.

DISSECT: proposed by Dake *et al.* in 2013,[199] this system encompasses five features characterised by the mnemonic DISSECT: *duration* (D), acute (< two weeks), subacute (two weeks to three months), chronic (> three months); *intimal tear* (I), assessing the location of the primary entry tear, which can be either in the ascending aorta (A), aortic arch (Ar), descending aorta (D), abdominal aorta (Ab), or unknown (Un); *size* (S), maximum transaortic diameter of the dissected segment measured on centreline reconstruction; *segmental extent* (SE), from the proximal to the distal boundary; *clinical complications* (C), including complicated (C), with aortic valve involvement, cardiac tamponade, rupture, branch vessel malperfusion, progression of aortic involvement with proximal or distal extension, and other (uncontrollable hypertension, uncontrollable clinical symptoms, rapid FL dilation and or overall transaortic enlargement >10 mm within the first two weeks of initial diagnosis); or uncomplicated (UC); and *thrombosis of the aortic false lumen* (T), categorised as patent (P), complete thrombosis (CT), or partial longitudinal or circumferential FL thrombosis (PT).

TEM: introduced by Sievers *et al.* in 2020,[200] TEM stands for type, entry, and malperfusion: *type*, expands on the Stanford classification, introducing the non-A, non-B category to define aortic arch dissections (involvement either at the primary entry tear or via retrograde extension). This non-A, non-B terminology has also been endorsed by the European Association for Cardio-Thoracic Surgery (EACTS)/ESVS consensus statement;[201] *entry*, identified and verified by the treating physician: E0, no entry; E1, entry in the ascending aorta, between the aortic valve and the proximal edge of the brachiocephalic trunk; E2, entry in the aortic arch between the proximal edge of the brachiocephalic trunk and the distal edge of the LSA; E3, entry in the



B PENN CLASSIFICATION = Standford A/B + A-D

A	Uncomplicated, hemodynamically stable, no ischemia
B	Branch vessel malperfusion
C	Circulatory collapse with global malperfusion
B-C	Branch vessel malperfusion with circulatory collapse

C DISSECT Mnemonic classification system

Duration	Acute (< 2 weeks) Subacute (2 weeks – 3 months) Chronic (> 3 months)
Intimal tear	A: ascending aorta Ar: aortic arch D: descending aorta Ab: abdominal aorta Un: unknown
Size	Max. aortic diameter
Segment extent	From proximal to distal boundary
Clinical complications	C: Complicated UC: Uncomplicated
Thrombosis of the false lumen	P: Patent CT: complete thrombosis PT: partial thrombosis

Figure 4. Combined anatomic and pathophysiological classifications of aortic dissections.

descending aorta, below the distal edge of the LSA; and *malperfusion*: M0, no radiological or clinical signs of malperfusion; M1, dissection of at least one main coronary artery with (M1+) or without (M1−) indicators of cardiac ischaemia; M2, dissection of at least one supra-aortic vessel or aortic arch TL collapse with (M2+) or without (M2−) clinical symptoms of cerebral or upper extremity malperfusion; M3, dissection or FL origin of at least one renovisceral or iliac artery, or aortic TL collapse entailing functional closure of at least one renovisceral or iliac artery, with (M3+) or without (M3−) clinical symptoms of bowel, kidney, or lower extremity ischaemia.

Dissection chronicity: TBAD can be defined as hyperacute (< 24 hours), acute (within 14 days of the onset of symptoms), subacute (between 15 days and 90 days), and chronic (after 90 days).[27] In the acute phase, the dissection flap is considered thin and highly compliant, whereas in the chronic phase it becomes thicker and less compliant.[202]

Uncomplicated or complicated TBAD: ATBAD typically presents with the abrupt onset of severe chest and or back pain, present in about 80% of patients.[194] Migrating pain is observed in 20% of patients and pulse deficits in 15%. Approximately 40 − 50% of TBADs are classified as complicated in the acute setting. A complicated TBAD can be defined as the presence of one or more of the following:[190,191,203] (1) aortic rupture, associated with hypotension and shock; (2) intractable pain or uncontrolled hypertension, refractory symptoms despite multi-agent antihypertensive therapy and pain relief may be indicative of impending aortic rupture;[204] (3) rapid aortic expansion; (4) retrograde propagation into the arch; and (5) clinical signs of malperfusion syndromes (visceral, renal, spinal, and or lower limb ischaemia) caused by hypotension, TL compression, or branch involvement.

Serial creatinine testing and monitoring of urine output are essential for early detection and treatment of renal malperfusion.[192] Visceral ischaemia[189,190,194] may not always be associated with abdominal pain, and abdominal pain can be non-specific in ATBAD. Serum lactate elevation occurs only in advanced or irreversible intestinal ischaemia.[205,206] Therefore, a high suspicion of mesenteric ischaemia is crucial, and a low threshold for laparoscopy or laparotomy is advised.[207] Acute limb ischaemia may affect one or both lower limbs.[208] Neurological symptoms may occur secondary to malperfusion or thromboembolism, including cerebral, cerebellar, and spinal stroke. Acute paraplegia, due to SCI, is rare and may mimic lower limb ischaemia (and *vice versa*).[209]

High risk features: More recently, the SVS and STS introduced reporting standards for TBAD. Alongside uncomplicated and complicated, they proposed the additional category uncomplicated with high risk (for aortic related complications or death) TBAD. However, it is important to note that evidence supporting these high risk criteria is limited and based primarily on retrospective studies. Further validation is needed before definitive clinical recommendations can be established. The high risk criteria proposed are shown in Table 7.[27,210]

Table 7. Suggested high risk imaging features for aneurysm degeneration during the chronic phase after type B aortic dissection.[210]

Consistent evidence	Need for additional evidence
Descending aortic diameter >40 mm	Primary entry tear >1 cm
	Entry tear location in the lesser curvature (*vs.* outer curve)
	False lumen diameter >22 mm
	Bloody pleural effusion
	Radiographic but not clinically apparent malperfusion
	Retrograde aortic dissection originating from a tear in the descending aorta with extension into the arch

4.1.1. Diagnostics. Timely and accurate diagnosis of AAS is crucial to prevent life threatening complications such as rupture or dissection progression. Diagnosis is driven by clinical symptoms and confirmed through advanced imaging techniques. Multiphase CTA is the gold standard for aortic imaging owing to its high resolution, rapid completion, and widespread availability.[211,212] CTA has a 100% sensitivity and specificity and is significantly superior ($p < .05$) to MRI and TOE for evaluating supra-aortic branch involvement. It not only confirms or excludes ATBAD, but also provides crucial anatomic details for initial management[213,214] (see Chapter 3.1.1).

Despite renal impairment, in cases of strong clinical suspicion, CTA should not be delayed and should be performed without hesitation.[211]

4.1.2. Medical management. Management of ATBAD involves a combination of medical therapy and surgical interventions, requiring a multidisciplinary team at a dedicated high volume aortic centre.[215]

Blood pressure control is the cornerstone of medical management, aimed at reducing shear stress on the diseased aorta.[216] Advances in medical management have significantly improved outcomes, reducing the historical 40% acute mortality rate[217] to 10 − 14% in all ATBAD cases and as low as 1.2 − 8% in uncomplicated TBAD (uTBAD).

The primary goal is to achieve shear wall stress reduction by lowering the SBP to < 120 mmHg and maintaining a heart rate < 60 bpm.[36,188,194,218,219]

Beta blockers are considered first line therapy, with intravenous esmolol or labetalol being the preferred agents owing to their ability to reduce both heart rate and blood pressure.[190,211] If further blood pressure reduction is needed, calcium channel blockers or renin angiotensin system inhibitors may be used as adjuncts.[211,220] Vasodilators such as sodium nitroprusside should always be combined with beta blockers to avoid reflex tachycardia. Other alternative or adjunctive therapies include A1 adrenergic and non-selective blockers.[221]

While an aggressive reduction to a SBP < 100 − 120 mmHg is traditionally recommended, rapid lowering of blood pressure is associated with adverse events, including renal,

cardiac, and cerebral ischaemia. There are no RCTs supporting the published clinical experience of anti-impulse therapy described above.[222] Patients with ATBAD should be closely monitored in high dependency or intensive care units (ICUs) to ensure early detection of complications and to guide further medical and or surgical intervention.[203] If signs of renal hypoperfusion occur due to induced hypotension, the extent of blood pressure reduction should be reassessed, in light of symptom evolution and dissection progression.

In addition to haemodynamic control, effective pain management is crucial, with intravenous opiates being the mainstay of therapy. Anxiolytics, such as benzodiazepines, can also be administered to mitigate stress induced haemodynamic fluctuations.[36,188,194]

In cases of malperfusion, medical therapy must also address associated complications such as acidosis, hyperkalaemia, haemodynamic instability, and left heart failure. However, medical therapy should never delay surgical intervention in cases where urgent repair is required.

Recommendation 36		Changed
Prompt medical therapy, with pain control and anti-impulse therapy*, and invasive haemodynamic monitoring† are recommended as initial treatment for all patients with acute type B aortic dissection.		
Class	**Level**	**Reference**
I	C	Consensus

* Systolic blood pressure <120 mmHg and heart rate <60 bpm, which may be adjusted for maintaining adequate organ perfusion.
† Arterial line and continuous three lead electrocardiogram recording in high dependency or intensive care units.

4.1.3. Endovascular repair

4.1.3.1. Uncomplicated acute type B aortic dissection.

Currently, there is no conclusive evidence supporting routine TEVAR in patients with uTBAD.[223] When performed in patients with suitable anatomy, following the manufacturer's instructions for use, outcomes appear favourable.[224] Nevertheless, TEVAR is not without risks and can be associated with severe complications.[225,226]

Two RCTs have attempted to clarify the role of TEVAR in uTBAD (Table 8). The industry sponsored ADSORB (Acute Dissection Stentgraft OR Best Medical Treatment) trial is the only RCT specifically evaluating uTBAD, comparing best medical treatment (BMT) alone (31 patients) with BMT and TEVAR (30 patients) in the acute phase. At 30 days, there were no deaths in either group. The primary endpoint, a composite of incomplete or absent FL thrombosis, aortic dilatation, or rupture, was assessed at one year, but the study was not sufficiently powered to detect differences in the overall mortality rate.[227] Another industry sponsored trial, the INSTEAD (INvestigation of STEnt Grafts in Aortic Dissection) trial, randomised 140 patients with subacute ($\geq$ two weeks post-dissection) uTBAD to either BMT alone ($n=68$) or BMT with TEVAR ($n=72$).[228] At two years, TEVAR promoted favourable aortic remodelling but did not provide a survival benefit over BMT alone (two year survival rate, 95.6% BMT *vs.* 88.9% BMT + TEVAR; $p=.15$). The INSTEAD-XL study later reported five year outcomes, showing a non-significant absolute reduction in all cause mortality by 8.2% in patients who underwent TEVAR.[229] All patients had undergone complete protocol guided follow up, with no patients lost during the follow up. A landmark analysis of years two to five suggested that TEVAR significantly reduced aortic related deaths but did not improve overall survival compared with medical therapy alone. Given the ongoing uncertainty regarding the role of TEVAR in uTBAD, three additional RCTs are currently underway (Table 8).[34,230–232] These trials aim to address the limitations of ADSORB and INSTEAD, with the goal of providing conclusive evidence regarding the optimal management strategy for patients with uTBAD.

Patients with uTBAD managed with BMT, as well as those at high risk of aneurysmal degeneration in the chronic phase (Table 3) who do not undergo urgent TEVAR, require close surveillance with repeated CTA.[233] If the patient remains stable without new symptoms or imaging progression, the recommended imaging schedule typically includes a follow up CTA at 48 hours and one week after symptom onset in the acute phase, followed by CTA or MRA at one, three, and six months, and annually thereafter if the aortic diameter remains stable. However, patients who develop new or recurrent symptoms, particularly those suggestive of malperfusion, should undergo immediate CTA to reassess their condition.

Emerging research may further refine risk stratification by incorporating advanced imaging techniques. In particular, 4D flow MRI is being investigated as a potential tool to identify patients classified as "uncomplicated" but who may be at increased risk of progressive aortic growth and late complications.[234] These advances could help guide individualised follow up strategies and early intervention in high risk patients.

Recommendation 37			New
Routine thoracic endovascular aortic repair is not indicated for uncomplicated acute type B aortic dissections outside clinical trials.			
Class	**Level**	**References**	**ToE**
IIIa	B	Brunkwall *et al.* (2014),[227] Nienaber *et al.* (2009),[228] Nienaber *et al.* (2013)[229]	

Recommendation 38		New
Patients with uncomplicated* acute type B aortic dissection should be considered for continued conservative management, with anti-impulse therapy and surveillance with serial† cross sectional imaging.		
Class	**Level**	**Reference**
IIa	C	Consensus

* No signs of rupture or malperfusion.
† Suggested after 48 hours, one week, and one and three months during the acute/subacute phase.

Table 8. Randomised controlled trials on the management of uncomplicated type B aortic dissection (uTBAD).

Name	Country	Inclusion criteria	Patients	Primary endpoint	Results
Past randomised controlled trials					
ADSORB: Acute Dissection Stentgraft OR Best Medical Treatment[227]	17 European countries; industry sponsored	Acute uTBAD	$n = 61$: 31 BMT, 30 BMT + TEVAR	Composite endpoint, including incomplete/no FL thrombosis, aortic dilatation, or aortic rupture at one year	Underpowered
INSTEAD: INvestigation of STEnt Grafts in AD[228] INSTEAD-XL[229]	17 European countries; industry sponsored	Subacute (>14 days after onset) uTBAD	$n = 140$: 68 BMT, 72 BMT + TEVAR	Two and five year survival	Non-significant 8.2% absolute reduction in five year all cause mortality for BMT + TEVAR cohort
Ongoing randomised controlled trials					
SUNDAY: Scandinavian Trial of Uncomplicated Aortic Dissection Therapy	23 centres, Europe and New Zealand; investigator driven	uTBAD; randomisation after seven days to 12 weeks of symptom onset	$n = 554$: 227 BMT, 227 BMT + TEVAR	All cause mortality at five years	N/A
IMPROVE-AD: IMPRoving Outcomes in Vascular DisEase — Aortic Dissection	60 North American centres	uTBAD; randomisation between 48 hours and six weeks of symptom onset	$n = 1\,100$	Composite of all cause mortality or major aortic complications, including rupture, malperfusion, spinal cord ischaemia, stroke, and need for aortic intervention at four years	N/A
EARNEST: Early Aortic Repair in patients Needing Endovascular/open Surgery for Type B Aortic Dissection	25+ centres, UK	uTBAD; randomisation between 10 days and 90 days	$n = 470$: 235 BMT, 235 BMT + TEVAR	Composite including aortic related death, major stroke, paralysis, or serious heart and lung illness at five years	N/A

uTBAD = uncomplicated type B aortic dissection; BMT = best medical treatment; TEVAR = thoracic endovascular aortic repair; FL = false lumen; N/A = not available.

4.1.3.2. Uncomplicated high risk acute type B aortic dissection. TEVAR for uTBAD may be considered in selected patients to reduce the risk of aneurysm related complications during the chronic phase.[210] The risk factors for these complications are outlined in Table 7. In observational studies, an aortic diameter ≥ 4.0 cm is the only parameter consistently shown to predict aneurysmal degeneration.[210] Other factors, such as early total lumen expansion, total aortic diameter, total FL diameter, location of the primary entry tear, and retrograde dissection originating from a tear in the DTA with extension into the aortic arch, may also indicate increased risk of aneurysm related complications in the chronic phase, although supporting evidence remains inconclusive. Based on these factors, early TEVAR may be considered on selected individuals with uTBAD and rapid expansion or large initial diameter (> 4.0 cm), preferably during the subacute phase.[203,224,210,235–240] Furthermore, given the relative indication, TEVAR for uTBAD and large initial diameter (> 4.0 cm) should only be performed in patients with suitable anatomy that permits low risk treatment.

A recent meta-analysis of 1 051 patients (623 early *vs.* 428 late TEVAR) suggested that early TEVAR for uTBAD may reduce one year aortic related events (OR 0.48). However, the vast majority of early TEVAR cases were performed in the hyperacute phase (< 48 hours), a timing associated with significantly higher 30 day mortality (OR 10.26) and complication rates (OR 1.58). This raises concerns about the balance between short term risk and potential long term benefit. Moreover, the indication for delayed TEVAR is unclear, as delayed intervention may have been prompted by evolving high risk features.[241] A more relevant comparison would be early or subacute TEVAR *vs.* optimal medical therapy, which remains the standard for most patients. Until data from ongoing RCTs are available, routine early TEVAR for uTBAD cannot be recommended.

Recommendation 39 — **Changed**

Selected patients with uncomplicated* acute type B aortic dissection and suitable anatomy with early aortic expansion or large initial diameter (> 4.0 cm) may be considered for thoracic endovascular aortic repair, preferably during the subacute phase.

Class	Level	Reference
IIb	C	Consensus

* No signs of rupture or malperfusion.

4.1.3.3. Complicated acute type B aortic dissection. In complicated ATBAD, numerous studies have reported that interventions during the acute phase are associated with better outcomes compared with BMT alone, although no RCTs have been conducted to confirm this.[194,242,243] When intervention is indicated, the treatment paradigm for ATBAD has shifted towards endovascular procedures as first line therapy over open surgical intervention.[189,244–250]

A prospective, multicentre European clinical registry reported a 30 day mortality rate of 8%, a stroke rate of 8%, and a SCI rate of 2% in 50 patients with ATBAD treated with TEVAR.[245] Among patients with complicated ATBAD, those presenting with visceral malperfusion experience the poorest outcomes, with 30 day mortality rates ranging from 17 – 34%.[251]

Immediate life threatening complications in ATBAD include signs of rupture, such as haemothorax, increasing peri-aortic and mediastinal haematoma, and or hypotension or shock. Clinical signs of malperfusion syndromes affecting visceral, renal, spinal, or limb circulation are also regarded as critical emergencies. In non-A, non-B AD cases, mediastinal effusion and cerebral malperfusion are additional concerns.[252] These patients typically require immediate surgical intervention, either through endovascular treatment or open cardiothoracic surgery, particularly in the presence of ascending aortic haematoma. Endovascular repair may be an option for acute non-A, non-B dissections if the entry tear is located in the DTA.[201]

Impending rupture also necessitates intervention. Indicators include persistent, refractory, or recurrent pain despite adequate pain management, including intravenous opioids, and uncontrolled hypertension despite treatment with three or more antihypertensive medications at maximum recommended or tolerated doses.[219] TEVAR may also be considered in selected individuals with uTBAD and suitable anatomy and early aortic expansion or large initial diameter (>4.0 cm). In such cases, endovascular or open surgery should be delayed beyond the hyperacute phase when clinically feasible, to optimise aortic related outcomes and remodelling.[225,253,254]

The primary objective of acute endovascular treatment in TBAD is to divert blood flow and pressure away from the FL into the TL, thereby correcting malperfusion and ideally restoring the original aortic anatomy to promote complete aortic remodelling.[255] This is typically achieved by deploying a covered endograft in the proximal DTA, sealing the proximal entry tear, and addressing dynamic malperfusion when the FL compresses the TL. In cases where the LSA is involved, intentional coverage is often required to secure a stable LZ.

For ATBAD aortic rupture cases, TEVAR also aims to seal the aorta both proximally and distally. Distally by distal extension of an appropriately sized endograft to just above the origin of the coeliac artery (CA).[256] No universal rule for oversizing can be given since it is influenced by morphology and device characteristics. In the setting of rupture, however, a greater degree of oversizing may be necessary compared with non-ruptured ATBAD to secure an effective seal.

In cases of remaining static malperfusion, where the FL extends into an aortic branch, selective branch vessel stenting may be required.[257,258] In patients with unilateral limb ischaemia where endovascular revascularisation fails or is not feasible, a femorofemoral bypass is an alternative option. If TEVAR alone does not sufficiently relieve dynamic malperfusion of the visceral aorta, adjunctive techniques may also be necessary. These include isolated aortic fenestration to restore perfusion[259–261] or placement of uncovered stents in the aorta to re-expand the TL, a strategy known as PETTICOAT (provisional extension to induce complete attachment). An evolution of the PETTICOAT technique, known as stent assisted balloon induced intimal disruption and relamination in aortic dissection repair (STABILISE), involves ballooning of the covered stent graft and the distal bare stents deployed in the TL to further promote aortic remodelling.[262,263] TEVAR remains the first line treatment for ATBAD; PETTICOAT and STABILISE are not yet considered standard therapy and are currently being evaluated for their long term efficacy.

Adequate stent graft design is an important aspect of the endovascular management of ADs, as considerable tapering of the TL, often caused by compression from the pressurised FL and the elastic properties of the intima, may present a significant challenge.[264] To prevent retrograde type A aortic dissection (RTAAD), excessive proximal oversizing should be avoided in non-ruptured ATBAD, with some studies suggesting an oversizing threshold of 0 – 10%.[265,266] Furthermore, the use of tapered endografts has been proposed to mitigate excessive distal oversizing, a primary risk factor for distal stent graft induced new entry (dSINE).[265] A dissection specific endograft with a modified low radial force distal stent has been proposed to lower the risk of dSINE.[267] However, while safe and technically feasible, this approach does not entirely eliminate the occurrence of dSINE over time.[267] The distal LZ should preferably be in a straight segment of the aorta, and ballooning should be avoided. It remains unclear whether endografts with proximal barbs or other fixation elements should also be avoided when the proximal seal zone involves a dissected or diseased aorta.[268]

Transcatheter electrosurgical septotomy is an adjunctive technique that disrupts the dissection lamella, optimising sealing zones and luminal expansion during endovascular repair. Specifically, it can create viable distal LZs for adequate endovascular treatment.[269,270] While early results appear promising in select cases, concerns remain regarding the risk of lamella dislodgment, particularly in patients with acute complicated ATBAD.[271] Moreover, long term outcome data are limited, necessitating further research before widespread adoption.

TEVAR for ATBAD carries an increased risk of iatrogenic RTAAD, which is diagnosed in approximately 2.5% of cases. The risk is particularly high in patients with an ascending aortic diameter >38 mm, bicuspid aortic valve, arch abnormalities, dilation of the sinotubular junction, and extensive ascending aortic length.[201,265] RTAAD is a life threatening complication requiring urgent OSR of the ascending aorta, but despite

intervention it is associated with high mortality rates.[272] To mitigate this risk, careful endograft selection is crucial. Additionally, whenever possible, treatment during the hyperacute phase should be deferred.[244,273]

Stroke is another significant complication of TEVAR, reported in 3 — 10% of patients, with a higher incidence in those with severe aortic atherosclerosis.[255,274,275] The underlying mechanisms include embolisation due to endovascular manipulation within the aortic arch[276] and compromised cerebral perfusion following LSA coverage without revascularisation. The risk of stroke is significantly higher in patients with ATBAD compared with other aortic pathologies (OR 3.47).[277] Pre-operative assessment of supra-aortic trunk patency and morphology, including the circle of Willis, is essential, particularly when LSA coverage is planned.

LSA coverage also increases the risk of SCI, particularly in patients with haemodynamic shock, extensive aortic coverage, or a history of previous thoracic and abdominal aortic surgery.[105,136,278—280] An analysis from the VQI TEVAR module, including 501 patients with ATBAD requiring LSA coverage, showed that those undergoing LSA revascularisation had a lower risk of neurological complications (OR 0.4).[279] The availability of off the shelf, single branched stent grafts with high technical success and favourable short term outcomes has facilitated proximal landing in zone 2 with simultaneous endovascular LSA revascularisation.[281] Alternative endovascular revascularisation techniques include *in situ* laser fenestration, PMEGs, and PGs (see Chapter 3.4 on LSA coverage for further details).[282]

Failure to adhere to manufacturer's instructions for use, particularly in patients with unfavourable aortic anatomy, further increases the risk of early and late complications. These include false aneurysm formation at the stent graft edges, stent graft induced new entry (SINE), RTAAD, and acute aortic rupture.[9] Consequently, rigorous clinical and imaging surveillance is mandatory to detect these potential complications and to monitor post-TEVAR aortic dilatation, particularly in patients without early aortic remodelling.[283—288]

Recommendation 40 — Changed

Patients with complicated* acute type B aortic dissection are recommended for immediate thoracic endovascular aortic repair to cover the primary entry tear.

Class	Level	References	ToE
I	C	Steuer *et al.* (2011),[247] Fattori *et al.* (2008),[248] Fattori *et al.* (2013)[249]	

* Rupture or malperfusion.

Recommendation 41 — New

Distal extension with an appropriately sized endograft should be considered to ensure a distal seal in ruptured acute type B aortic dissection.

Class	Level	Reference
IIa	C	Consensus

Recommendation 42 — New

Patients with residual malperfusion following thoracic endovascular aortic repair for complicated acute type B aortic dissection are recommended to undergo immediate selective endovascular revascularisation of the affected target vessels.

Class	Level	Reference
I	C	Consensus

Recommendation 43 — New

Proximal landing of the thoracic endograft in non-dissected aorta should be considered in patients undergoing treatment for acute type B aortic dissection.

Class	Level	Reference
IIa	C	Consensus

Recommendation 44 — New

Balloon moulding and or excessive oversizing of the thoracic endograft is not recommended in patients with acute type B aortic dissection as it increases the risk of proximal or distal stent graft induced new entry.

Class	Level	Reference
IIIb	C	Consensus

4.1.4. Open surgical repair. OSR should be considered in patients with complicated ATBAD if BMT has failed and endovascular interventions are contraindicated. Contraindications to TEVAR in ATBAD are rare but may occur in challenging aortic arch angulation and the absence of a suitable proximal LZ.[203] Access challenges in ATBAD can usually be overcome by the use of conduits or low profile TEVAR devices (see Chapter 3.7).

The classic OSR including graft replacement of the proximal DTA via a thoracic or thoraco-abdominal incision, alongside open distal fenestration or closure of the FL at the distal anastomotic site to direct the blood flow into the TL of the aorta, is no longer current practice.[289] In most patients, the proximal entry tear is located near to or at the origin of the LSA, necessitating a proximal anastomosis in zone 2. These open procedures typically require partial cardiopulmonary bypass or deep hypothermic circulatory arrest when an "open" proximal anastomosis of the non-dissected aortic arch is preferred to reduce the risk of iatrogenic RTAAD.[290] An alternative is the frozen elephant trunk technique, which is performed via median sternotomy and involves simultaneous ascending aorta and aortic arch reconstruction. This approach eliminates the rare but serious risk of RTAAD associated with TEVAR.[201,291,292]

In patients with genetic aortopathy, the use of endografts remains controversial and is generally discouraged due to concerns about long term durability and potential complications. However, a retrospective study across 18 global centres, including 171 patients with CTDs

undergoing endovascular aortic repair, reported high rates of early technical success, low peri-operative mortality, and midterm survival comparable with that of OSR. Estimated overall survival for patients with complicated dissections or symptomatic or ruptured aneurysms was 92.4% at one year. While the rate of secondary procedures was high, few patients required conversion to OSR, suggesting that TEVAR may be considered a viable treatment option in select patients with CTDs with complicated ATBAD[293] (see Chapter 10.1).

Complications associated with OSR include death, SCI, stroke, mesenteric ischaemia and infarction, and acute renal failure.[189,246,248,289,292,294] In cases of malperfusion syndrome, including lower limb, visceral, or renal malperfusion, open surgical interventions such as fenestration, extra-anatomic bypass, or aortic replacement can be performed. However, these procedures are associated with significantly higher complication and 30 day mortality rates compared with endovascular techniques.[295–297]

Recommendation 45		Unchanged
Open surgical repair of complicated* acute type B aortic dissection should be considered in selected patients unsuitable for endovascular repair.		
Class	**Level**	**Reference**
IIa	C	Consensus

* Signs of rupture or malperfusion.

4.1.5. Follow up. Long term follow up after TBAD is essential due to the progressive nature of the disease and the potential for late complications, including aneurysmal degeneration, aortic rupture, and malperfusion syndrome. Even in cases managed conservatively with BMT, serial imaging is crucial to monitor for late aortic expansion, with long term aortic diameter change (median growth rate of 1.0 mm/year) and disease progression occurring in 20 – 55% of medically treated patients with TBAD after five years.[216,298] Surveillance allows early detection of high risk features such as rapid aortic growth (> 10 mm/year), persistent FL perfusion, and increasing aortic diameter beyond 55 mm, factors that may necessitate intervention.

For uncomplicated cases managed with BMT, an initial follow up CT or MRI after the acute phase at one, three, six, and 12 months should be considered, followed by annual imaging if stability is confirmed (see Recommendation 38). For information on follow up in the chronic phase, see Chapter 5.1. and Recommendation 55.

Patients treated with TEVAR or OSR also require routine follow up to assess for endograft related complications, including endoleak, RTAAD, or device migration and disease progression.

Recommendation 46		Changed
Individualised long term clinical follow up and serial cross sectional imaging is recommended after endovascular and open surgical treatment of acute type B aortic dissection, taking into account extent of disease and repair, imaging findings, patient fitness, and life expectancy.		
Class	**Level**	**Reference**
I	C	Consensus

4.2. Intramural haematoma and penetrating aortic ulcer

IMH is defined as the presence of blood within the medial layer of the aortic wall without an identifiable intimal defect or entry tear on imaging (Fig. 5).[299,300] The exact pathophysiology of IMH remains debated. One theory suggests that bleeding originates from a ruptured vas vasis, leading to haematoma formation within the aortic media.[301,302] Another theory proposes that IMH results from an intimal tear, allowing blood to infiltrate the aortic wall, which subsequently thromboses, rendering the entry tear undetectable.[302]

The natural history of IMH is not well defined. During its evolution, IMH may progress and propagate proximally or distally along the medial layers, resolve completely, or remain stable. IMH can occur in association with AD and PAU; however, it is distinguished from AD by the absence of an intimal flap, and from PAU by the lack of a direct connection with the aortic lumen. Despite these differences, IMH, AD, and PAU can present with similar clinical symptoms and may evolve into AD.

Reported rates of type B IMH progression vary widely from 4 – 47%.[299,303–305] IMH accounts for 10 – 30% of AAS presentations.[306] Approximately 60 – 70% of IMH cases involve the aortic arch and DTA.[307] Haematoma expansion can lead to infarction of the aortic wall, increasing the risk of aneurysmal dilatation and rupture.[304] Aortic expansion and rupture occur in 20 – 45% of IMH cases.[308,309]

PAUs are characterised by focal disruptions of the intima, with erosion extending through the elastic lamina into the aortic media, resulting in an outpouching of contrast filled blood on CTA, typically surrounded by atherosclerosis (Fig. 6).[310] The underlying pathophysiology is believed to involve erosion of atherosclerotic plaques and inflammatory changes within the vessel wall. PAUs predominantly affect elderly patients with multiple co-existing cardiovascular comorbidities.[304]

It is important to distinguish atherosclerotic PAUs from ulcer like projections or focal intimal disruptions; the latter being focal, blood filled outpouches that extend into an IMH and typically occur in 20 – 60% of patients with type B IMH.[311] High risk ulcer like projections, defined by a depth ≥ 5 mm and location in the proximal aorta, represent a dynamic and potentially unstable pathology, associated with an increased risk of adverse aortic events despite

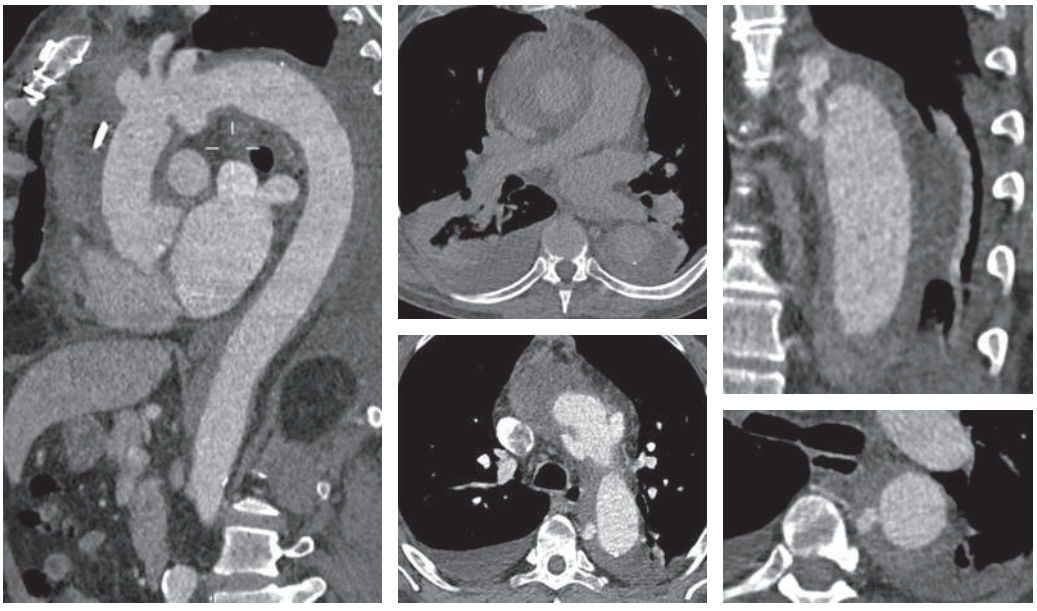


Figure 5. Intramural haematoma of the thoracic aorta (artist and copyright Carlota F. Prendes, Uppsala, Sweden).

optimal medical therapy and may warrant close surveillance and timely intervention.[312]

The true incidence of asymptomatic PAU in the general population remains unknown. However, PAUs account for approximately 2 — 7% of AAS cases.[313] While PAUs can develop anywhere along the aorta, 90% occur in the DTA.[301] Many asymptomatic PAUs are detected incidentally during imaging for other conditions. PAUs are typically

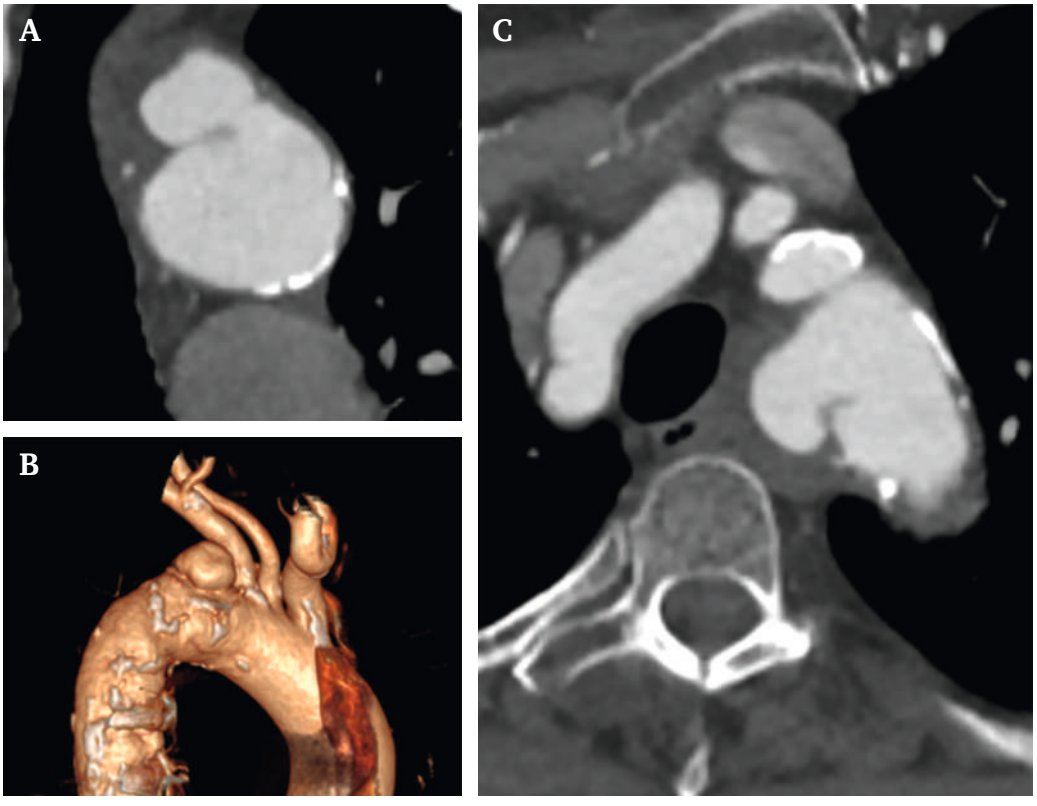


Figure 6. Penetrating aortic ulcer of the proximal descending aorta. (A) Computed tomography angiography (CTA) in coronary-style projection, (B) 3D volume-rendered reconstruction highlighting the morphology and extent of the penetrating aortic ulcer (PAU), (C) Axial CTA image at the level of the supra-aortic trunk origins, showing the precise location of the PAU. (artist and copyright Carlota F. Prendes, Uppsala, Sweden).

classified based on their anatomic location; ascending, arch, descending, or abdominal aorta. They may occur in isolation or in association with IMH. Patients with PAU and concomitant IMH are more likely to present urgently, have a larger baseline aortic diameter, and are at increased risk of SINEs following endovascular repair.[314]

Isolated PAUs without complicating features generally have a slow growth rate and can safely be treated with a conservative approach.[315] A PAU is considered complicated if it is associated with symptoms, pseudoaneurysm formation, or aortic rupture, all of which necessitate urgent intervention.[316] Symptomatic PAUs, particularly those with a depth >15 mm, have been associated with poor outcomes when treated conservatively and could warrant early endovascular intervention.[317]

4.2.1. Diagnostics. CTA and MRI are the primary cross sectional imaging techniques used to differentiate between AD, IMH, and PAU. The hallmark radiological feature of IMH is high attenuation, crescentic thickening of the aortic wall (>5 mm) without an intimal defect, extending longitudinally without causing luminal narrowing.[310] A delayed venous phase on CTA can further aid in differentiating IMH in the acute setting.

In contrast, aortic mural thrombus appears irregular, can cause luminal narrowing, and lacks the longitudinal extension characteristic of IMH. Similarly, an irregular aortic lumen suggests an atherosclerotic process or PAU. While CTA is the preferred modality in emergency settings owing to its widespread availability, MRI is useful for long term surveillance.[310]

Several studies have evaluated radiological markers that may predict disease progression and increased risk of aortic adverse events or aortic related death in both IMH and PAU. Suggested high risk imaging features for type B IMH include: maximum aortic diameter $>47-50$ mm;[116,318,319] focal intimal disruption and or ulcer like projections in the thoracic aorta in the acute phase;[311] increasing or recurrent pleural effusion;[305] progression to AD; and increasing haematoma. The corresponding high risk feature for PAU is a PAU associated with a saccular aneurysm.[313] However, most available data on imaging markers are retrospective, based on small cohorts, and prone to confounding.[318] For example, Ganaha *et al.* found a correlation between pleural effusion and adverse events, but the study included only symptomatic patients and did not distinguish between type A and type B IMH, limiting its generalisability.[305] A common methodological concern, similar to studies on uTBAD, is the classification of aortic intervention (e.g., TEVAR or OSR) as an adverse event. This introduces circular bias, as the radiological findings under investigation, such as increasing aortic diameter or haematoma thickness, often serve as indications for intervention, inflating their association with adverse outcomes. Consequently, although some of these high risk features are discussed, current evidence remains insufficient to support firm treatment recommendations.

4.2.2. Medical management. Optimal medical therapy has traditionally been recommended for uncomplicated, symptomatic IMH and PAU, with antihypertensive medications and analgesia forming the cornerstone of treatment. The clinical course can be categorised as acute (<14 days), subacute ($15-90$ days), and chronic (>90 days).[310] In the acute phase, IMH and PAU management generally follows the same principles as ATBAD, with complicated cases requiring endografting and uncomplicated cases initially managed with medical treatment, as outlined in Section 4.1.2.

Reported failure rates of medical treatment alone for IMH range from $47-72\%$,[320–322] with failure defined as aortic rupture, aorta related death, aneurysmal enlargement >55 mm, or growth >10 mm within 12 months.[322]

There is limited evidence regarding optimal surveillance and follow up strategies for IMH and PAU. It has been suggested that patients managed conservatively undergo an initial post-discharge CT at three months. An initial IMH thickness >8 mm is associated with an increased likelihood of BMT failure.[322] If no signs of progression are observed, further surveillance scans are suggested at six and 12 months. Thereafter, annual imaging is advised until year three, after which surveillance may be extended to every two years if stability is maintained.[46] Given the risks associated with radiation exposure and contrast administration, MRI may be a suitable alternative for long term surveillance.

Recommendation 47			Unchanged
Patients with uncomplicated* penetrating aortic ulcer or intramural haematoma of the descending thoracic aorta are recommended for conservative management, including best medical therapy and surveillance with serial cross sectional imaging.			
Class	**Level**	**References**	**ToE**
I	C	Nathan *et al.* (2012),[313] Salim *et al.* (2020),[315] Evangelista *et al.* (2015)[323]	

* No rupture, rapid expansion, peri-aortic haematoma, pseudoaneurysm, embolisation, malperfusion, or refractory or recurrent pain.

4.2.3. Endovascular repair. TEVAR should be considered for complicated IMH cases. Complicating features include refractory pain, haemodynamic instability, radiological evidence of disease progression (such as haematoma or aortic expansion), rupture, or progression of pleural effusion. Suggested predictors of a complicated type B IMH course include a maximum aortic diameter $>40-45$ mm, a maximum aortic wall thickness ≥ 10 mm, the presence of peri-aortic haemorrhage, development or progression of pleural effusion, and evidence of focal intimal disruption or ulcer like projections.[304,307,310] Similar complication criteria have been suggested for PAU; however, no consensus exists regarding the cutoff size for ulcer width or depth. PAU surveillance should consider ulcer growth rate and maximum aortic diameter.

TEVAR for IMH and PAU can be technically challenging, particularly when a suitable LZ free from IMH is unavailable. A minimum of 20 mm of healthy aorta is typically recommended for an adequate LZ.[46] TEVAR related adverse

events include RTAAD and SINE, with reported rates between 7% and 10% in IMH cases.[324] Additionally, patients with PAU may present increased challenges for TEVAR owing to extensive atherosclerosis, which can complicate vascular access.[325,326]

Literature supports favourable outcomes for TEVAR in IMH and PAU, with high technical success rates, delayed disease progression, increased IMH regression, and enhanced aortic remodelling.[327,292,303,314,317,320,321,323,328–331]

Recommendation 48		Unchanged
Patients with complicated* penetrating aortic ulcer or intramural haematoma of the thoracic aorta should be considered for thoracic endovascular aortic repair.		
Class	**Level**	**Reference**
IIa	C	Consensus

* Signs of rupture, pseudoaneurysm, malperfusion, rapid expansion, or refractory/recurrent pain.

5. CHRONIC TYPE B AORTIC DISSECTION

TBAD is classified as chronic TBAD (CTBAD) after 90 days.[27] The clinical presentation of CTBAD may vary; patients with initially diagnosed ATBAD entering the chronic phase of the disease may be asymptomatic or present with symptoms similar to the acute phase of the dissection, with the most common symptom being chest pain.[190] Patients with a first diagnosis of CTBAD without previous known ATBAD are commonly asymptomatic. The diagnosis in these patients is usually incidental during imaging for other reasons. In these cases, the patient's history has to be carefully evaluated for a previous acute pain event in order to define the correct timing of the dissection. Rarely, patients with CTBAD may present with other signs and symptoms (e.g., abdominal angina, limb ischaemia, renal function deterioration) indicating malperfusion, or hoarseness and acute chest pain indicating enlarging or ruptured aorta.[233]

Aortic related complications in patients with CTBAD include aneurysmal degeneration of the dissected aorta, recurrent dissection, retrograde dissection, and rupture. The expansion rate of the dissected aorta varies, but it is estimated that 20 – 55% of medically treated patients with CTBAD will develop aneurysmal degeneration after five years.[298] For CTBAD and pregnancy, refer to Chapters 3.3 and 10.1.

5.1. Medical management

Lifelong antihypertensive therapy, with a target blood pressure < 130/80 mmHg, is recommended in patients with CTBAD to prevent aortic expansion and to reduce the risk of aortic related death.[98,220,249,332–338]

Long term treatment with beta blockers has been shown to reduce aneurysmal degeneration of the dissected aorta and the incidence of late dissection related aortic procedures in non-randomised studies.[332] An analysis of the IRAD database showed that beta blockers in patients after TAAD and TBAD were the most commonly prescribed medication and suggested that their use was associated with improved long term survival.[333] A large retrospective cohort study from Taiwan, including almost 7 000 patients with AD, demonstrated that the use of beta blockers, ACE inhibitors, or ARBs after hospital discharge was associated with long term mortality reduction.[336] A Swedish registry study also demonstrated that ACE inhibitors were associated with higher long term survival in medically treated patients after AD.[339] Finally, calcium channel blockers were also shown to be associated with decreased aortic expansion during follow up and improved long term survival in patients with TBAD.[340]

Several studies have suggested that an important proportion of late deaths in patients with CTBAD are non-aorta related but rather caused by comorbidities.[189,341] Atherosclerosis has been shown to be an independent predictor of late death, and smoking has been shown to increase late death after TEVAR for TBAD,[342] while statins have been shown to increase survival in medically treated patients with AD.[339] In a retrospective study from China on 645 patients with ATBAD treated endovascularly, long term statin therapy was associated with a significantly reduced risk of all cause death, aorta related death, and aorta related adverse events.[343] This implies that atherosclerotic risk factors should be systematically assessed and adequately managed in this patient population, with particular consideration given to statin therapy.

After initial management of the dissection (either medical and or interventional), disease progression may occur causing aortic enlargement, extension of the dissection, malperfusion, rupture, or other complications. Surveillance is therefore required to diagnose such complications, but also to proactively identify risk factors that may predispose to these complications before their occurrence.

There are currently no robust data to support the superiority of any one particular surveillance programme against another. Lifelong serial imaging is required with either CTA or MRI, or a combination of both, with the aim of reducing the radiation burden especially in younger patients. The frequency of surveillance imaging should be individualised based on several parameters such as the aortic diameter, expansion rate, the status of the FL (thrombosed or not), and the presence of heritable thoracic aortic disease (HTAD).[98,344] Unfortunately, recent data have shown that almost a third of patients do not attend the first recommended follow up visit after AD, and such non-compliance with subsequent follow up was associated with a higher lifetime risk of death compared with compliant patients.[287]

Recommendation 49		Changed
Patients with chronic type B aortic dissection are recommended for life long antihypertensive therapy, with target blood pressure < 130/80 mmHg, and surveillance with serial cross sectional imaging after six and 12 months and thereafter individualised as appropriate.		
Class	**Level**	**Reference**
I	C	Consensus

Recommendation 50			Changed
Beta blockers, angiotensin converting enzyme inhibitors, angiotensin receptor blockers, and or calcium channel blockers are recommended for long term antihypertensive medical treatment in patients with chronic type B aortic dissection.			
Class	**Level**	**References**	**ToE**
I	B	Genoni *et al.* (2001),[332] Suzuki *et al.* (2012),[333] Chen *et al.* (2021),[336] Smedberg *et al.* (2022),[339] Jonker *et al.* (2012)[340]	

Recommendation 51			New
Patients with type B aortic dissection should be considered for long term statin therapy.			
Class	**Level**	**References**	**ToE**
IIa	C	Smedberg *et al.* (2022),[339] Cheng *et al.* (2024)[343]	

5.2. Indications for repair

The most common indication of aortic repair in patients with CTBAD is the development of a chronic post-dissection aneurysm of the thoracic or thoraco-abdominal aorta.[1] The goal of the repair is to prevent aortic rupture. Other less common situations that mandate evaluation and may indicate repair of a CTBAD include recurrent symptoms, rapid enlargement of the aortic diameter ($\geq$ 10 mm/year), and clinical and imaging signs of end organ malperfusion.[1,233]

In asymptomatic patients, the maximum aneurysm diameter remains the most important indicator for treatment. Available data on rupture risk mainly pertain to degenerative thoracic aortic aneurysm (TAA) and TAAA, while there is a lack of data specifically on aneurysm development after CTBAD.

A study of the natural history of 907 degenerative TAAs and TAAAs followed over time indicated that complications (rupture, dissection, and death) increase dramatically at two hinge points, 6.0 cm and 6.5 cm. Also, five year complication free survival progressively decreased with increasing aortic height index.[345]

A prospective study of the UK National Health Service, including 886 patients with arch aneurysm or DTAA, demonstrated a significant association between aneurysm size and all cause deaths (hazard ratio [HR] 1.9 per cm) and aneurysm related deaths (HR 2.2 per cm). For aneurysms of 4 — 6 cm diameter, the predicted one year mortality was below 10%, which increased to 12% and 22% for 7 cm and 8 cm aneurysms, respectively. By three years, patients with 6 cm aneurysms have a predicted mortality of 21%, while probabilities for those with 7 cm and 8 cm aneurysms have increased to 36% and 58%, respectively. The predicted one year aneurysm related event probabilities for aneurysms of 6 cm, 7 cm, and 8 cm were 3.5%, 7.1%, and 13.8% respectively. Aneurysms of $\geq$ 4 cm increased by 0.07 cm per year. Each 1 cm increase in the maximum diameter within a patient more than doubled the risk of overall death (HR 2.0) and aneurysm related death (HR 2.4).[346]

In conclusion, evidence from DTAA and TAAA studies supports considering preventive intervention for CTBAD aneurysms at a diameter $\geq$ 6 cm, with decisions guided by patient fitness, aneurysm anatomy, and individual preferences (see ToE on Recommendation #61). As aortic diameter alone can be influenced by body size, normalising it by body surface area or body height provides a more standardised measure of aortic size relative to an individual's body, which may be a better predictor of aortic aneurysm rupture and growth than simple aortic diameter, particularly in women.[347] Thus, a lower diameter threshold ($\geq$ 5.5 cm) for repair may be considered in selected patients based on aortic size index (ASI) or in significant growth cases (see ToE on Recommendation #62).

Recommendation 52			Unchanged
Patients with chronic type B aortic dissection and an aortic diameter ≥ 6.0 cm should be considered for repair, taking into account fitness, aneurysm anatomy, and patient preferences.			
Class	**Level**	**Reference**	
IIa	C	Consensus	

Recommendation 53			Unchanged
A lower aortic diameter threshold (≥ 5.5 cm) for repair may be considered in selected* patients with chronic type B aortic dissection.			
Class	**Level**	**Reference**	
IIb	C	Consensus	

* Based on aortic size index or significant growth.

5.3. Endovascular repair

5.3.1. Thoracic endovascular aortic repair. The role of TEVAR in the treatment of CTBAD has not been clearly defined. There are several studies available, but different anatomic and clinical characteristics among included patients make it difficult to interpret their findings and produce general conclusions. Available evidence suggests that TEVAR in appropriately selected patients with CTBAD appears to be safe with low peri-operative mortality and morbidity rates.[348] The long term data from the INSTEAD RCT also showed that TEVAR in the early chronic phase of the dissection was associated with improved five year aorta specific survival compared with BMT only.[229] Nevertheless, accumulating evidence suggests that TEVAR alone in CTBAD does not result in favourable aortic remodelling in the abdominal aorta, but only in the thoracic aorta, and is associated with significant re-intervention rates in midterm follow up. A study showed that TEVAR for CTBAD resulted in complete abdominal FL thrombosis in only 60% of

patients, resulting in aortic re-intervention rates of 39%.[348] Another study demonstrated a high rate of re-intervention after TEVAR for CTBAD (31% at five years), with aneurysmal degeneration distal to the treated segment being the most common reason for re-intervention.[349] Additional groups have suggested that in patients undergoing TEVAR for CTBAD, late aortic expansion in both the thoracic and abdominal aorta remains an important issue and aggressive re-intervention should be considered.[350] A systematic review showed growth of the thoracic aorta in 7 — 25% and of the abdominal aorta in 7 — 45% of patients following TEVAR for CTBAD.[288] Treatment failure (death, need for re-intervention, or failure to achieve thrombosis of the dissection related aneurysmal degeneration) has been reported in almost 40% of patients receiving TEVAR for CTBAD.[313] Particularly for extensive thoraco-abdominal dissections, TEVAR alone results in total FL thrombosis in only 13 — 30 % of patients.[351] Most recently, a meta-analysis of pooled reconstructed time to event data comparing TEVAR in different temporal settings in patients with TBAD demonstrated that TEVAR in the chronic phase was associated with poor outcomes including high rates of aortic related death and late aortic re-intervention during follow up.[352] In view of the above, TEVAR alone seems to have a limited role in the treatment of extensive thoraco-abdominal CTBAD. TEVAR alone should be considered mainly for those CTBAD in which the aneurysmal degeneration is confined to the thoracic aorta, and there is an adequate distal sealing zone in non-dissected aorta above the CA.[353] Proximal landing of the thoracic endograft should be considered in non-dissected aorta or surgical graft.

5.3.2. Fenestrated and branched endovascular aortic repair. Over recent years, specialised aortic centres have been using FBEVAR for the treatment of extensive thoraco-abdominal CTBAD. In contrast to proximal TEVAR, aiming to close the proximal entry tear, FBEVAR aims to achieve complete sealing of the post-dissection aneurysm proximally and distally. Additional technical issues in post-dissection aneurysms should be considered (narrow TL, target vessels originating from the FL, dissection progressing into distal sealing zones). FBEVAR appears to be safe and effective in the midterm with nearly identical outcomes in patients with post-dissection and degenerative TAAA.[354] Published series from high volume aortic centres report 30 day mortality rates of 0 — 5.6% with technical success rates of 86 — 100%. SCI rates range between 4.2% and 11%.[169,353,355—359] Freedom from re-intervention at two years ranged between 58% and 86%.[358] Complete FL thrombosis during follow up was shown in 65 — 92% of patients.

5.3.3. Adjunctive endovascular procedures. Various endovascular procedures have been described to induce FL thrombosis in CTBAD, mostly as an adjunct to TEVAR. These include mainly embolisation of the FL with coils, plugs, or glue, and the Candy Plug technique.[360,361] Published results of these techniques with coils, plugs or glue remain limited. The Candy Plug technique has been increasingly reported. A larger multicentre retrospective series with 155 patients treated with the Candy Plug technique for distal FL occlusion reported 100% technical success and early complete thoracic FL thrombosis in 77% of patients. During a median follow up of 23 months, the CTBAD aneurysm size was reduced in 47%, remained stable in 49%, and increased in 4%.[362] The high aortic remodelling and FL thrombosis rates support the concept of distal FL occlusion as an adjunct in the treatment of AD. Limiting aortic coverage through FL occlusion reduces the complexity of the repair and the risk of SCI.

Most recently, transcatheter electrosurgical septotomy (TES), an adjunctive technique for the management of structural valvular diseases, has been used to disrupt the dissection lamella during endovascular repair of chronic post-dissection aneurysms, for creating suitable LZs, enlarging aortic luminal diameter in patients with TL compression, and facilitating branch vessel catheterisation. TES could be particularly useful when treating patients with CTBAD with focal areas of aneurysmal enlargement allowing for a limited extent of aortic coverage, and in patients with TAAAs repaired by FBEVAR, respectively. Following reports of lamella dislodgement and intima invagination in patients with acute complicated ADs, caution should be exercised when using TES in the acute setting.[271,363] Although early reports have shown promising results in the chronic setting, the technology remains in its early stages, and definitive conclusions about its role in clinical practice cannot yet be drawn. Consequently, the use of TES should currently be confined to clinical study settings.

Recommendation 54 — **Changed**

Thoracic endovascular aortic repair should be considered as the primary treatment option for patients with chronic type B aortic dissection and aneurysm formation confined to the thoracic aorta.

Class	Level	Reference	ToE
IIa	C	Conrad *et al.* (2010)[364]	

Recommendation 55 — **New**

Proximal landing of the thoracic endograft in non-dissected aorta or surgical graft should be considered in patients treated for chronic type B aortic dissection.

Class	Level	Reference
IIa	C	Consensus

Recommendation 56 — **New**

Endovascular repair with fenestrated and branched technologies should be considered first line therapy in patients with chronic type B aortic dissection and aneurysm formation involving the thoraco-abdominal aorta.

Class	Level	References	ToE
IIa	C	Tenorio *et al.* (2020),[354] Oikonomou *et al.* (2019),[356] Abdelhalim *et al.* (2023),[359] Conrad *et al.* (2010)[364]	

Recommendation 57			New
Distal false lumen occlusion may be considered as an adjunct to thoracic endovascular aortic repair in patients with chronic type B aortic dissection and aneurysm formation.			
Class	**Level**	**Reference**	
IIb	C	Consensus	

5.4. Open surgical repair

The basic OSR steps for chronic dissection are similar to those for degenerative TAA or TAAA repair (see Chapter 6). Contemporary high volume, single centre series report operative mortality rates ranging between 6% and 16%,[141,365–369] and SCI rates range between 3% and 16 %.[370] In contrast, lower volume centres report significantly higher operative mortality rates, exceeding 20%.[364,370] These data strongly suggest that OSR for chronic post-dissection aneurysms should be limited to high volume specialist aortic centres.

Several series report long term outcomes after OSR for chronic dissections. A study published in 2011 reported 53% survival and 70% freedom from re-intervention at five years.[365] Later studies reported better survival rates of 71% and freedom from death and re-intervention of 68% at ten years.[367] A recent study reported survival of 61% and freedom from aortic related re-interventions of 85% at ten years.[369]

Since the advent of endovascular techniques, OSR for CTBAD aneurysm has played a decreasing role. However, it may still be considered for selected younger patients with a long life expectancy and or genetic aortopathy and low surgical risk.[369]

Recommendation 58			Changed
Open surgical repair may be considered in selected patients* at low surgical risk with chronic type B aortic dissection and thoraco-abdominal aortic aneurysm formation.			
Class	**Level**	**References**	**ToE**
IIb	C	Gombert *et al.* (2022),[141] Alfonsi *et al.* (2018),[367] Preventza *et al.* (2018),[368] Tanaka *et al.* (2021)[369]	

* Young patients with long life expectancy and or with genetic aortopathy.

5.5. Follow up

Given the high risk of late complications and disease progression requiring re-intervention, long term clinical follow up and imaging surveillance is recommended after any type of CTBAD repair. The frequency of imaging surveillance should be individualised based on factors such as extent and type of repair, previous imaging findings, and life expectancy.

Recommendation 59			Changed
Individualised long term clinical follow up and serial cross sectional imaging are recommended after endovascular and open surgical treatment of chronic type B aortic dissection, taking into account the extent of disease, repair, imaging findings, fitness, and life expectancy.			
Class	**Level**	**Reference**	
I	C	Consensus	

6. DESCENDING THORACIC AND THORACO-ABDOMINAL AORTIC ANEURYSMS

TAAs are defined as aortic dilatation with at least a 50% increase in diameter[371] located in any segment of the aorta between the LSA origin and the diaphragm. Type A DTAA involves the proximal DTA, terminating at the level of T6; type B DTAA involves the distal DTA, starting at T6; and type C DTAA involves the entire DTA.[294,372] Aortic aneurysms are defined as TAAA when they involve the portion of aorta at the diaphragmatic crura. The thoracic and abdominal segments may be involved with a variable extension according to Crawford's classification[373] (Fig. 7). Safi *et al.* added an extent V to Crawford's classification for aneurysm with an extension from the distal half of the DTA to the abdominal aorta, limited to the visceral segment.[374] Extent IV TAAAs are addressed in the ESVS 2024 AAA guidelines,[8] to which the reader is referred for further details.

The estimated incidence of TAA is 5 — 10/100 000 person years.[12,375,376] In the Copenhagen general population study, including 11 294 individuals with a median age 62 years, the prevalence of DTAA was 1.2% in men and 0.1% in women.[377] In a meta-analysis, the prevalence of simultaneous DTAA was 14.1 % in patients with AAA.

A meta-analysis reported a prevalence of synchronous and metachronous TAA in patients with AAA of 19.2%, with a twofold increased risk for women, while diabetes mellitus was associated with a 43% decreased risk.[378] In a Danish

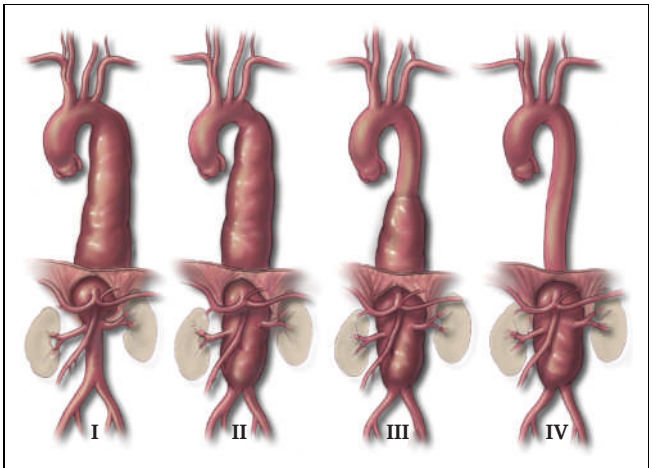


Figure 7. Crawford's classification of thoraco-abdominal aortic aneurysm. Permission to reproduce granted from Elsevier *J Thorac Cardiovasc Surg.*[373]

screening study of 14 989 participants aged 60 — 74 years, the strongest independent predictors of DTA dilatation were concomitant dilations of the ascending aorta (OR 5.8), aortic arch (OR 12.7), abdominal aorta (OR 5.5), and iliac arteries (OR 4.7).[379] These findings support the recommendation that patients with an aortic aneurysm should undergo imaging of the entire aorta.[378]

Histopathologically, TAA and TAAAs are characterised by fragmentation of elastic tissue and loss of smooth muscle cells leading to medial degeneration, with increased deposition of proteoglycans, with or without atherosclerosis.[380]

Risk factors include hypertension, especially diastolic,[377,381] atherosclerosis, and smoking.[382—384] A recent prospective, population based case control study found a clear difference in risk factor profile between AAAs and TAAs. Smoking, hypertension, and CAD were significantly associated with later diagnosis of AAAs, while hypertension was the only factor associated with TAA. This suggests a partially different aetiology for TAAs and AAAs.[385] A meta-analysis including five population based cohort studies and five case control studies, including more than one million patients, reported an inverse association between diabetes mellitus and TAA.[386] A Mendelian randomisation study also found that genetic predisposition to type 2 diabetes decreases the risk of TAA.[387]

DTAAs can also be associated with genetically triggered diseases such as MFS, LDS, vEDS, as well as other syndromic and non-syndromic conditions that may be familial or sporadic (see Section 10.1). TAA and TAAA can also be secondary to AD (see Chapter 5). Giant cell arteritis (GCA) (see Section 10.4), trauma (see Chapter 8), and infections (see Section 10.5) are less common aetiologies.

The estimated average expansion rate of DTAAs and TAAAs is 1.2 — 2.9 mm/year.[388—391] Factors associated with increased expansion rate are female sex, aneurysm size, smoking, presence of intraluminal thrombus, familial or syndromic aetiology, age, coexisting peripheral arterial disease, and COPD.[345,380,381,388,391—393]

Baseline aneurysm diameter and growth rate are strong predictors of both overall and aneurysm related death in patients with DTAA and TAAA. In the Effective Treatments for Thoracic Aortic Aneurysms (ETTAA) study, each 1 cm increase in aneurysm size, whether as a difference between patients at baseline or as growth within an individual patient, was associated with a twofold increase in the hazard of death.[346] In a prospective study of 721 patients with DTAA and a mean follow up of over nine years, the five year mortality rate among those managed conservatively was 46%. Aortic diameter was a strong predictor of adverse outcomes, including rupture, dissection, and death. For aneurysms exceeding 60 mm, the annual risk of rupture was 4%, rupture or dissection 7%, death 12%, and the combined risk of death, rupture, or dissection was 16%. In contrast, patients who underwent elective surgical repair had a life expectancy approaching that of the general population.[394] In a study of 907 patients with DTAA or TAAA, aneurysms measuring $\geq$ 6.0 cm were associated with a 19% annual rate of rupture, dissection, or death. The probability of aortic events (aortic rupture or aortic related death) increased sharply at two hinge points: 6.0 cm and 6.5 cm. Notably, 80% of dissections occurred at a diameter below 5 cm.[345] This study also reported the association between aortic aneurysm and aortic height index (AHI), defined as the ratio of aortic diameter (in cm) to patient height (in meters). AHI $\geq$ 4.2 was associated with a sixfold increase in the hazard of aortic events compared with an AHI of 3.0 — 3.5.

In addition to aneurysm size, other factors associated with increased rupture risk include older age, female sex, active smoking, diastolic hypertension, COPD, and renal impairment.[395—397] In patients with genetically triggered aortopathies, aortic complications may occur at smaller diameters than in those with degenerative atherosclerotic aneurysms.[380] Evaluation of pain as a predictor of rupture is challenging, but the presence of pain even if uncharacteristic has been associated with subsequent rupture.[393]

6.1. Management of small descending thoracic aortic aneurysms and thoraco-abdominal aortic aneurysms

All patients with DTAA and TAAA should be considered for extensive cardiovascular risk factor management, with smoking cessation, blood pressure control, statin and antiplatelet therapy, and lifestyle advice, as appropriate.[398,399] For more recommendations on risk assessment and medical management, see Chapter 3; for specific advice regarding medical management in patients with aneurysm development based on chronic AD, see Chapter 5; and for genetic aortopathy, see Chapter 10.

Patients with smaller aneurysms who may become candidates for elective repair once the diameter reaches the treatment threshold should be enrolled in routine imaging surveillance programmes (see also Chapter 3.1). The frequency of imaging surveillance should be tailored based on aneurysm size and patient risk factors.

Recommendation 60 New

Patients with a small descending thoracic or thoraco-abdominal aortic aneurysm who are deemed fit for repair are recommended for surveillance with serial* cross sectional imaging to monitor growth.

Class	Level	Reference	ToE
I	C	Sharples *et al.* (2022)[346]	

* Computed or magnetic tomography annually as baseline, less frequently for small or stable aneurysms, and individualised in case of rapid growth and when almost reaching the threshold for repair.

6.2. Indication for surgical repair

Indications for aneurysm repair in DTAA and TAAA are less extensively studied than for AAA owing to their lower incidence and prevalence. Aneurysm diameter is the key determinant. Other critical factors for surgical decision making include aneurysm anatomy, extent, and growth rate, aneurysm aetiology (e.g., genetic aortopathy), history of previous aortic surgery, comorbidities, fitness for repair, age and life expectancy, and patient preferences.

Balancing the risk of aortic complications against operative risks, a maximum aneurysm diameter of ≥ 6.0 cm on CTA is the recommended threshold for considering repair for DTAA and TAAA.[345]

At present, the available evidence does not support reducing the threshold for the general population. However, in selected patients with specific anatomic or clinical features, elective repair at a lower threshold (≥ 5.5 cm) may be appropriate to prevent fatal aortic complications. Indications for treatment of smaller diameter aneurysms include large ASI (> 3)[347,400] or rapid growth (equivalent to ≥ 10 mm in 12 months). In a prospective cohort study including 886 patients with arch aneurysm or DTAA ≥ 4 cm, women had significantly worse survival without intervention. All cause mortality was higher in women after adjustment for diameter (HR 1.65; $p < .001$), but this difference diminished after adjustment for aneurysm size index (HR 1.11; $p = .359$).[400] The shape of the aneurysm may also possibly be of importance. In a VQI analysis of 2 007 patients who underwent TEVAR for degenerative aneurysms, 712 with saccular and 1 295 with fusiform TAAs, saccular TAAs were significantly more likely to be treated as an emergency or urgently (for rupture or symptoms) below standard repair thresholds. Among women, 38% of saccular TAAs were treated below 5.0 cm compared with 10% of fusiform TAAs, while in males 47% of saccular TAAs were treated below 5.5 cm *vs.* 21% of fusiform TAAs.[401] Patients with genetically triggered DTAA and TAAAs may also be considered for repair at a smaller diameter, depending on the specific genetic condition (see Chapter 10).

In the future, machine learning may enhance current diameter based criteria for surgical intervention. In a study of 1 083 patients with DTAA measuring ≥ 3.0 cm (mean diameter 4.1 cm) and a mean follow up of 3.5 years, six machine learning classifiers were trained using 44 clinical variables to predict the risk of dissection, rupture, or all cause death at one, two, and five years. The models achieved area under the receiver operating characteristic (ROC) curves of 0.847 − 0.856 for predicting type B dissection or rupture and 0.820 − 0.845 for predicting type B dissection, rupture, or all cause death. All machine learning models consistently outperformed descending aortic diameter alone, which had ROC values of 0.713 − 0.733. Key predictive variables beyond aortic diameter included history of MI, hypertension, and sex.[402]

Recommendation 61			Unchanged
Patients with descending thoracic or thoraco-abdominal aortic aneurysms and an aortic diameter ≥ 6.0 cm should be considered for repair, taking into account fitness, aneurysm anatomy, and patient preferences.			
Class	**Level**	**References**	**ToE**
IIa	C	Zafar *et al.* (2021),[345] Sharples *et al.* (2022),[346] Davies *et al.* (2002),[394] Kim *et al.* (2015)[403]	

Recommendation 62			Unchanged
A lower aortic diameter threshold (≥ 5.5 cm) for repair may be considered in selected* patients with descending thoracic or thoraco-abdominal aortic aneurysms.			
Class	**Level**	**References**	**ToE**
IIb	C	Chen *et al.* (2021),[311] Zafar *et al.* (2021),[345] Pouncey *et al.* (2024)[400]	

* Based on aortic size index or significant growth.

Recommendation 63			New
Non-infected saccular descending thoracic aortic aneurysms may be considered for early surgical treatment with a lower diameter threshold for elective repair than for standard fusiform aneurysms.			
Class	**Level**	**Reference**	
IIb	C	Consensus	

6.3. Surgical repair of descending thoracic aortic aneurysms and thoraco-abdominal aortic aneurysms

6.3.1. Endovascular repair. TEVAR is the preferred treatment for patients with DTAA. When the anatomy is favourable, the procedure is typically straightforward and can be performed using a standard tube graft. To achieve adequate sealing during TEVAR, both proximal and distal LZs should be at least 20 mm in length, with an aortic diameter ≤ 4.2 cm at the LZs.

Evidence comparing TEVAR and OSR for DTAA primarily derives from systematic reviews and meta-analyses of retrospective series and non-randomised or population based studies. A 2016 Cochrane database systematic review concluded that non-randomised studies suggest TEVAR is associated with improved early outcomes, including lower rates of paraplegia and death and shorter hospital stays, compared with OSR.[404] In a large, population based study using Medicare data from patients treated between 1999 and 2010 with follow up through 2014, 1 235 OSR patients were propensity matched to 2 470 TEVAR patients. OSR was associated with a higher peri-operative mortality rate but a lower late hazard of death. The odds ratio for peri-operative mortality with OSR compared with TEVAR was 1.97 in high volume centres and 3.62 in low volume centres. Despite this late survival advantage for OSR, overall mean survival favoured TEVAR (−209 days for OSR). However, the risk of re-intervention was significantly lower after OSR (HR 0.40).[405] A meta-analysis comparing OSR and TEVAR for DTAA included 10 672 OSR patients and 3 908 TEVAR patients. OSR patients were significantly younger (mean age 65.1 years *vs.* 70.0 years), and the majority in both groups underwent elective repair (OSR 83.4% *vs.* TEVAR 81%). OSR was

associated with significantly longer ICU stay (8.5 days *vs.* 4.5 days) and overall hospital stay (9.5 days *vs.* 5.7 days). The rates of major complications, including paraplegia (5.5% *vs.* 3.3%), renal failure (8.3% *vs.* 6.2%), and cardiac complications (13.5% *vs.* 3.1%), were significantly higher in the OSR group. However, the 30 day mortality rate was higher in the TEVAR group (3.2% *vs.* 4.4%). Importantly, there were no significant differences in one and five year mortality rates between the two groups.[406] A recent study reporting ten year outcomes following TEVAR for DTAA showed Kaplan—Meier estimates of overall survival at 76%, 59%, and 34% at one, five, and ten years, respectively. Freedom from re-intervention was 84% at one year, 73% at five years, and 58% at ten years. In multivariable analysis, factors independently associated with an increased risk of re-intervention included fusiform aneurysm morphology, proximal LZ 2, and hypertension, while device generation was not a significant predictor.[407] In a cohort study of 685 patients undergoing TEVAR for isolated DTAA, of whom 54% were women, no significant differences in short and long term outcomes were observed between sexes. Perioperative mortality was 4.1% in women and 2.2% in men ($p = .25$), while the overall complication rate was 16% in women and 21% in men ($p = .16$). At five years, women and men had comparable outcomes: mortality rate (58% *vs.* 53%), re-intervention (39% *vs.* 30%), and late aortic rupture (0.6% *vs.* 1.2%). After adjustment for baseline differences, outcomes remained similar between the sexes over the five year follow up period.[408]

The risk of stroke following TEVAR is approximately 3—4%.[155,409] A systematic review of 2 594 patients (61% male; mean age 72 years) undergoing TEVAR for DTAA reported a pooled stroke incidence of 4.1%. Stroke risk varied depending on the management of the LSA; when left uncovered the stroke incidence was 3.2% compared with 5.3% when the LSA was covered but revascularised with a simultaneous bypass, and 8.0% when the LSA was covered without revascularisation. These findings suggest preventive revascularisation of the LSA should be considered whenever possible[110,410] (see also Chapter 3).

SCI is one of the most feared complications of TEVAR. In an analysis of 7 889 TEVAR patients (mean age 67.6 ± 13.9 years; 65% male) from the VQI dataset 2014—2018, the incidence of transient SCI was 1.5% and permanent SCI 2.1%.[409] For more information on prevention and treatment of SCI, see Chapter 5.1.

Coverage of the CA may be required to achieve an adequate distal seal during TEVAR. A recent meta-analysis of 15 observational studies including 3 506 patients reported 236 cases of intentional CA coverage. The pooled rates of visceral ischaemia, SCI, and 30 day or in hospital mortality were 13%, 5%, and 4%, respectively. Notably, 44% of patients who developed visceral ischaemia died within 30 days or during the hospital stay.[165] Further evidence from a VQI analysis of 628 patients undergoing TEVAR for TAAA, with endograft extension to the level of the CA, compared outcomes between those with intra-operative CA occlusion ($n = 44$) and those with CA preservation ($n = 584$). CA occlusion was associated with significantly higher 30 day mortality rate (11% *vs.* 4%) and a higher incidence of a composite endpoint including death and bowel ischaemia (23% *vs.* 9%). On multivariable analysis, CA occlusion was independently associated with an increased 30 day mortality rate (OR 3.9) and the composite endpoint (OR 3.0).[411] Therefore, in cases where CA coverage is planned, collateral circulation should be assessed on preoperative CTA. If adequate collaterals through the gastroduodenal artery are demonstrated, CA coverage without revascularisation may be considered.[412,413] Otherwise, CA revascularisation should be performed to reduce the risk of visceral ischaemia,[411,414,415] preferably by endovascular means with a CMD or *in situ* fenestration or PGs.

A VQI based risk calculator for predicting 30 day mortality following endovascular repair of an intact DTAA identified six predictive variables: age, CAD, American Society of Anesthesiologists (ASA) physical status class IV and or V, urgency of the procedure, previous carotid revascularisation, and a proximal LZ located proximal to zone 3.[91]

Endovascular TAAA treatment is more challenging due to renovisceral artery involvement, the extensive length of the affected aortic segment, and the presence of vital intercostal and lumbar arteries. FBEVAR is the most widely adopted endovascular technique for TAAA treatment. Initially, these devices were manufactured on a customised basis to match the individual patient's anatomy.[416] CMDs offer several advantages, particularly in terms of anatomic precision, but they are limited by their production time, which typically requires several weeks. This delay makes them unsuitable for urgent or emergency cases and carries a significant risk of aneurysm rupture during the waiting period, especially in patients with large aneurysms.[417,418] To address this limitation, a number of off the shelf branched devices have been developed for TAAA. These are designed based on the most common anatomic configurations of target vessels to accommodate a broad range of patients.[419] As of the publication of these guidelines, two such devices have received CE mark approval and are commercially available, while others are currently under evaluation. An alternative approach involves the use of PMEGs. These modifications can either be performed on the back table before device deployment or created "*in situ*" after graft release using needles, laser probes, or electrified wires.[378,420,421] Both techniques have yielded favourable outcomes in selected cases within a limited number of specialised aortic centres. However, they remain experimental, have not gained widespread acceptance, and should preferably be reserved for use within the context of clinical research or in emergency settings. Procedures should be systematically documented in prospective databases to support ongoing evaluation.

FBEVAR has demonstrated acceptable outcomes in patients with TAAA. In a meta-analysis, the pooled rates of SCI were 13.5% (95% confidence interval [CI] 10.5—16.7%), permanent paralysis 5.2% (95% CI 3.8—6.7%), postoperative dialysis 6.4% (95% CI 3.2—9.5%), permanent dialysis 3.7% (95% CI 2.0—5.9%), stroke 2.7% (95% CI

1.9 — 3.6%), and peri-operative death 7.4% (95% CI 5.9 — 9.1%).[54]

Target vessel instability is a composite definition including any stent stenosis, stent separation, or type Ic or type IIIc endoleak necessitating re-intervention. It also includes stent occlusion, aneurysm rupture, or death due to complications involving target vessels. Retrospective data suggest that the renal arteries are more susceptible to target vessel related complications than the visceral arteries (6% *vs.* 2%) during complex abdominal and thoraco-abdominal procedures.[422]

Single centre and multicentre retrospective observational studies have shown that endograft designs incorporating renal fenestrations are associated with higher patency rates than those using renal branches.[423,424] Based on these observations, fenestrations may be preferable to branches for renal arteries, provided the anatomy allows for close apposition of the fenestration to the aortic wall.[425]

A variety of balloon and self expandable stent grafts from different manufacturers have been used to bridge the target vessels in FBEVAR.[423,426] Most stent grafts have shown acceptable outcomes, although long term failure of some earlier generation bridging stent grafts has raised concerns.[427,428] While used in FBEVAR, it is important to note that their use in this specific application is considered off label for many available stent grafts. Currently, the only on label bridging devices for FBEVAR are the balloon expandable BeGraft, BeFlared (Bentley InnoMed GmbH, Hechingen, Germany) and Gore VBX (Gore Medical, Flagstaff, AZ, USA).[429,430] The Advanta V12/iCast balloon expandable covered stent (Getinge Maquet, Rastatt, Germany; Getinge/Atrium Medical Corporation, Merrimack, NH, USA) was recently approved as a bridging stent in the US market. Hence, the constant developments in bridging stent technology and the lack of comparative data and long term results make clear recommendations challenging.

For fenestrated main body designs, balloon expandable covered stents are widely accepted as the preferred bridging stent design due to the robust radial strength, low profile, and accuracy in deployment at the fenestrations. A diameter of $\leq$ 0.4 cm, more aortic protrusion in the renal target vessels as well as a pre-operative tortuosity index and oversizing of the bridging stent in the visceral target vessels have been associated with adverse events in fenestrated repair.[431]

For branched main body designs, extensive experience is available both with self expandable and balloon expandable bridging stents as well as with the combination of these two.[354,432—434] Target vessel diameter, bridging length (gap), and horizontal misalignment seem to be associated with adverse target vessel outcomes in branched repair.[435] The choice between balloon expandable and self expanding stent grafts for directional branches remains an evolving field, with rapidly emerging evidence and ongoing innovation. A 2024 meta-analysis suggested slightly inferior outcomes with balloon expandable bridging stents.[436] Similar findings have been reported in retrospective single centre and multicentre studies showing favourable results with self expanding stents or combinations of the two.[354,432] Nevertheless, current evidence does not support a universal recommendation for any single bridging stent design in directional branches. Stent selection should be individualised based on patient anatomy, procedural complexity, operator experience, and the available long term performance data of the specific stent used in TAAA repair.

Recommendation 64 — **Changed**

Thoracic endovascular aortic repair is recommended as the first line surgical treatment option in patients with descending thoracic aortic aneurysms.

Class	Level	References	ToE
I	B	Abraha *et al.* (2016),[404] Chiu *et al.* (2019),[405] Harky *et al.* (2019)[406]	

Recommendation 65 — **Changed**

Endovascular repair with fenestrated and branched technologies should be considered as the first line surgical treatment option in patients with thoraco-abdominal aortic aneurysms.

Class	Level	References	ToE
IIa	C	Oderich *et al.* (2021),[419] Dias-Neto *et al.* (2023),[437] Jobim *et al.* (2024),[438] Finnesgard *et al.* (2025)[439]	

Recommendation 66 — **Changed**

Open surgical repair may be considered in selected patients* at low surgical risk with descending thoracic or thoraco-abdominal aortic aneurysms.

Class	Level	References	ToE
IIb	C	Rinaldi *et al.* (2025),[440] Zeigler *et al.* (2025),[441] Cambiaghi *et al.* (2024)[442]	

* Young patients with long life expectancy or with genetic aortopathy.

Recommendation 67 — **New**

If coverage of the coeliac artery is required to achieve an adequate seal during endovascular repair for descending thoracic aortic repair, revascularisation should be considered if collateral flow from the superior mesenteric artery is insufficient, preferably by endovascular techniques.

Class	Level	References	ToE
IIa	C	King *et al.* (2020),[411] Hanna *et al.* (2022),[415] Mezzetto *et al.* (2023)[443]	

Recommendation 68			New
In endovascular thoraco-abdominal aortic repair, fenestrations may be considered preferred over branches for the renal arteries when the endograft is adjacent to the aortic wall at the level of the renal arteries.			
Class	**Level**	**References**	**ToE**
IIb	C	Panuccio *et al.* (2015),[423] Katsargyris *et al.* (2023),[424] Martin-Gonzalez *et al.* (2016)[444]	

Recommendation 69		New
Balloon expandable bridging covered stent design should be considered as first choice for fenestrations.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 70		New
Balloon expandable, self expanding, and combinations of bridging covered stent designs may be considered for branches during thoraco-abdominal aortic repair, taking into account the bridging distance, target vessel anatomy, and properties of the bridging stent.		
Class	**Level**	**Reference**
IIb	C	Consensus

6.3.2. Open surgical repair. Although endovascular repair is the preferred treatment for DTAA and TAAA in patients with suitable anatomy, OSR may be considered in the following situations: (1) lack of adequate arterial access or contraindications to aortic or iliac conduit placement (e.g., severe aorto-iliac occlusive disease); (2) absence of suitable proximal or distal LZs; (3) genetically triggered DTAA; (4) DTAA in young, otherwise healthy patients without major contraindications to OSR; (5) presence of aorto-oesophageal fistula; and (6) symptoms resulting from compression of adjacent structures by a large DTAA, such as chronic pain due to vertebral erosion, dyspnoea from tracheobronchial compression, dysphagia from oesophageal compression, or cardiac symptoms.[278,445,446]

The risk of post-operative renal failure is increased in patients with pre-existing renal impairment. These patients benefit from pre-operative hydration and maintenance of adequate renal perfusion during the peri- and post-operative periods.[447] Robust evidence shows that the risk of SCI, as well as mesenteric and renal ischaemia, is closely associated with the duration of aortic cross clamping. Cross clamp time is recognised as one of the most critical predictors of post-operative neurological complications.[447] To mitigate these risks, various methods of extracorporeal circulation may be employed, including left heart bypass (LHB), temporary axillary to femoral artery shunting, cardiopulmonary bypass without cardiac arrest, and deep hypothermic circulatory arrest. These more advanced perfusion strategies may be particularly beneficial in aortic rupture cases or when proximal clamping carries a high procedural risk.[440,448,449]

Mortality rates for DTAA repair remain significantly different between elective and emergency settings, reported at approximately 10% and 45%, respectively.[450] However, outcomes following elective OSR of DTAA have improved substantially over recent decades. A meta-analysis including 12 245 patients undergoing OSR for DTAA reported a pooled operative mortality rate of 6.6% for DTAA, with a pooled late mortality rate of 0.6% per person year. Pooled post-operative complication rates for DTAA included: stroke 4.5%; permanent SCI 2.9%; renal failure 5.3%; respiratory failure 19.9%; and MI 4.1%. Meta-regression analysis found that use of the clamp and sew technique and CSFD were associated with lower operative mortality, whereas ruptured aneurysm was associated with higher mortality. The authors noted that the lower mortality rate observed with the clamp and sew technique might reflect selection bias, but could also be attributed to the technique's increased procedural speed and reduced risk of coagulopathy.[451]

During open DTAA and TAAA repair, extracorporeal circulation techniques are employed to maintain distal perfusion and avoid haemodynamic disturbances such as severe afterload increase and organ ischaemia. Common strategies include LHB, cardiopulmonary bypass, and deep hypothermic circulatory arrest.[440,452,453] In LHB, a temporary bypass circuit is established from the left atrium to the distal aorta or femoral artery. Following completion of the proximal anastomosis, selective visceral perfusion is typically performed by direct cannulation and perfusion of the CA and superior mesenteric artery using blood from the LHB circuit.[374] With regard to renal perfusion, neither isothermic nor cold blood perfusion has demonstrated superior efficacy over cold crystalloid solutions in protecting renal function.[454] Histidine—tryptophan—ketoglutarate (HTK) solution (Bretschneider's solution, also commercially known as Custodiol) is a low potassium, high flow crystalloid solution primarily used for organ preservation in transplantation, particularly for liver, kidney, heart, lung, and pancreas. It is known for its protective effects, including buffering capacity, low electrolyte content, and ability to stabilise cell membranes. Renal perfusion with HTK at 4°C has demonstrated superior renoprotective effects in a RCT.[455,456]

For visceral artery re-implantation, a commonly used method is the inclusion technique (Carrel patch), which involves a tailored side cut on the aortic graft to incorporate visceral vessels. This technique is relatively straightforward and reduces the number of anastomoses, potentially shortening organ ischaemia time. However, when the visceral ostia are widely separated, this technique may predispose to aneurysmal aortic patch degeneration over time.[457] To mitigate this risk, individual re-attachment of widely spaced visceral vessels (often the left renal

artery) may be preferred, either directly or using an interposition graft. Alternatively, the use of pre-manufactured branched grafts allows for separate anastomoses to each visceral artery, which may offer greater durability and reduced patch related complications. These individualised techniques are particularly suitable for younger patients and those with genetically triggered aortopathies, such as MFS.

Post-operative pain following DTAA or TAAA OSR can significantly impact recovery. It may limit patient mobilisation, impair respiratory effort, and lead to secretion retention, all contributing to prolonged hospital stay, increased risk of pulmonary complications, and a potentially higher post-operative mortality rate. Post-operative pain management may include several analgesic strategies, including systemic opioids, epidural analgesia, intercostal nerve blocks, and local anaesthetic infusions. While these methods offer varying degrees of pain relief, none have fully resolved the challenge of post-thoracotomy pain, and each is associated with its own drawbacks. In this context, intercostal nerve cryoablation has emerged as a promising adjunctive approach for post-operative analgesia. Recent studies have reported encouraging outcomes, demonstrating improved pain control and reduced reliance on systemic analgesics.[458]

Recommendation 71 — **Changed**

Distal aortic perfusion is recommended during open surgical repair for thoracic and thoraco-abdominal aortic aneurysms to maintain end organ perfusion and reduce the risk of ischaemic complications.

Class	Level	References	ToE
I	C	Kouchoukos *et al.* (2019),[453] Fehrenbacher *et al.* (2010)[459]	

Recommendation 72 — **New**

Renal perfusion using cold crystalloid solutions, such as histidine–tryptophan–ketoglutarate (HTK) solution, should be considered during open surgical repair of thoracic and thoraco-abdominal aortic aneurysms to minimise ischaemia–reperfusion injury.

Class	Level	References	ToE
IIa	B	Tshomba *et al.* (2014),[455] Kahlberg *et al.* (2023)[456]	

6.3.3. Hybrid repair. Hybrid repair, which combines surgical re-routing (bypassing) of the visceral and renal arteries with endovascular exclusion of the aortic aneurysm using a standard stent graft, represents an alternative strategy for managing TAAAs. Originally proposed as a less invasive option compared with conventional OSR,[460] hybrid repair has not demonstrated a consistent reduction in complication rates at early or midterm follow up.[461–463] Despite the advantage in the avoidance of thoracotomy and aortic cross clamping, in practice the approach tends to inherit the early risks of OSR and the late complications of endovascular therapy. A meta-analysis of 528 hybrid TAAA repairs across 14 studies reported a peri-operative mortality rate of 14.3%, with SCI rates of 7.0%, mesenteric ischaemia 4.5%, and permanent renal failure 7.0%.[461] Given the established efficacy of conventional OSR and the ongoing advances in fenestrated and branched endovascular techniques, the role of hybrid repair in contemporary TAAA management has become increasingly limited. Nevertheless, surgical bypass from the iliac artery to one or more visceral arteries remains a valuable bailout option in cases of endovascular target vessel failure during or after FBEVAR.

6.3.4. Management of bleeding and antithrombotic therapy. Management of peri-operative bleeding remains one of the major challenges in TAAA OSR. Standard plasma coagulation tests and plasma fibrinogen level measurements have notable limitations in guiding the real time management of bleeding disorders. Viscoelastic testing methods, such as thrombo-elastography (TEG) or rotational thromboelastometry (ROTEM), offer valuable insights by assessing clot formation kinetics and clot strength. These tests enable real time identification of specific coagulation defects and provide targeted guidance for haemostasis management during surgery.[464] In this context, evidence from an RCT supports the use of fibrinogen concentrate as an effective alternative to fresh frozen plasma for correcting coagulopathy during TAAA OSR, when clinically appropriate.[465]

The endovascular approach offers the advantages of reduced invasiveness and lower intra-operative blood loss compared with OSR. However, non-negligible bleeding can still occur during endovascular procedures, particularly in complex or prolonged cases. Additionally, extensive thrombosis within the aneurysm sac may lead to platelet consumption and activation of the coagulation cascade, potentially resulting in coagulopathy.[466] In prolonged procedure cases, significant blood loss, clinically evident bleeding, pre-existing coagulation disorders, or laboratory evidence of coagulopathy, the use of viscoelastic testing (e.g., TEG or ROTEM) may be valuable. These methods allow real time assessment of clot formation and strength, facilitating the diagnosis and management of acquired coagulation disturbances during and after endovascular repair.[467]

Proper heparinisation during open DTAA and TAAA repair is essential to prevent blood clots from forming. Activated clotting time (ACT) is the most commonly used test to monitor the effect of heparin during surgery. Although there is limited evidence for the efficacy of heparin in endovascular DTAA and TAAA repair, it is a general vascular surgery principle. Accepted doses range between 50 IU/kg and 100 IU/kg, and heparin efficacy may be tested using ACT to ensure adequate anticoagulation, with a suggested target ACT of 200 – 250 seconds.[468,469]

Long term target vessel patency remains a major concern in endovascular TAAA repair. Reported rates of target vessel instability after FBEVAR range from 5 – 30%,[470] with higher occlusion rates observed for branches compared with fenestrations.[471] The optimal medical management

strategy to mitigate the risk of target vessel occlusion remains the subject of debate, with considerable variation in practice across centres.[467,472] A recent multicentre retrospective study including 1 430 patients undergoing FBEVAR found that dual antiplatelet therapy (DAPT) was associated with a lower incidence of cardiovascular ischaemic events and higher target vessel patency compared with single antiplatelet therapy, without an observed increase in bleeding complications.[473] These findings were corroborated by a separate analysis from the US Aortic Research Consortium involving 1 525 patients, which also reported improved target vessel patency in those receiving DAPT.[472] However, both studies are limited by their retrospective, non-randomised design, relatively short follow up, and lack of detailed bleeding risk assessment in patients selected for single antiplatelet therapy *vs.* DAPT. A multicentre study focusing specifically on branch stent grafts included 120 patients and 416 target vessels. At five years, the stent occlusion rate was 10.6% for renal branches and 3.7% for visceral branches. In multivariable Cox proportional hazards analysis, absence of any antithrombotic therapy was associated with a nearly 11 fold increased risk of stent occlusion (HR = 10.7). The median time to graft occlusion was 260 days (interquartile range 144, 422). Notably, several occlusions occurred during aspirin monotherapy following an initial period of DAPT, although this observation did not reach statistical significance.[474] Thus, available data suggest that DAPT may be beneficial after FBEVAR in patients without an increased bleeding risk. Initiation of DAPT is appropriate once the immediate post-operative phase has passed, generally upon discharge. The optimal duration of DAPT continues to be a topic of discussion. The duration of DAPT is often individualised, with three to six months being considered for most patients,[467] while longer durations (beyond 12 months) may be considered for patients at high risk of vessel occlusion and who tolerate DAPT without bleeding events and are at low risk of bleeding. Patients with a high bleeding risk, such as those with a history of bleeding or taking other medications that increase bleeding risk, may require shorter durations of DAPT, possibly even just one month if three months poses safety concerns. The bleeding risk in patients considered for DAPT can be assessed using risk scores such as the PRECISE-DAPT score, which incorporates five parameters: age, haemoglobin, creatinine clearance, white blood cell count, and history of previous spontaneous bleeding.[475]

Recommendation 73 — New

Viscoelastic testing* to assess real time clot formation and stability should be considered during open surgical repair of thoracic and thoraco-abdominal aortic aneurysms to guide haemostatic management.

Class	Level	Reference ToE
IIa	C	Monaco *et al.* (2019)[464]

* Thromboelastography (TEG) or rotational thromboelastometry (ROTEM).

Recommendation 74 — New

Intra-operative administration of intravenous heparin (50 – 100 IU/kg) is recommended during elective endovascular repair of descending thoracic and thoraco-abdominal aortic aneurysms to prevent thromboembolic complications.

Class	Level	Reference
I	C	Consensus

Recommendation 75 — New

Activated clotting time monitoring should be considered during open and endovascular repair of thoracic and thoraco-abdominal aortic aneurysms to assess heparin efficacy and guide additional dosing.

Class	Level	Reference
IIa	C	Consensus

Recommendation 76 — New

For patients without an increased bleeding risk, temporary dual antiplatelet therapy may be considered after endovascular thoraco-abdominal aortic aneurysm repair with fenestrated or branched endografts to reduce thrombotic complications and improve target vessel patency.

Class	Level	Reference
IIb	C	Consensus

6.3.5. Accessory renal arteries. Accessory renal artery (ARA) is a common anatomic variant, with an incidence of 9 – 20% among patients with AAA, which may pose challenges during both endovascular and open TAAA repair.[476]

In endovascular repair, ARA coverage has been associated with complications such as persistent type II endoleaks and renal infarction. Conversely, efforts to preserve ARAs often necessitate CMDs and prolonged procedure times due to the need for complex bridging manoeuvres.[477,478] A recent meta-analysis of ten retrospective studies published between 2004 and 2020, including 1 014 patients (302 with ARA coverage and 712 without ARA or without coverage), found that coverage of ARAs measuring less than 4 mm was associated with a higher risk of renal infarction. However, there was no significant impact on renal function or death during the early post-operative period or long term follow up.[479]

In the setting of OSR, ligation of ARAs also carries a risk of renal infarction. Alternatively, re-attachment of these arteries to the aortic graft may prolong the operating time and increase the risk of late complications, such as anastomotic degeneration or pseudoaneurysm formation.[480]

Although emerging imaging techniques offer improved tools to assess the functional contribution of ARAs to renal perfusion, it remains challenging to precisely quantify the volume of parenchyma perfused by an individual vessel.[481] Careful pre-operative planning is essential for managing ARAs, particularly given that coverage or ligation of these

vessels may lead to renal infarction, which can increase the risk of post-operative renal failure, especially in patients with pre-existing renal impairment.[482] Current evidence supports a selective management strategy: small ARAs may be safely excluded, while preservation is recommended for larger ARAs ($\geq$ 4 mm in diameter) or those presumed to supply a significant portion of the renal parenchyma (e.g., more than one third).[479]

There is currently no evidence supporting routine pre-emptive embolisation of ARAs before endovascular TAAA repair. However, embolisation may be considered for larger ARAs ($>$ 3 mm in diameter) arising directly from the aneurysm sac, where the risk of persistent type II endoleak is increased.[483]

Recommendation 77		New
Preservation of large accessory renal arteries (≥ 4 mm) or those that supply a significant portion of the kidney (≥ one third) should be considered during thoraco-abdominal aortic aneurysms repair to minimise the risk of post-operative renal dysfunction.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 78		New
Pre-emptive embolisation of accessory renal arteries may be considered in selected cases* before endovascular thoraco-abdominal aortic aneurysm repair.		
Class	**Level**	**Reference**
IIb	C	Consensus

* Large accessory renal arteries arising directly from the aneurysm sac, where the risk of persistent type II endoleak is deemed elevated.

6.4. Follow up

Given the high risk of late complications and disease progression requiring re-intervention, long term clinical follow up and imaging surveillance is recommended after any type of DTAA or TAAA repair. The frequency of imaging surveillance should be individualised based on factors such as extent and type of repair, previous imaging findings, and life expectancy.

Recommendation 79		Changed
Individualised long term clinical follow up and serial cross sectional imaging are recommended after thoracic or thoraco-abdominal aortic aneurysm repair, taking into account extent of disease, repair, imaging findings, fitness, and life expectancy.		
Class	**Level**	**Reference**
I	C	Consensus

7. RUPTURED DESCENDING THORACIC AND THORACO-ABDOMINAL AORTIC ANEURYSMS

Ruptured DTAA and TAAA are lethal without intervention and approximately 40 – 60% of patients die before reaching hospital.[484] When aneurysms of the DTA extend proximally into the distal aortic arch, proximal repair may require inclusion of the aortic arch. The management of aortic arch pathologies is addressed in a separate ESVS/EACTS position paper.[201] This chapter focuses specifically on ruptured thoracic aortic disease due to degenerative aneurysm or chronic dissection, including cases with thoraco-abdominal extension, while the management of acute dissection with rupture is discussed separately (see Chapter 4).

The incidence of ruptured DTAA and TAAA is difficult to determine, largely due to the low rate of postmortem examinations. Consequently, deaths resulting from ruptured DTAA and TAAA may be misclassified, e.g., as cardiac death.[12] While data suggest that hospital admissions for DTAA repair are increasing in some countries, the rate of emergency admissions for ruptured DTAA has been stable.[20,485,486]

Rupture may occur into the mediastinal tissues, pleural space, lung parenchyma, or a combination of these. In patients who survive to hospital presentation, the rupture is often contained by adjacent structures such as the pleura, pericardium, oesophagus, heart, lung, or diaphragmatic crus, preventing immediate exsanguination.[233]

Clinical manifestations typically include chest or back pain and signs of haemodynamic instability such as syncope or hypotension. Less common presentations, such as dysphagia, haemoptysis, or haematemesis, may occur in the setting of primary or secondary aorto-oesophageal or aortobronchial fistulae.[487]

The diagnosis of ruptured DTAA is primarily established by CTA (Fig. 8). The hallmark finding is contrast extravasation from the aortic wall in the setting of an aneurysmal aorta. However, in the absence of overt extravasation, rupture should still be strongly suspected when a DTAA is accompanied by peri-aortic haematoma, haemothorax, or haemomediastinum, and the aorta should be treated as ruptured until proven otherwise.

7.1. Initial management

Patients diagnosed with ruptured DTAA or TAAA should be promptly considered for transfer to a high volume aortic centre.[488,489] The establishment of standardised protocols and transfer pathways facilitates timely and appropriate management of these critically ill patients, similar to existing models for ruptured AAAs.[490] The feasibility and risks of surgical intervention are influenced by both the extent of the aneurysm and the patient's comorbidities. Therefore, early involvement of a multidisciplinary aortic team is essential to guide decisions regarding transfer and treatment strategy.

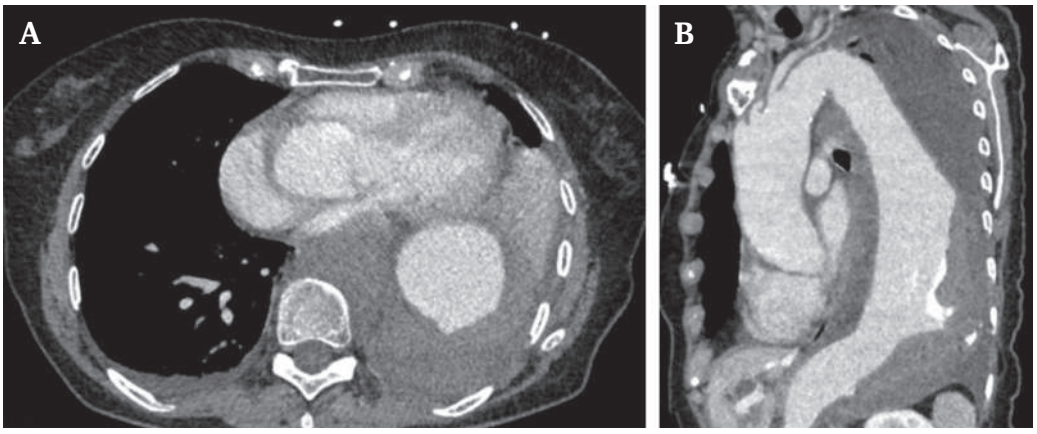


Figure 8. Ruptured thoracic aortic aneurysm with bilateral haemothoraces. (A) Axial computed tomography angiography (CTA) demonstrating rupture of the descending thoracic aortic aneurysm with a large, left sided haemothorax. (B) Sagittal multiplanar reconstruction showing the rupture site, extent of the aneurysm, and associated haemorrhage (artist and copyright Carlota F. Prendes, Uppsala, Sweden).

In patients with significant comorbidities, complex aneurysm morphology, and or advanced age, SDM involving the patient and family may lead to a preference for palliation rather than active intervention. Despite recent advances in treatment, the mortality rate following ruptured DTAA repair remains high including beyond the early post-operative period. Outcomes are particularly poor in patients with end stage renal disease and in octogenarians, who have noticeably limited long term survival after repair.[59,81,491] Even when using less invasive approaches such as TEVAR, the potential for limited long term benefit in frail, elderly patients should be carefully considered, and in selected cases palliation may be the more appropriate course of action[492] (see Recommendation #19).

While studies have demonstrated that transfer of patients with aortic emergencies to high volume centres is safe,[488,493,494] there is a lack of specific evidence regarding optimal management during transfer in ruptured DTAA or TAAA cases. Adherence to a protocol of permissive hypotension, established in the context of infrarenal AAA rupture, appears reasonable.[495,496] This typically involves avoiding aggressive fluid resuscitation and provided the patient remains conscious, maintaining a target SBP of 70 — 90 mmHg until definitive aortic control is achieved.

Recommendation 80 **New**

A policy of permissive hypotension* during the pre-operative period should be considered in patients with a ruptured thoracic or thoraco-abdominal aortic aneurysm, provided adequate end organ perfusion is maintained†.

Class	Level	Reference
IIa	C	Consensus

* By restricting fluid resuscitation.
† Consciousness and ability to speak are practical indicators of adequate cerebral perfusion.

7.2. Endovascular repair

7.2.1. Endovascular repair of ruptured descending thoracic aortic aneurysm. TEVAR is the first line treatment for patients with a ruptured DTAA when anatomically feasible. Epidemiological data show that TEVAR has become the predominant modality for managing ruptured DTAA in many countries.[25] Although RCTs comparing TEVAR and OSR are lacking, registry based studies consistently demonstrate that TEVAR is associated with lower early mortality and complication rates.[497,498] In a multinational analysis involving 13 countries, the peri-operative mortality following TEVAR for ruptured DTAA was 26.8%, with a combined rate of death, paraplegia, stroke, and renal impairment reaching 40.3%.[25] National data from Sweden reported 24 hour and 30 day mortality rates of 10.7% and 20.7%, respectively, among patients undergoing TEVAR for ruptured DTAA.[499] In contrast, OSR is associated with a 30 day mortality ranging from 20% to 45%.[500] A national German analysis based on Diagnosis Related Group (DRG) data reported a significantly lower in hospital mortality rate after TEVAR compared with OSR (22.3% *vs.* 42.9%).[20] Similarly, a systematic review of 18 studies including 2 088 patients found that TEVAR was associated with reduced peri-operative and one year mortality rates, renal impairment, and pulmonary complications. Rates of stroke and paraplegia were similar between groups, although re-intervention was more common after TEVAR.[501] Importantly, the advent of TEVAR has expanded the pool of patients eligible for treatment, as its minimally invasive nature allows intervention in individuals previously considered unfit for OSR.[486]

The technical principles of TEVAR for ruptured DTAA due to degenerative disease are similar to those applied in elective repair of intact DTAA. Access can be achieved either percutaneously or via femoral cutdown, depending on patient anatomy and operator preference.[502] Like

ruptured AAA,[8] TEVAR for ruptured DTAA is preferably performed under local anaesthesia.[503,504]

Data on optimal endograft sizing in the setting of ruptured DTAA remain limited. A recent study in the context of BTAI demonstrated that patients presenting with hypotension (SBP $<$ 90 mmHg, mean arterial pressure $<$ 70 mmHg) had, after resuscitation and TEVAR, a 13% larger proximal aortic diameter compared with pre-resuscitation imaging. In this setting, 30% graft oversizing relative to the pre-resuscitation LZ was shown to be safe.[505] These findings suggest that endograft oversizing should be tailored to the patient's haemodynamic status, with greater oversizing considered when pre-operative imaging is obtained under hypotensive conditions. However, a universally applicable degree of oversizing cannot be recommended, as it depends on both patient specific and device specific factors.

To achieve an adequate sealing zone during TEVAR for ruptured DTAA, supra-aortic branch coverage, primarily the LSA, for proximal sealing,[506] and of the CA for distal sealing,[507] may be required. In urgent settings, selective pre-emptive revascularisation of the LSA is generally reserved for patients with strong indications, such as those with a LIMA coronary bypass, or when the posterior cerebral circulation is dependent on a patent left vertebral artery.[508,509] For more information about LSA revascularisation, see Chapter 3.4. If necessary, primary coverage of the CA, without revascularisation, may be acceptable in the acute setting. In selected cases, revascularisation using a PG, or *in situ* and PMEG fenestration may be considered.[510]

Recommendation 81 New

Thoracic endovascular aortic repair is recommended as the preferred treatment modality for ruptured descending thoracic aortic aneurysm.

Class	Level	References	ToE
I	C	Geisbüsch *et al.* (2019),[20] Ultee *et al.* (2017),[486] Jonker *et al.* (2010),[497] Yamaguchi *et al.* (2019),[498] Kilic *et al.* (2014)[511]	

Recommendation 82 New

Increased endograft oversizing should be considered in emergency endovascular repair of ruptured descending thoracic aortic aneurysm when pre-operative imaging was obtained during hypotension.

Class	Level	Reference
IIa	C	Consensus

7.2.2. Endovascular repair of ruptured thoraco-abdominal aortic aneurysm. Historically, TAAAs were managed with OSR, which, despite its definitive nature, has been associated with substantial peri-operative morbidity and mortality rates. The highly invasive nature of the procedure, along with the requirement for cardiopulmonary bypass and full anticoagulation, renders OSR unsuitable for most patients presenting with ruptured TAAAs. The treatment landscape has been markedly transformed by the advent of FBEVAR, which has expanded the therapeutic options both for intact and ruptured TAAAs.

Few studies have focused specifically on ruptured TAAAs. In a small Swedish study including 11 patients treated with emergency BEVAR for ruptured TAAA, the 30 day mortality rate was 27% (three deaths).[512] More recently, a multicentre transatlantic study including 100 patients with ruptured TAAA treated with a multibranched off the shelf device (Zenith t-Branch; Cook Medical Inc., Bjaeverskov, Denmark) reported a primary technical success rate of 89% and a 30 day or in hospital mortality rate of 24%.[57] Additionally, a multinational study including 416 non-elective TAAA repairs with fenestrated and branched endografts reported an early mortality rate of 17%.[437] Off the shelf multibranched stent grafts provide immediate availability, which is critical in urgent settings; however, their applicability is limited by anatomic constraints, preventing universal use.

In emergencies where off the shelf devices are unavailable or unsuitable, alternative techniques such as PMEGs, *in situ* fenestration, and PGs offer feasible solutions. PMEGs involve on table modification of a standard endograft to accommodate individual anatomy and have demonstrated a technical success rate of 95% and early mortality rate of 10% in urgent cases, according to a recent meta-analysis.[12] PMEGs and *in situ* fenestration are particularly suited for type I TAAA with a narrow aortic lumen at the level of the visceral branches or in post-dissection aneurysms, where close apposition between the graft and aortic wall promotes sealing and bridging stent stability.[117,513] Several series have reported high technical success and favourable early outcomes using PMEGs or *in situ* laser fenestration for urgent TAAA repair in experienced centres.[117,514]

Although PG techniques have demonstrated acceptable technical success and short term survival in the repair of ruptured TAAA, their use remains limited due to the inherent risk of gutter related endoleaks and incomplete aneurysm exclusion.[515]

SCI remains a major complication after acute endovascular TAAA repair, with reported rates ranging from 20 — 30%.[57,512] This is largely attributable to haemodynamic instability at presentation and the need for extensive, non-staged aortic coverage in emergency situations. In selected haemodynamically stable patients with contained rupture and a high anticipated risk of SCI, a staged repair over a short interval, to reduce the risk of SCI, has been suggested.[139]

Recommendation 83 New

Endovascular repair using off the shelf branched stent grafts, physician modified endografts, or *in situ* fenestration techniques should be considered the preferred treatment modality for ruptured thoraco-abdominal aortic aneurysms.

Class	Level	Reference
IIa	C	Consensus

7.2.3. Endovascular repair of ruptured chronic dissection aortic aneurysm. Endovascular repair of ruptured descending or thoraco-abdominal chronic dissection aortic aneurysm presents specific technical challenges. When the dissection is limited to the thoracic aorta, TEVAR typically aims to achieve proximal and distal sealing within non-dissected segments. However, in cases involving thoraco-abdominal dissection with rupture of a post-dissection thoracic aneurysm, adequate sealing may be more difficult to achieve.

In selected patients, TEVAR targeting the primary entry tear and all significant secondary entry tears in the DTA may be sufficient to control the rupture. In other cases, more extensive coverage, from the LSA to the CA, may be preferred to promote aortic remodelling and reduce the likelihood of future re-interventions.[516]

Adjunctive techniques such as FL coiling or occlusion plugs may be required to prevent retrograde flow into the FL and continued pressurisation of a ruptured DTAA in the setting of chronic dissection.[517] FL occlusion can reduce the risk of backflow from distal re-entry tears and is achievable using dedicated FL occluder plugs, which may be commercially available or physician modified from standard stent grafts. An alternative approach is the Knickerbocker technique, which involves intentional rupture of the dissection membrane within the distal thoracic aorta, allowing the stent graft to expand and seal against the outer wall of the FL to prevent retrograde perfusion.[518] Both occluder plug techniques and the Knickerbocker method have been successfully applied in the treatment of chronic dissection related DTAA.[519] The use of TES has recently been described as a technique to disrupt the dissection lamella during endovascular repair of chronic post-dissection aneurysms, enabling the creation of suitable LZs. TES may be particularly useful in the management of ruptured chronic thoraco-abdominal ADs with focal aneurysmal degeneration confined to the thoracic aorta[363] (see Chapter 5.3.3).

Recommendation 84		New
Compartmentalisation of a ruptured post-dissection thoraco-abdominal aortic aneurysm, by false lumen occlusion techniques or focal, controlled rupture of the dissection lamella, may be considered to enable endovascular exclusion of an isolated thoracic rupture.		
Class	**Level**	**Reference**
IIb	C	Consensus

7.3. Open surgical repair

While endovascular repair remains the primary treatment modality for ruptured DTAA and TAAA, OSR may be appropriate in selected cases, such as patients with unfavourable anatomy or younger individuals with genetic aortopathy.[59,520,521] OSR for rupture generally follows the same principles as elective procedures, including the use of distal perfusion strategies during aortic clamping and peri-operative adjuncts to mitigate the risk of SCI. However, due to the emergency nature of these cases, retrospective data indicate that techniques such as CSFD and intercostal artery re-implantation are used less frequently.[522]

Data suggest that OSR for ruptured DTAA and TAAA can be performed with acceptable short term outcomes in experienced high volume centres, with operative mortality rates ranging between 20% and 40%.[498,500,523] For selected patients with TAAA, hybrid repair, combining visceral debranching with thoracic stent grafting, may be considered. However, published outcomes indicate that this technique is associated with a high complication rate.[462]

Recommendation 85		New
Open surgical repair may be considered in selected patients with a ruptured descending thoracic or thoraco-abdominal aortic aneurysm when endovascular repair is not feasible.		
Class	**Level**	**Reference**
IIb	C	Consensus

7.4. Early post-operative complications

Important post-operative complications after ruptured DTAA repair include respiratory failure (2 — 33% after endovascular repair *vs.* 13 — 43% after OSR), renal failure (4 — 22% *vs.* 3 — 25%), stroke (2 — 11% *vs.* 5 — 15%), and paraplegia (2 — 10% *vs.* 5 — 16%).[486,491,522,524,525]

Two complications that warrant specific attention are: (1) respiratory failure associated with haemothorax secondary to aortic rupture; and (2) SCI, resulting from the combination of hypotensive shock and loss of intercostal arteries during emergency DTAA repair.

Limited data suggest that early haemothorax drainage following TEVAR for ruptured DTAA may improve respiratory outcomes.[526,527] The timing must carefully balance the risk of endoleak related bleeding against the potential for respiratory compromise due to retained haemothorax. Early multiphase CTA can be valuable to exclude significant endoleak before chest decompression. While percutaneous drainage is often sufficient, video assisted thoracoscopic surgery (VATS) should be considered in cases with organised haematoma or inadequate percutaneous evacuation. Although the ESVS guidelines on vascular graft infections do not specifically address prophylactic antibiotic use for haemothorax drainage in the setting of ruptured DTAA treated with TEVAR, it is considered prudent to administer prophylactic antibiotics, given the potential risk of direct communication between the skin and the stent graft via the rupture site.[9]

The risk of SCI after ruptured DTAA repair is significantly higher than after intact DTAA repair.[528] In the emergency setting of rupture, prophylactic CSFD is often not feasible. Nevertheless, established post-operative protocols, such as early neurological assessment using a wake up test in sedated patients, can facilitate timely recognition of SCI. This enables prompt initiation of adjunctive measures, including CSFD, revascularisation of important collaterals, and haemodynamic optimisation to support spinal perfusion and potentially reverse neurological deficits.[128,529] For more information on SCI, see Chapter 3.5.

Recommendation 86		New
Haemothorax drainage may be considered after endovascular repair of a ruptured descending thoracic aorta once the patient is haemodynamically stable.		
Class	**Level**	**Reference**
IIb	C	Consensus

Recommendation 87		New
Video assisted thoracoscopic decompression of haemothorax should be considered for organised haematoma or inadequate percutaneous drainage cases after endovascular ruptured descending thoracic aorta repair.		
Class	**Level**	**Reference**
IIa	C	Consensus

8. BLUNT THORACIC AORTIC INJURY

BTAI most commonly results from sudden deceleration associated with high energy mechanisms, such as motor vehicle collisions, falls from heights, or automobile pedestrian, or bicycle accidents.[530,531] In the setting of war or terrorism, the incidence of BTAI depends on the mechanism of injury, with high energy explosives being more likely to cause BTAI. BTAI remains a leading cause of death following blunt trauma. Recent data indicate that 79% of patients with BTAI die at the scene or during transport. Among those who survive to hospital admission, in hospital mortality ranges from 19 — 31% primarily due to associated extra-aortic injuries.[532,533] Overall mortality from BTAI approaches 85%. Advances in pre-hospital care, earlier diagnosis, and rapid intervention have increased the likelihood of successful repair in patients who survive the initial trauma.

The aortic isthmus is the most frequently affected site, accounting for approximately 80 — 90% of BTAIs in patients who reach hospital alive.[534] Other segments of the thoracic aorta are involved less commonly.[535] The mechanism of injury in BTAI involves a combination of shear, torsion, stretch, and hydrostatic forces. These forces typically act together and are attributed to the differential mobility between the relatively fixed DTA and the more mobile aortic arch.[535] The resulting aortic injury may be partial or circumferential. In cases where complete transection and rupture do not occur, the damage often begins with disruption of the intima and media layers, with the potential for progressive extension into the adventitia over time.[535]

Several classification systems for BTAI have been proposed and are summarised in Table 9.[2,536,537] The most recent ESVS classification closely aligns with the North American SVS and Harborview classifications. To facilitate consistency in data reporting and clinical decision making across vascular trauma studies, use of the ESVS classification (Fig. 9) is recommended. Given that patients with BTAI often present with multiple concomitant injuries, the use of injury severity scoring systems is essential for risk stratification, outcome comparison, and standardised reporting. The Injury Severity Score (ISS) is the most widely used and internationally accepted tool for this purpose.[530,531]

The clinical presentation of BTAI ranges from asymptomatic to severe, including chest pain, back pain, and shortness of breath. On physical examination, findings may include signs of chest wall trauma, a systolic murmur, pseudocoarctation syndrome, or neurological deficits such as paraplegia. Risk assessment begins with a high index of suspicion, primarily based on the mechanism of injury. High risk scenarios include high energy blunt trauma and absence of seatbelt or airbag use in motor vehicle accidents. Presence of associated thoracic or abdominal injuries further raises the suspicion of BTAI.[531]

CTA is the recommended imaging modality for the evaluation of suspected BTAI.[538] It is fast, widely available, and highly reproducible, with a sensitivity and specificity approaching 100% for the detection of BTAI. In addition to its diagnostic accuracy for vascular injury, CTA is also well suited for comprehensive assessment of concurrent non-arterial injuries, including trauma to the brain, spine, pelvis, spleen, liver, and kidneys, making it the preferred imaging modality in trauma patients. CTA findings suggestive of BTAI include mediastinal haematoma, haemothorax, localised dissection with a double lumen, pseudoaneurysm

Table 9. European Society for Vascular Surgery (ESVS), North American Society for Vascular Surgery (SVS), and Harborview classifications of blunt thoracic aortic injury.

ESVS[2]		SVS[536]		Harborview[537]	
Grade	**Injury**	**Grade**	**Injury**	**Grade**	**Injury**
1	Partial wall injury Normal external wall contour	I II	Intimal tear Intramural haematoma	Minimal	No external contour abnormality Intimal tear and or thrombus is <10 mm
2	Complete wall injury Abnormal external wall contour Contained bleeding	III	Pseudoaneurysm	Moderate	External contour abnormality or intimal tear >10 mm
3	Complete wall injury Uncontained haemorrhage	IV	Rupture	Severe	Active extravasation LSA haematoma >15 mm

LSA = left subclavian artery.

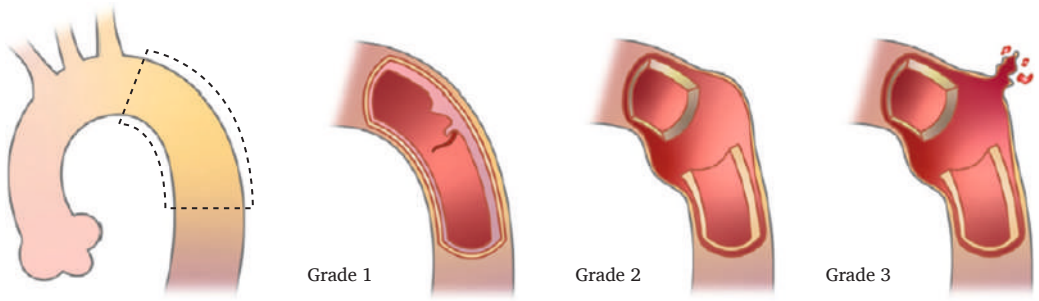


Figure 9. European Society for Vascular Surgery (ESVS) classification of traumatic thoracic aortic injury: ESVS grade 1, injury confined to the intima or vessel wall with normal external wall contour; ESVS grade 2, abnormal external wall contour or external wall disruption with contained haemorrhage (e.g., pseudoaneurysm); and ESVS grade 3, complete wall transection with free rupture.[2]

formation, aortic wall contour irregularity, and the presence of an intimal flap or intraluminal thrombus.[530] Caution is warranted when interpreting aortic diameter measurements on acute CTA, particularly in haemodynamically unstable patients, as it may lead to underestimation of the true luminal diameter.[539] Furthermore, if the CTA is not ECG gated, aortic dimensions may vary substantially throughout the cardiac cycle. This is especially relevant in young patients with BTAI who typically have a healthy and compliant aortic wall. In such cases, aortic pulsatile changes may result in diameter variations of 10 – 18% between systole and diastole.[539]

8.1. Indication and timing of surgical treatment

Management of BTAI should be individualised based on the severity of the aortic lesion and concomitant injuries, in accordance with the principles of trauma resuscitation and damage control — treat first what kills first![540]

Both prognosis and treatment strategy are strongly influenced by the grade of aortic injury. A systematic review of 35 studies including 2 897 patients reported an aortic related mortality rate of 1% for SVS grade I – II injuries, compared with 18% for grades III – IV, underscoring the prognostic significance of injury grading in guiding treatment decisions.[541] A retrospective analysis from the VQI, including 1 311 patients, found low in hospital aortic related mortality with non-operative management in SVS grade I (2.4%) and grade II (0.9%) injuries, supporting a conservative approach for low grade BTAI.[542]

Therefore, ESVS grade 1 injuries (corresponding to SVS grades I and II) should be managed conservatively, with close clinical and imaging surveillance and optimal medical therapy.[543,544] Optimal medical therapy consists of anti-impulse control, targeting a SBP of 90 – 110 mmHg and a heart rate < 100 bpm, to reduce aortic wall stress and promote stabilisation of the lesion.[538,545] For patients managed conservatively, disease progression should be evaluated with a follow up CTA performed 48 – 72 hours after admission.

In cases of concomitant BTAI and traumatic brain injury, early TEVAR has been a subject of debate. A retrospective single centre study found early TEVAR (< 24 hours) was associated with worsening brain injury and poorer aortic related outcomes.[546] However, more recent data from the Aortic Trauma Foundation contradicted these findings, showing that neurological and aortic outcomes were not significantly affected by the timing of TEVAR, thereby supporting an individualised approach based on clinical factors such as haemodynamic stability and the severity of associated injuries.[547]

In the setting of severe traumatic brain injury, where standard anti-impulse therapy for BTAI, such as strict blood pressure control, may compromise cerebral perfusion, TEVAR may be considered even for ESVS grade 1 lesions to enable maintenance of higher cerebral perfusion pressures without increasing the risk of aortic injury progression.

Surgical repair is indicated for ESVS grade 2 injuries (corresponding to SVS grade III); however, the optimal timing of intervention remains controversial and should be guided by the presence of concomitant injuries, patient stability, and anatomic characteristics.

While immediate repair was previously considered the standard of care, more recent evidence suggests that delayed intervention may be associated with reduced morbidity and mortality rates.[548–550] Delaying repair allows for haemodynamic stabilisation and prioritisation of other life threatening injuries, supporting a more individualised, staged approach. A systematic review and meta-analysis published by the Eastern Association for the Surgery of Trauma in 2015 suggested that delaying aortic repair may be beneficial, particularly in patients with serious concomitant injuries, provided that effective blood pressure control is maintained in the interim.[548] However, a key limitation of the available evidence is that most studies supporting delayed intervention did not stratify outcomes based on the grade of aortic injury. A recent observational study demonstrated that, after propensity score matching, a delayed approach to repair, compared with immediate intervention, was associated with a lower in hospital mortality rate, shorter overall hospital and ICU length of stay, and fewer days on a ventilator. These findings support the importance of individualised assessment in patients with ESVS grade 2 (SVS grade III) injuries.[551]

Clinical and anatomic high risk features of a more severe injury include left sided haemothorax, large mediastinal haematoma, aortic coarctation, large pseudoaneurysm, hypotension (SBP <90 mmHg), and associated traumatic brain injury.[552–557] Accordingly, for ESVS grade 2 injuries with high risk features, urgent endovascular aortic repair (<24 hours) is recommended, as delaying intervention may increase the risk of aortic rupture and death. Conversely, in the absence of high risk features, a delayed repair (>24 hours) with close monitoring may be considered to allow for stabilisation and treatment of other life threatening injuries. However, if no other serious concomitant injuries are present, there is little justification for postponing surgical repair.

Immediate surgical repair is recommended for ESVS grade 3 injuries (corresponding to SVS grade IV).[548,550]

Recommendation 88 — **New**

Non-operative management, with blood pressure control and follow up imaging to assess lesion stability, is recommended for ESVS grade 1 blunt thoracic aortic injury without concomitant traumatic brain injury.

Class	Level	References	ToE
I	C	Jacob-Brassard *et al.* (2019),[538] Soong *et al.* (2019),[541] Yadavalli *et al.* (2023),[542] Golestani *et al.* (2024),[543] De Freitas *et al.* (2024)[544]	

Recommendation 89 — **New**

Thoracic endovascular aortic repair may be considered for ESVS grade 1 blunt thoracic aortic injury and presence of concomitant traumatic brain injury to permit higher cerebral perfusion pressures when strict blood pressure control is not feasible.

Class	Level	Reference
IIb	C	Consensus

Recommendation 90 — **Changed**

Delayed thoracic endovascular aortic repair (> 24 hours) should be considered for ESVS grade 2 blunt traumatic aortic injury without high risk features*.

Class	Level	References	ToE
IIa	C	Jacob-Brassard *et al.* (2019),[538] Soong *et al.* (2019),[541] Fox *et al.* (2015),[548] Marcaccio *et al.* (2018),[550] Romijn *et al.* (2023),[551] Starnes *et al.* (2012),[556] Yadavalli *et al.* (2024)[558]	

* Left haemothorax, large mediastinal haematomas, aortic coarctation, large pseudoaneurysms, hypotension (systolic blood pressure <90 mmHg), and associated traumatic brain injury.

Recommendation 91 — **Changed**

Urgent thoracic endovascular aortic repair (< 24 hours) is recommended for ESVS grade 2 blunt traumatic aortic injury presenting with high risk features*.

Class	Level	References	ToE
I	C	Jacob-Brassard *et al.* (2019),[538] Soong *et al.* (2019),[541] Fox *et al.* (2015),[548] Romijn *et al.* (2023),[551] Starnes *et al.* (2012),[556] Yadavalli *et al.* (2024)[558]	

* Left haemothorax, large mediastinal haematomas, aortic coarctation, large pseudoaneurysms, hypotension (systolic blood pressure <90 mmHg), and associated traumatic brain injury.

Recommendation 92 — **Unchanged**

Immediate thoracic endovascular aortic repair is recommended for ESVS grade 3 blunt thoracic aortic injury.

Class	Level	References	ToE
I	C	Jacob-Brassard *et al.* (2019),[538] Soong *et al.* (2019),[541] Fox *et al.* (2015)[548]	

8.2. Endovascular repair

Endovascular repair is associated with significantly better early outcomes than OSR in patients with BTAI.[559,560] Hence, TEVAR is recommended as the first line treatment for BTAI,[561] while OSR is reserved for young patients with unsuitable aortic anatomy.[562]

A systematic review of 74 studies involving 1 882 patients with BTAI treated endovascularly reported an early (30 day or in hospital) mortality rate of 7.5%, which correlated with trauma severity as assessed by the ISS. Primary technical success was achieved in 98.4% of cases, with an endograft related early mortality rate of 0.8%. The rates of paraplegia and stroke were 0.4% and 1.5%, respectively. LSA coverage was required in 39.6% of cases, while pre-emptive revascularisation was performed in only 4.3%. Post-operative symptoms following LSA coverage occurred in 5.3% of patients, primarily presenting as left upper limb ischaemia (4%) or subclavian steal syndrome (0.8%). The stroke rate did not differ significantly between patients with LSA coverage (2.0%) and those without (1.3%) (for detailed guidance on LSA coverage management, see Chapter 4). After a mean follow up of 3.0 years, major stent graft related complications, including significant endoleak, endograft collapse, or graft infection requiring conversion to OSR with explantation, occurred in 6.9% of patients, with a 1% procedure related mortality rate.[563] An international registry study, including non-selected data from 1 108 real world patients across 13 countries, similarly demonstrated a correlation between death and the severity of aortic injury.[25] A recent meta-analysis also

confirmed high technical success rates, low cerebrovascular morbidity and mortality, and low midterm re-intervention rates.[436]

Although long term outcome data are limited, available evidence suggests low rates of device related complications and death and a low rate of re-intervention.[564,565] In a recent Swedish observational study, including 95 patients who underwent endovascular repair for BTAI at four tertiary trauma referral centres, the estimated overall survival was 57% at 15 years follow up. Aortic re-intervention (re-stenting, coiling, or explantation) was performed in 14 patients (16%), six of whom underwent stent graft explantation. Seven of the 14 patients (50%) who underwent aortic re-intervention presented with symptoms, and six had a device related complication. All complications that required aortic re-intervention were diagnosed within 18 months of the index procedure.[533]

Several approved devices are available and may be used for this purpose.[566–568] Optimal endograft oversizing in BTAI remains controversial due to several factors. Hypotension at the time of imaging may lead to underestimation of aortic diameter, resulting in inadvertent undersizing of the endograft and an increased risk of migration, endoleak, or device collapse following haemodynamic stabilisation.[548–550] Conversely, excessive oversizing (> 20%) has also been associated with an increased risk of endograft collapse, particularly when based on pre-operative CTA measurements obtained under normotensive conditions.[569] To balance these risks, an endograft oversizing range of 10 – 20% is usually appropriate, with the higher end of the range applied in patients experiencing hypovolaemic shock at the time of imaging.[570,571] See also Chapter 7 on DTAA and TAAA rupture. The small, angulated aorta seen in these often very young patients increases the risk of bird's beak and graft collapse. Newer generation endografts with improved flexibility and smaller profiles may help mitigate this issue.

Certain clinical scenarios pose significant technical challenges; in paediatric patients, no dedicated thoracic stent grafts are currently available with lengths shorter than 10 cm or diameters less than 21 mm. As an alternative, the use of covered balloon expandable stents has been described; these devices can accommodate smaller aortic diameters and allow for subsequent re-dilation to match somatic growth.[572] Another challenging context involves young patients who frequently present with an acutely angulated ("gothic") aortic arch. This anatomic configuration increases the risk of proximal stent graft malapposition (bird beaking), which may predispose to type I endoleak and late stent graft collapse.[566–568]

Systemic heparinisation during TEVAR may be contraindicated in the setting of associated injuries, particularly traumatic brain injury. A retrospective analysis of 77 patients subjected to TEVAR under full, low dose, and no intra-operative systemic heparinisation demonstrated no clear benefit from heparin use in this context; in fact, omission of heparin was associated with shorter procedure times and a favourable safety profile in trauma patients.[573] Therefore, the decision to administer intra-operative systemic heparin should be individualised, taking into account the risk of bleeding, potential for thromboembolic events, and the severity of concomitant cerebral injury.

Recommendation 93 — Unchanged

Thoracic endovascular aortic repair is recommended as the first line treatment for blunt thoracic aortic injury.

Class	Level	References	ToE
I	C	Mill *et al.* (2025),[533] Demetriades *et al.* (2008),[559] Tang *et al.* (2008),[560] van der Zee *et al.* (2019),[563] Gennai *et al.* (2023),[564] Takagi *et al.* (2008)[574]	

Recommendation 94 — New

Systemic heparinisation during thoracic endovascular aortic repair for blunt traumatic aortic injury should be considered individualised, taking into account the risk of bleeding, thromboembolic complications, and the severity of any associated traumatic brain injury.

Class	Level	Reference
IIa	C	Consensus

Recommendation 95 — New

Moderate stent graft oversizing should be considered in emergency endovascular repair of blunt thoracic aortic injury when pre-operative imaging was obtained during hypotension.

Class	Level	References	ToE
IIa	C	Jonker *et al.* (2010),[497] Garcia Reyes *et al.* (2018),[569] Muhs *et al.* (2007)[570]	

8.3. Open surgical repair

Given the superior outcomes associated with TEVAR, OSR is reserved for selected patients with anatomies unsuitable for endovascular treatment. OSR involves a left thoracotomy, single lung ventilation, and direct repair or replacement of the injured aortic segment with a synthetic graft.[575] The traditional clamp and sew technique, performed without distal organ perfusion, is associated with high mortality and paraplegia rates and is therefore generally discouraged. Instead, distal aortic perfusion techniques, such as partial cardiopulmonary bypass, are used during OSR for BTAI, as these strategies have been shown to reduce the risk of SCI.[2,575]

8.4. Follow up

Surveillance should include monitoring for potential graft related complications such as endoleak, stent graft migration, re-stenosis (which may present as "pseudocoarctation"), thrombus formation, and stent induced

dissection.[576,577] The implantation of a rigid endograft alters the biomechanical properties of the aorta, which may, over time, lead to systemic cardiovascular effects.[578–580] Increased aortic stiffness has been associated with elevated blood pressure and may contribute to progressive aortic and cardiac damage, underscoring the need for active screening and appropriate management.[579,580] Finally, 2–8% of patients with initially unrecognised BTAI may develop a chronic post-traumatic pseudoaneurysm during follow up.[581]

To reduce cumulative radiation exposure, particularly relevant in younger patients, MRI should be considered as an alternative to CTA whenever clinically feasible. Aortic endografts, including those made with stainless steel, are considered MRI conditional. However, stainless steel stents cause significant artefacts on MRI owing to their ferromagnetic properties. These artefacts can appear as signal voids or distortions, making it difficult or impossible to assess the stent graft and the surrounding tissues. For some patients, particularly those with stainless steel stents and a need for detailed post-operative surveillance, CTA might therefore be preferred due to its lower susceptibility to artefacts.

Serial follow up imaging is recommended for conservatively managed BTAI (i.e., without endovascular stent graft repair) until aortic remodelling is confirmed. A suggested follow up schedule includes imaging 48–72 hours after admission and at one week, one month, 12 months, and annually thereafter (Fig. 10), ideally using MRA to avoid cumulative radiation exposure.

Following endovascular stent graft repair of BTAI, follow up imaging is recommended at one month, one year, and annually thereafter for a minimum of five years, followed by continued imaging follow up every five to ten years for life (Fig. 10).[41,533,576]

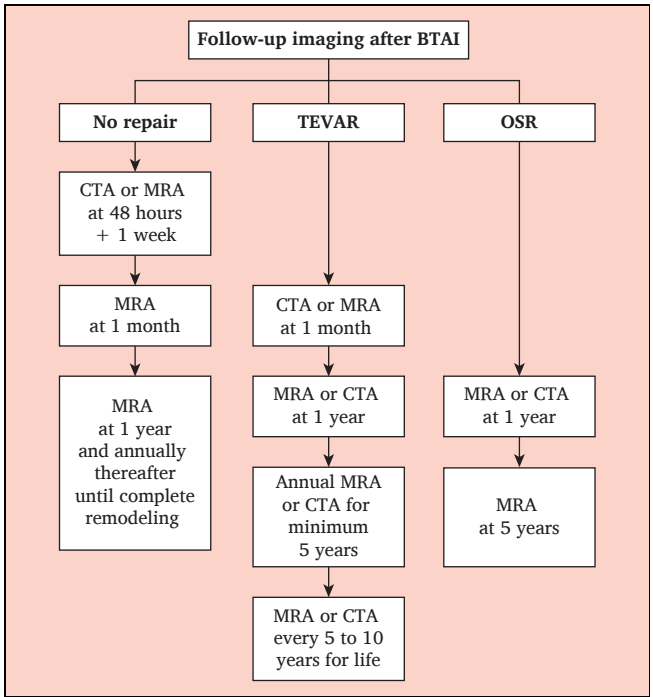


Figure 10. Proposed follow up protocol after blunt thoracic aortic injury (BTAI). TEVAR = thoracic endovascular aortic repair; OSR = open surgical repair; CTA = computed tomography angiography; MRA = magnetic resonance angiography.

Recommendation 96 — New

Continued medical therapy with anti-impulse treatment and serial imaging until aortic remodelling is recommended for patients with blunt thoracic aortic injuries managed non-operatively.

Class	Level	Reference
I	C	Consensus

Recommendation 97 — New

After endovascular stent graft repair of blunt thoracic aortic injury, follow up imaging is recommended at one month, one year, and annually thereafter for a minimum of five years, followed by continued imaging follow up every five to ten years for life.

Class	Level	Reference
I	C	Consensus

Recommendation 98 — New

Magnetic resonance angiography should be considered the preferred imaging modality for long term surveillance after blunt thoracic aortic injury, whenever feasible.

Class	Level	Reference
IIa	C	Consensus

9. POST-OPERATIVE COMPLICATIONS

All patients undergoing DTA and TAbdAo repair, whether open or endovascular, are at risk of both long term complications of the repair and or progression of residual aortic pathology, which warrants lifelong follow up.

Complications following OSR include graft infection and anastomotic pseudoaneurysm. Additionally, progressive aneurysmal degeneration of adjacent or remote aortic segments is also common. After endovascular repair, patients face risks such as RTAAD, endoleaks, dSINE, malperfusion, endograft migration, fracture collapse (i.e., bird beak configuration), endograft infection, and bridging stent graft or target vessel instability (i.e., kinking, stenosis, and occlusion), as well as aortic dilation and dissection progression. These events often require re-intervention and may adversely impact long term survival.[201,582,583]

Therefore, a structured surveillance programme is essential. As no standardised protocol has proven superior, follow up should be individualised based on patient factors, aortic pathology, treatment modality, and interim findings.

CT is generally the preferred imaging modality. Baseline CTA for follow up should include three phases: (1) a non-contrast phase to distinguish calcifications from endoleaks; (2) an arterial phase to detect early or direct endoleaks; and (3) a venous or delayed phase to identify

delayed endoleaks and signs of malperfusion. Additional imaging techniques may be required for clarification of specific findings.

9.1. Endoleaks

Endoleaks are common after complex EVAR, observed in 10 — 20% during follow up.[86,584] They represent an important concern, requiring close surveillance and management owing to the risk of aneurysm expansion and rupture.

Endoleaks are classified into five types (Fig. 11):[86] type I endoleak is a leak at the proximal (Ia) or distal (Ib) attachment of the main aortic stent graft, or at side branch attachment (Ic); type II endoleak is a retrograde leak from branch vessels, such as lumbar arteries or the inferior mesenteric artery, into the aneurysm sac; type III endoleak is a leak in the connection between aortic stent graft components (IIIa), due to a defect in the graft fabric (IIIb), or in the attachment of aortic side branches or between side branch components (IIIc); type IV endoleak, related to endograft porosity; and type V endoleak, caused by endotension.

Temporally, a distinction is made between primary endoleaks present at the time of the procedure, and secondary endoleaks that develop during follow up.

Retrograde FL perfusion via aortic branches or distal entry tears beyond the endograft should not be classified as endoleaks. Additionally, FL re-perfusion caused by membrane rupture at the distal graft edge should be described as dSINE, not as an endoleak.[585]

CTA is the primary imaging modality for detecting and characterising endoleaks, especially types I and III, and often type II. Accurate characterisation (type and source) is critical for guiding management. Duplex ultrasound (DUS), especially when enhanced with contrast (contrast enhanced ultrasound [CEUS]), can complement CTA in follow up of repairs involving the abdominal aorta and its branches.

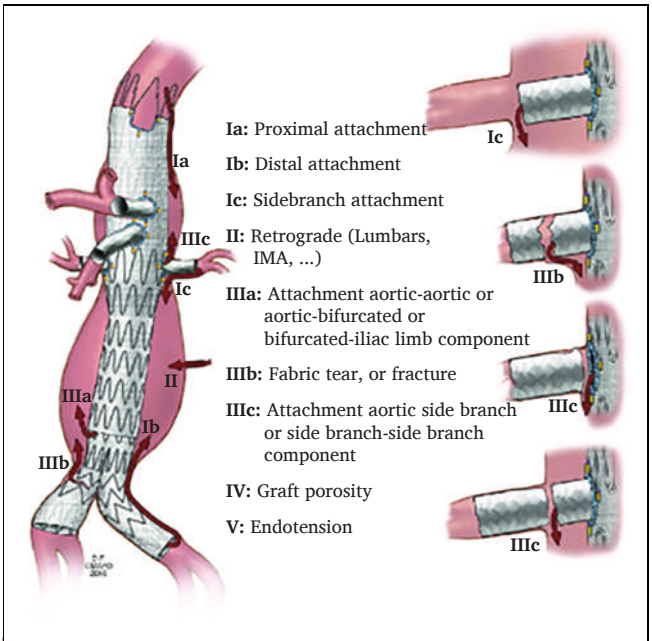


Figure 11. Endoleak classification by Kärkkäinen *et al.* IMA = inferior mesenteric artery. Permission to reproduce granted from Elsevier *J Vasc Surg*.[144]

CEUS improves DUS sensitivity (up to 0.98) but may slightly reduce specificity (to 0.88). CEUS has shown superior sensitivity over CTA for detecting endoleaks and identifying their source and type. However, ultrasound contrast agents (e.g., perfluorocarbon, sulphur hexafluoride) are contraindicated in patients with unstable angina, recent acute coronary syndrome, or severe pulmonary hypertension.[586,587]

Meta-analyses suggest that CEUS has a pooled sensitivity and specificity of 0.94 and 0.93, respectively, for all endoleaks, and 0.97 and 1.00 for types I and III. While DUS and MRI may detect type II endoleaks more sensitively than CTA, they offer no advantage for types I and III. In contrast, CEUS has demonstrated equivalence to CTA, outperforming DUS in identifying clinically significant endoleaks requiring re-intervention.[588—590]

Dynamic (time resolved) CTA is useful when endoleak origin or classification is uncertain, as it helps differentiate between direct (types I and III) and type II endoleaks. However, standardised protocols are lacking, and further validation is needed.

9.1.1. Direct endoleaks. Type I and III endoleaks involve persistent pressurisation of the aneurysm sac and are associated with sac expansion and rupture, warranting close surveillance and often prompt intervention.[584]

Type I endoleaks result from inadequate sealing at the proximal (type Ia; 0.2 — 10%) or distal (type Ib; up to 6.6%) LZs.[584,591] A common cause of type Ia endoleak is the bird beak configuration (graft malapposition due to insufficient conformability and hostile aortic anatomy), present in up to 44% of TEVARs.[582,592] Computational models have identified both diameter and curvature mismatch as key contributors.[593] Short proximal LZs, multiple components, and arch involvement are also risk factors. Progressive aortic dilation may lead to delayed type I endoleak, emphasising the need for surveillance and early re-intervention in cases with compromised sealing zones.[594]

Type III endoleaks, caused by component separation or fabric disruption, may affect up to 11% of patients and account for 80% of all endoleaks. They commonly occur at mesenteric vessels, particularly the CA or superior mesenteric artery, especially where multiple bridging stents are used or in directional branches. Renal artery involvement is rare.[595,596]

Management options are largely based on case series and include endovascular techniques such as ballooning and or stent graft extension or relining. Treatment must be individualised, and in complex cases hybrid or OSR may be required.[584,597] 3D-CTA based analysis may aid in identifying underlying mechanisms and guiding targeted intervention.[598]

9.1.2. Indirect endoleaks. Type II endoleaks, caused by retrograde flow from patent aortic branches within the aneurysm sac, occur in 3.3 — 8.7% of TEVAR patients and

6 — 29% of those undergoing complex endovascular repair. While often benign, up to 30% require intervention, and as many as 20% may be associated with aneurysm related death following secondary rupture.[599,600] After TEVAR, the LSA (43%), intercostal and bronchial vessels (43%), and visceral arteries (13%) are the most common sources, with subclavian related type II endoleaks having higher re-intervention rates. The timing of onset is relevant: early type II endoleaks (< one year) resolve spontaneously in about 75% of cases, while late type II endoleaks (> one year) resolve in only about 29% and more often require embolisation (55% *vs.* 8%).[601] Persistent type II endoleaks (> six months) are linked to older age, large collateral vessels, internal iliac artery occlusion, and distal graft extensions.

Although specific thresholds are lacking, intervention is justified for aneurysm sac growth > 10 mm, which can be attributed to type II endoleak and provided alternative causes including type I or III endoleaks have been excluded, similar to the ESVS 2024 clinical practice guidelines for AAAs.[8] Treatment options include endovascular embolisation (via transarterial or direct sac puncture) or surgical ligation (open, laparoscopic, or VATS).[600] The choice of approach should be individualised based on anatomy and centre expertise.

Type IV and V endoleaks are rare with modern stent graft designs. If persistent and or associated with significant sac growth, efforts should be made to rule out other sources of endoleak or low grade endograft infection.[602]

Recommendation 99		**Changed**
Direct (type I and III) endoleak after thoracic or thoraco-abdominal endovascular repair should be considered for re-intervention.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 100		**New**
Compromised or inadequate landing zones without visible endoleak after thoracic or thoraco-abdominal endovascular repair may be considered for re-intervention to improve seal.		
Class	**Level**	**Reference**
IIb	C	Consensus

Recommendation 101		**New**
Type II endoleak after thoracic or thoraco-abdominal endovascular aortic repair may be considered for re-intervention in the presence of significant (≥ 1.0 cm) aneurysm sac growth, after ruling out type I and III endoleaks.		
Class	**Level**	**Reference**
IIb	C	Consensus

9.2. Distal stent graft induced new entry

dSINE is an increasingly recognised complication after TEVAR for chronic AD and refers to a new intimal tear at the distal end of a thoracic endograft, allowing re-entry into the FL and leading to FL pressurisation and aneurysm progression (Fig. 12).

The reported incidence of dSINE originates from retrospective, single centre case series and varies substantially, up to 18%. In a systematic review that analysed 17 studies assessing the data of 3 962 patients, the pooled proportion of dSINE in TBAD cases treated with TEVAR was 10%. The chronicity of TBAD and excessive distal oversizing ratio were demonstrated to be positively associated with dSINE with odds ratios of 2.25 and 2.06, respectively. Endografts with connecting bars were also positively associated with dSINE, but this lacked statistical significance.[603] Aortic wall fragility has been suggested as a risk factor for dSINE, with a reported incidence of 63% in patients with MFS *vs.* 27% in non-genetic aortopathy patients with AD.[604] Other reported risk factors are hostile distal sealing zone morphology, with the elastic recoil (spring back) of straight

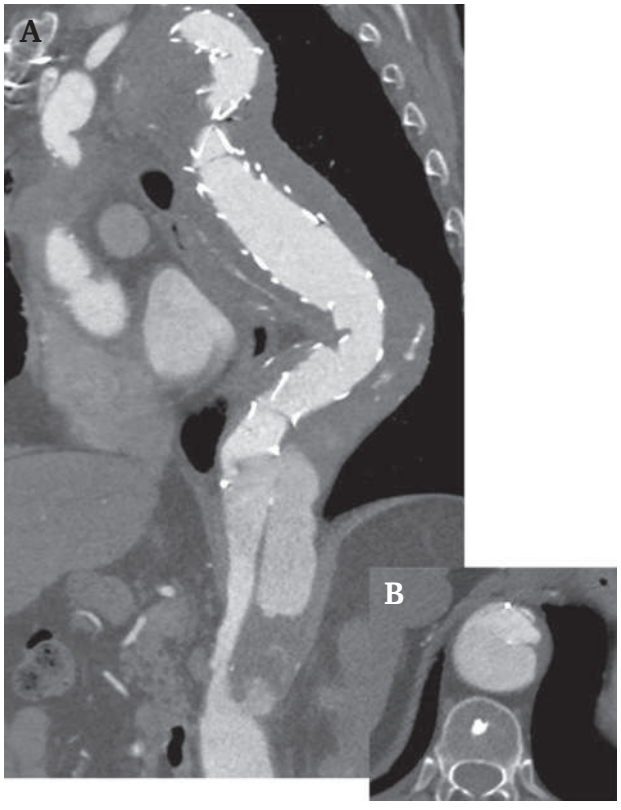


Figure 12. Post-operative computed tomography angiography (CTA) demonstrating a distal stent induced new entry (SINE). (A) Sagittal multiphase reconstruction (MPR) showing a distal SINE originating at the distal end of the thoracic stent graft. (B) Axial CTA image highlighting the precise interface between the distal graft edge and the native aortic wall, corresponding to the site of the SINE (artist and copyright Carlota F. Prendes, Uppsala, Sweden).

thoracic endografts landed in a curved and sometimes tortuous aorta, which may generate significant biomechanical stress along the outer curve of the distal LZ.[605,606]

dSINE should be considered for re-intervention, especially in cases of FL reperfusion with new onset of symptoms, sac expansion, malperfusion, or contained rupture. Treatment typically involves distal endograft extension to seal the new entry and promote FL thrombosis.[607,608]

Recommendation 102		New
Distal stent graft induced new entry should be considered for re-intervention with distal endograft extension to seal the defect.		
Class	**Level**	**Reference**
IIa	C	Consensus

10. MISCELLANEOUS

10.1. Genetic aortopathy

Heritable thoracic aortic disease (HTAD) refers to a clinically and genetically heterogeneous group of inherited disorders characterised by aneurysm or dissection of the thoracic aorta. HTAD includes familial forms, confirmed genetic conditions (familial or sporadic), and syndromes associated with increased thoracic aortic risk. Clinically, HTADs are classified as syndromic or non-syndromic. Genetic aortic syndrome is sometimes used synonymously with HTAD, but more often refers to the syndromic subset of HTAD. Genetic aortopathy is a broader term encompassing both hereditary and non-hereditary genetic diseases that predispose individuals to aortic problems such as aneurysms and dissections. Thus, every HTAD is a form of genetic aortopathy, but not every genetic aortopathy is HTAD.

This chapter discusses a selection of the most common genetic aortopathies, such as MFS, LDS, vEDS, and non-syndromic familial thoracic aortic disease.

Due to their rarity, evidence is limited for many clinical scenarios, such as treatment thresholds, surgical approach, endovascular *vs.* OSR, and pregnancy management. Given the diagnostic complexity, variable progression, and treatment response, an individualised approach is essential. Referral to a multidisciplinary aortic team at a specialised centre is recommended for patients with suspected genetic aortopathy.

Young patients (<60 years) with thoracic aortic pathology and those with a positive family history of aneurysm or dissection or with physical features associated with monogenetic syndromes (loose skin, joint hypermobility, multiple or atypical aneurysms) should undergo genetic counselling and genetic testing to guide management and identify at risk family members.[609]

10.1.1. Marfan syndrome. MFS, a heritable disorder caused by mutations in the fibrillin-1 (*FBN1*) gene on chromosome 15, has a reported prevalence of 1.5 — 17.2 per 100 000 individuals. It is inherited in an autosomal dominant pattern, with a 50% transmission risk. In about 25% of cases MFS results from a *de novo* mutation, often associated with a more severe phenotype.

MFS is diagnosed using clinical, imaging, and genetic criteria. The Ghent criteria, introduced in 1996, define phenotype and genotype characteristics based on medical history, physical examination, imaging, and genetic testing.[610,611] MFS is a systemic disorder of variable severity, commonly affecting the heart valves and aortic root, and is associated with an increased risk of aortic aneurysm and dissection. Beyond the cardiovascular system, multiple organ systems are often affected, such as the eyes and skeleton.

TAAD is the most serious complication. Aortic root diameter is the major predictor of TAAD, with rupture risk increasing above 50 mm.[612] Additional risk factors include rapid aortic growth (≥ 3 mm/year), family history of dissection at small diameter, pregnancy, and hypertension. TBAD is also common and may occur at smaller diameters, below standard surgical thresholds. Previous aortic root replacement or mitral valve surgery is associated with increased risk of TBAD.

Annual TTE imaging surveillance of the aortic root and ascending aorta[611] and periodical evaluation of the entire aorta and peripheral arteries with MRA or CTA and DUS (every three to five years depending on disease progression) is indicated.[611,613,614]

Preventive medical therapy with beta blockers and angiotensin II receptor blockers to reduce aortic growth and complications is standard care.[615—618]

Prophylactic aortic root surgery is commonly performed at diameters ≥ 50 mm, with earlier intervention considered in cases of rapid growth (≥ 5 mm/year), aortic regurgitation, or family history of dissection. Pregnancy is associated with an increased risk of AD in women with MFS, with reported risk increases of up to eightfold. In this context, pre-conception genetic counselling and aortic imaging are routinely advised (see Chapter 3.2), and some guidelines suggest prophylactic root surgery at diameters >4.0 — 4.5 cm in women planning pregnancy.[11] Similarly, prophylactic repair of aneurysms involving the ascending aorta, arch, descending thoracic, or abdominal aorta is often considered at threshold diameters ≥ 50 mm.[11,619]

10.1.2. Loeys—Dietz syndrome. LDS is a rare autosomal dominant disorder caused by mutations in genes within the transforming growth factor beta (TGF-β) pathway; *TGFBR1*, *TGFBR2*, *SMAD2*, *SMAD3*, *TGFB2*, or *TGFB3*. Phenotypic variation between genotypes is clinically relevant and influences thresholds for prophylactic aortic intervention.[609,620—622] It is characterised by arterial tortuosity, aortic root aneurysms, and a high risk of dissection or rupture at young age and small diameters.[623] Many patients experience premature death, frequently before 50 years of age.[624]

Although no LDS specific trials exist, beta blockers are often used to reduce aortic wall stress. Initial TTE with six month follow up is advised, with annual CTA or MRA thereafter to monitor the entire aorta and peripheral

vessels. Cerebral aneurysms occur in 10 − 18% of patients and require tailored surveillance.[624]

Prophylactic aortic root or ascending aortic surgery is indicated at > 4.0 − 4.5 cm, depending on the pathogenic variant and associated risk features, such as female sex and small body size, family history of AD, and growth rate.[11] Prophylactic repair of aneurysms involving the arch, descending thoracic, or abdominal aorta should be considered at diameters > 4.5 − 5.0 cm, depending on pathogenic variant, family history of AD, and growth rate.[625]

10.1.3. Vascular Ehlers−Danlos syndrome. vEDS is a rare, autosomal dominant CTD caused by mutations in the *COL3A1* gene, leading to defective or reduced collagen III. It is characterised by fragile arteries prone to dissection or rupture, spontaneous carotid−cavernous sinus fistula formation, spontaneous perforation of hollow organs such as the colon and uterus, and spontaneous pneumothorax.[622] Common features include thin, translucent skin with easy bruising, characteristic facial appearance (thin lips and nose, small chin, prominent eyes, protruding ears with small earlobes), and congenital club foot. While not as pronounced as in other Ehlers−Danlos syndrome types, joint hypermobility may be present, particularly in the distal joints.

While childhood events are rare, about 25% of individuals with vEDS experience a major complication by age 20 years, and 80% by age 40 years.[626−628] Median life expectancy is around 50 years.

Patients with vEDS should be managed in tertiary aortic centres by multidisciplinary teams including vascular surgeons, cardiologists, cardiothoracic surgeons, geneticists, and other relevant specialists, with expertise in genetic aortopathy and genetic family screening.[629,630] The European Reference Network on Rare Multisystemic Vascular Diseases (VASCERN) comprises seven expert centres across Europe dedicated to vEDS care (http://vascern.eu/).

Management focuses on preventing complications through medication and lifestyle modifications. The randomised Beta blockers in Ehlers-Danlos Syndrome Treatment (BBEST) trial showed that celiprolol, a β1 selective blocker with β2 agonist activity, reduced arterial rupture risk threefold over 47 months (HR 0.36).[631] Its protective effect has been confirmed in subsequent cohort studies.[632,633] A recent RCT demonstrated that adding the angiotensin II receptor blocker irbesartan to celiprolol further reduced major arterial events (HR 0.42), supporting earlier cohort findings.[634,635] Periodic MRI or CTA surveillance of the entire vascular tree is integral to vEDS management,[636] and genetic screening of family members is an essential element of care.

Due to the extreme vascular fragility, surgical and interventional procedures in vEDS carry high risk. Consequently, acute arterial complications are best managed conservatively unless life threatening. No clear diameter thresholds exist for prophylactic aortic intervention, and decisions should be individualised.[293,636,637]

10.1.4. Familial thoracic aortic disease. Familial thoracic aortic disease (FTAD) refers to TAA or AD occurring in families (≥ two family members) without syndromic features or a known CTD. It is typically inherited in an autosomal dominant pattern and characterised by a family history of aneurysms or dissections, often presenting at a younger age.

While most cases lack an identifiable genetic variant, pathogenic mutations have been found in several genes, including *ACTA2*, *FBN1*, *MYH11*, *TGFBR1*, *TGFBR2*, *SMAD3*, and *MYLK*. This genetic heterogeneity underscores the importance of genetic testing and counselling in individuals with a family history of thoracic aortic disease. Identifying causative variants enables early diagnosis, refined risk stratification, and individualised management.[196]

First degree relatives of patients with FTAD should be considered for imaging screening starting at age 50 years, with periodic surveillance thereafter. Secondary prevention strategies, such as the use of ACE inhibitors, may be appropriate in early onset FTAD.[196]

Whether patients with FTAD exhibit increased arterial fragility or reduced tolerance to endografts remains unclear. As such, no uniform treatment thresholds or preferred modalities can be recommended. Management should be individualised, taking into account aortic diameter, growth rate, family history, specific genotype, and clinical urgency.

10.1.5. Surgical treatment. OSR has traditionally been preferred in patients with genetic aortopathies owing to concerns about endograft related complications in fragile tissue and unknown durability.[638] When performing open vascular surgery in patients with fragile vessels, several specialised principles should be applied.

(1) Gentle tissue handling. Use fine, atraumatic instruments and avoid excessive traction. Limit direct clamping on the vessel wall, and if needed use atraumatic soft vascular clamps and broad vessel loops. Instruct the assistant on maintaining steady, gentle tension, avoiding any sudden pulling. Ensure the needle enters the vessel at the correct angle, and rotate it smoothly during passage to minimise trauma. The parachute technique may be unsuitable in severe vascular fragility cases, as tightening can create a cheese wire effect; instead, single sutures are preferred.
(2) Anastomotic reinforcement. Reinforce suture lines with pledgets, felt, or Teflon strips to prevent dehiscence. Wrapping the anastomosis with synthetic (e.g., Dacron) or biological materials (e.g., pericardium) and applying tissue glue can further support the anastomosis.
(3) Tension free anastomosis and suture line. Ensure adequate vessel mobilisation to avoid anastomotic tension, which lowers the risk of tearing. Patching vascular defects, such as after large bore access, rather than direct suturing is advised. Creating deliberately ugly, staggered suture lines instead of nice linear suture lines reduces the risk of suture line tears.

Nevertheless, OSR for DTA and TAbdAo pathologies is highly invasive and carries substantial peri-operative risk. Consequently, there is increasing use of endovascular repair in high risk patients, redo procedures, and acute settings such as rupture or dissection. However, available data on surgical treatment methods are limited to mainly heterogeneous retrospective case series with short follow up, and direct comparative studies between OSR and EVAR are lacking.[201,639,640] Furthermore, existing data do not adequately assess the impact of recent advances in endovascular technology.

Reported outcomes of TEVAR in patients with genetic aortopathies vary widely and are influenced by device characteristics, such as uncovered proximal bare stents, degree of stent graft oversizing, and LZ location, particularly whether landing occurs in native or surgical grafts. Proximal LZ complications, including RTAAD, endoleak, SINE, aneurysmal progression, and rupture, have been associated with re-intervention rates of 14 – 60% and open conversion rates up to 33%.[639–644] Proximal and distal landing of endografts in a surgical Dacron graft is generally considered safe and should be pursued if possible.[1,27,639] In the context of type B dissection, proximal landing in Ishimaru zone 2 is associated with lower complication rates compared with zone 3.[637,645,646] Employing endografts designed specifically for dissection, with lower radial force, may reduce the risk of dSINE in fragile aortic tissue.[267,605,647]

In a recent retrospective multicentre study, the Endovascular Aortic Intervention in Patients with Connective Tissue Disease (EVICTUS) trial, 171 patients (142 MFS, 17 LDS, 12 vEDS) treated endovascularly for complex aortic aneurysms and dissection were included. Technical success was 98% and 30 day mortality rate 2.9%. Most (80%) had had previous open aortic surgery, and 43% of the procedures involved arch or visceral branches. At one and five years, survival was 96% and 81% for MFS, 94% and 85% for LDS, and 75% and 44% for vEDS, respectively. After a median follow up of 4.7 years (range 1.9 – 9.2 years), 53% required secondary interventions, including 8.2% open conversions.[293]

For large bore vascular access in patients with genetic aortopathies, percutaneous closure devices are frequently used and appear to be safe and effective, with similar complication rates as patients without aortopathies. In a retrospective, single centre study of 33 patients with confirmed genetic aortopathy (including MFS $n = 7$, LDS $n = 7$, vEDS $n = 3$, and others with *ACTA2*, *PRKG1*, *FOXE3*, or *LOX* mutations) undergoing endovascular aortic procedures with large bore (median 20 F) percutaneous femoral access, no unplanned surgical cutdowns or access site complications were reported.[648] However, caution is advised for patients with extreme vascular fragility, such as vEDS, where primary surgical cutdown should be considered.

In conclusion, available evidence suggests OSR should be considered the preferred option for elective treatment of thoracic aortic disease in patients with genetic aortopathies, while endovascular aortic repair should be considered when open surgery is contraindicated, in high risk surgical candidates, in acute settings, or for redo procedures, preferably with LZs in pre-existing surgical grafts.

Recommendation 103 — New

Genetic counselling and testing are recommended in patients with thoracic aortic disease < 60 years of age, a family history of thoracic aortic disease, concomitant arterial aneurysms or dissections, or syndromic features.

Class	Level	Reference
I	C	Consensus

Recommendation 104 — New

Patients with suspected or confirmed genetic aortopathies are recommended for multidisciplinary management at highly specialised aortic centres.

Class	Level	Reference
I	C	Consensus

Recommendation 105 — New

Patients with genetic aortopathies should be considered for serial imaging surveillance of the aorta and its major arterial branches with computed tomography or magnetic resonance angiography.

Class	Level	Reference
IIa	C	Consensus

Recommendation 106 — Changed

Marfan syndrome patients with an aortic diameter ≥ 5.0 cm should be considered for descending thoracic aortic aneurysm repair.

Class	Level	Reference
IIa	C	Consensus

Recommendation 107 — Changed

Patients with Loeys–Dietz syndrome and an aortic diameter > 4.5 – 5.0 cm should be considered for descending thoracic aortic aneurysm repair, taking into consideration diameter progression rate, age, family history, and pathogenic variants.

Class	Level	Reference
IIa	C	Consensus

Recommendation 108 — Changed

Patients with vascular Ehlers–Danlos syndrome with descending thoracic aortic pathologies should be considered for individualised surgical treatment decision making.

Class	Level	Reference
IIa	C	Consensus

Recommendation 109		Changed
For preference, open surgical repair should be considered for elective treatment of thoracic aortic disease in patients with genetic aortopathies.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 110		Changed
Endovascular aortic repair should be considered in patients with genetic aortopathies when open surgery is contraindicated, in high risk surgical candidates, in acute settings, or for redo procedures, preferably with landing zones in pre-existing surgical grafts.		
Class	**Level**	**Reference**
IIa	C	Consensus

10.2. Floating thrombus

Aortic mural thrombi are uncommon in normal or minimally diseased aortas and are typically associated with underlying aortic pathologies such as aneurysm, dissection, aortitis, or advanced atherosclerosis. In contrast, mobile or "floating" thrombi, often pedunculated and projecting into the lumen[649] (Fig. 13), can develop in normal or mildly diseased aortas and are often idiopathic, although they may be linked to hypercoagulable states such as malignancy, heparin induced thrombocytopenia, or antiphospholipid syndrome. Aortic thrombus is most often located in the aortic arch or DTA.[650] The term "shaggy aorta" is a different entity and refers to a severely diseased aortic wall, characterised by extensive atherosclerosis, ulcerations, and thrombus formation (see Chapter 3.2).

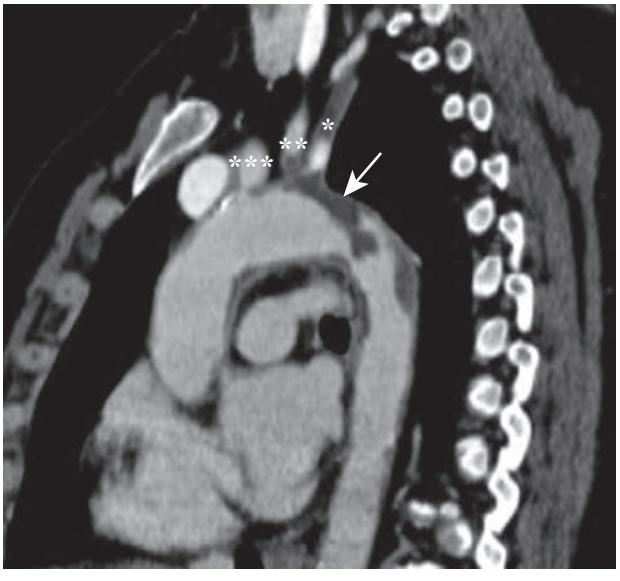


Figure 13. Computed tomography angiography (CTA) showing a "floating" thrombus in the aortic arch and proximal descending thoracic aorta. Permission to reproduce granted from Elsevier *J Vasc Surg*.[649]

Aortic mural thrombus carries some risk of peripheral embolisation causing stroke, renovisceral ischaemia, and or limb ischaemia.[650,651] Due to their mobility, floating thrombi are considered to carry a particularly high embolic risk.

Evidence guiding the management of aortic thrombus is limited. Asymptomatic patients are typically managed conservatively and do not require antithrombotic or additional antiplatelet agent therapy unless otherwise indicated for another cause.

Patients with evidence of embolisation, or those at high embolic risk due to mobile floating thrombi, should initially be considered for intensified antithrombotic medical therapy. The antithrombotic regimen should be individualised, as the choice and duration depend on factors such as embolic events, thrombus morphology, concomitant medical therapy, and bleeding risk. If medical management fails, with recurrent embolic events, endovascular exclusion may be considered.[651–655]

Recommendation 111		New
Escalation of antithrombotic therapy is not indicated for asymptomatic mural thrombus in the descending thoracic aorta.		
Class	**Level**	**Reference**
IIIa	C	Consensus

Recommendation 112		New
Antithrombotic therapy should be considered for symptomatic thrombus or asymptomatic "floating" thrombus in the descending aorta.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 113		New
Endovascular intervention may be considered for symptomatic thrombus or asymptomatic "floating" thrombus in the descending aorta when medical management is unsuccessful.		
Class	**Level**	**Reference**
IIb	C	Consensus

10.3. Inflammatory aortic disease (aortitis)

Inflammatory aortitis comprises large vessel vasculitis such as Takayasu arteritis (TA) and giant cell arteritis (GCA).[656,657] Other causes are immunoglobulin G4 related disease, systemic lupus erythematosus (SLE), rheumatoid arthritis, sarcoidosis, Behçet's disease, granulomatosis with polyangiitis, and HLA-B27 associated spondyloarthropathies.[658] In many cases, the aetiology remains idiopathic.

TA, or "pulseless disease", was first described in 1908 by the Japanese ophthalmologist Dr Takayasu.[659] The incidence ranges from 0.4 to 2.6 per million, and it is more prevalent in Asian populations, particularly in Japan, Southeast Asia, India, and Mexico.[660] TA predominantly affects young women and typically presents in the second or third decade of life. TA is believed to have an autoimmune aetiology, with immune mediated inflammation, vascular remodelling, and endothelial dysfunction driving disease progression. Triggered by unknown environmental or viral stimuli, vasculitogenic T cells infiltrate the arterial adventitia and media, leading to wall oedema (Fig. 14), extracellular matrix deposition, myofibroblast proliferation, and acute vascular inflammation. TA typically presents in two overlapping phases: (1) an early systemic acute phase with constitutional symptoms (fever, fatigue, weight loss, arthralgia); and (2) a chronic phase with signs of organ specific involvement. Hypertension is common (33 — 83%), often due to renal artery stenosis (28 — 75%). Aortic involvement occurs in 20 — 24% of cases and includes aortic dilation, arch branch stenosis, valve leaflet separation, and valve thickening.[661,662] The diagnosis is based on a combination of clinical, laboratory, and imaging findings. The European League Against Rheumatism (EULAR) and the American College of Rheumatology (ACR) has recently proposed classification criteria for TA (Table 11).[662]

GCA, also known as temporal or cranial arteritis, is the most common systemic vasculitis in the elderly, with incidence rates of 18 — 29 per 100 000 individuals over 50 years, being highest in Scandinavia and lowest in East Asia.[663] GCA is characterised by granulomatous inflammation of large and medium sized vessels, typically involving cranial arteries.[664] Its pathogenesis involves both innate and adaptive immunity, with endothelial and smooth muscle cells contributing to vessel wall inflammation. Key cytokines include interferon gamma (IFN-γ) and interleukin 2 (IL-2), produced by activated T cells.[663,665] Histology shows granulomatous infiltrates within the vessel wall. GCA typically presents with headache, scalp tenderness, jaw claudication, visual disturbances, and varying degree of constitutional symptoms.[666] In addition to cranial arteries, involvement of the aorta and major branches is increasingly recognised with modern imaging.[667,668] GCA is associated with an increased risk of vision loss, stroke, and TAA or dissection. Vascular complications of medium sized vessels are most common within the first year, while aortic events occur in 20 — 30% of patients, often more than five years after diagnosis, and are associated with a fivefold increase in mortality rate.[669] The EULAR/ACR classification criteria for GCA are shown in Table 12.[663]

Other chronic inflammatory diseases associated with thoracic aortitis include Behçet disease,[670,671] rheumatoid arthritis, sarcoidosis, Cogan syndrome, Kawasaki disease, ankylosing spondylitis, SLE, and granulomatosis with polyangiitis. Cardiovascular manifestations include aortic regurgitation, aneurysm, dissection, and stenosis, typically affecting the aortic root. DTA involvement is rare but may progress rapidly and rupture. In SLE, persistent inflammation despite steroid therapy may increase the risk of AD.[672]

The 2022 EULAR/ACR classification criteria highlight the role of CTA, MRA, and FDG-PET in diagnosing large vessel vasculitis.[662] CTA is the preferred modality for detecting aortic wall inflammation, peri-aortic thickening, and adjacent organ involvement. FDG-PET demonstrates high diagnostic accuracy, with pooled sensitivity and specificity, respectively, of 90 — 92% and 85 — 98% for GCA and 81 — 87% and 74 — 84% for TA.[663,673] Imaging features for differentiating common arteritides are summarised in Table 13.

Table 11. The European League Against Rheumatism (EULAR) and American College of Rheumatology (ACR) classification criteria for Takayasu arteritis.[662]

Absolute requirements	
Age ≤ 60 years at time of diagnosis	
Evidence of vasculitis on imaging	
Additional clinical criteria	
Female sex	+1
Angina or ischaemic cardiac pain	+2
Arm or leg claudication	+2
Vascular bruit	+2
Reduced pulse in upper extremity	+2
Carotid artery abnormality	+2
Systolic blood pressure difference in arms ≥ 20 mmHg	+1
Additional imaging criteria	
Number of affected arterial territories (select one)	
One arterial territory	+1
Two arterial territories	+2
Three or more territories	+3
Symmetric involvement of paired arteries	+1
Abdominal aortic involvement with renal or mesenteric involvement	+3
A total score of ≥ 5 points is required to classify Takayasu arteritis	

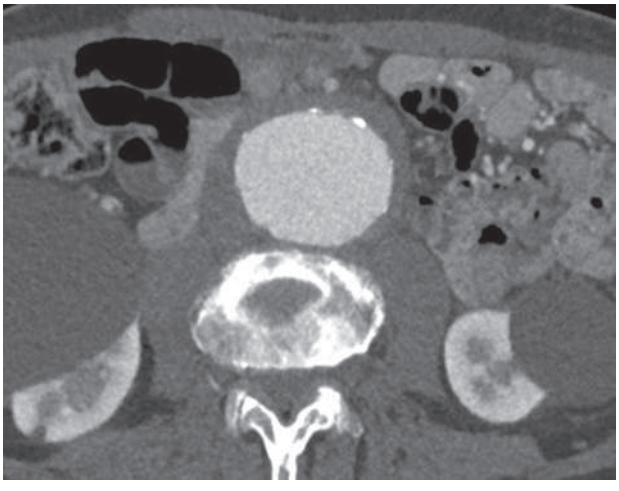


Figure 14. Axial computed tomography angiography (CTA) image demonstrating the characteristic circumferential peri-aortic inflammatory rind surrounding the aneurysm, typically appearing as a high attenuation, soft tissue mantle (artist and copyright Carlota F. Prendes, Uppsala, Sweden).

Table 12. The European League Against Rheumatism (EULAR) and American College of Rheumatology (ACR) classification criteria for giant cell arteritis.[663]

Absolute requirements	
Age ≥ 50 years at the time of diagnosis	
Additional clinical criteria	
Morning stiffness in shoulder and or neck	+2
Sudden visual loss	+3
Jaw or tongue claudication	+2
New temporal headache	+2
Scalp tenderness	+2
Abnormal examination of the temporal artery	+2
Laboratory findings	
Maximum ESR ≥ 50 mm/hour or maximum CRP ≥10 mg/L	+3
Biopsy and imaging findings	
Positive temporal artery biopsy or halo sign on temporal artery ultrasound	+5
Bilateral axillary involvement	+2
FDG-PET activity throughout aorta	+2
A total score of ≥ 6 points is required to classify giant cell arteritis	

ESR = erythrocyte sedimentation rate; CRP = C reactive protein; FDG-PET = fluorodeoxyglucose positron emission tomography.

The optimal management of inflammatory aortitis remains uncertain. It is recommended that all patients are closely monitored by a multidisciplinary team, typically involving vascular surgery, cardiothoracic surgery, vascular medicine, rheumatology, cardiology, and radiology.[674]

High dose glucocorticoids (e.g., prednisone 40 − 60 mg/day) remain the standard induction therapy for TA and GCA, achieving disease control and remission in most cases.[675,676] In TA, glucocorticoids are typically combined with a non-biological (synthetic) disease modifying antirheumatic drug (DMARD) such as methotrexate, sulfasalazine, hydroxychloroquine, and leflunomide, given the high relapse rates of up to 70% associated with glucocorticoid monotherapy. Biological DMARDs (e.g., tocilizumab or tumour necrosis factor [TNF] inhibitors) are considered second line options for patients who relapse despite initial combination therapy.[675,677,678] In GCA, combining a prednisone taper with tocilizumab, an interleukin 6 (IL-6) receptor inhibitor, has demonstrated superior rates of glucocorticoid free remission at one year, leading to its approval as adjunctive therapy in selected patients, with methotrexate remaining a recommended alternative.[679,680] Patients with TA or GCA with involvement of the aorta and its branches often experience insidious onset and slow progression, leading to irreversible vascular lesions despite medication therapy.

Indications for surgical treatment include uncontrolled hypertension due to renal artery stenosis, symptomatic coronary or cerebrovascular disease, severe aortic regurgitation, critical limb threatening ischaemia from stenotic or occlusive lesions, and large aneurysms at risk of rupture.[681]

As active inflammation is linked to increased peri-operative complication rates, adequate pre-operative immunosuppression is essential to reduce procedural failure.[682] To minimise peri-operative risk, surgical intervention is best timed during remission, with ^{18}F-FDG-PET/CT recommended to assess disease activity before treatment.[683,684]

Available literature on surgical treatment of inflammatory aortitis is restricted to small case series and isolated reports across heterogeneous pathologies and vascular territories, limiting the possibility to provide clear advice.

Table 13. Imaging features for Takayasu arteritis and giant cell arteritis, adopted from the European League Against Rheumatism (EULAR) and American College of Rheumatology (ACR).[663]

Imaging modality	Takayasu arteritis	Giant cell arteritis
US	"Macaroni sign" Sensitivity 81% and specificity >90%	"Halo sign" "Compression sign" Sensitivity 77% and specificity 96%
CTA	Mural thickening and enhancement, late contrast uptake Vascular stenosis, occlusion, or ectasia Surrounding oedema or tissue reaction Sensitivity 100% and specificity 100%	Same as Takayasu arteritis Sensitivity 84.6% and specificity 84.6%
MRA	Mural thickening and enhancement Vascular stenosis, occlusion, or ectasia Surrounding oedema or tissue reaction Carotid artery involvement (internal carotid artery more common) Sensitivity 100% and specificity 100%	Same as Takayasu arteritis (external carotid artery more common) Sensitivity 73% and specificity 88%
FDG-PET	Mural thickening and tracer uptake Vascular stenosis, occlusion, or ectasia Surrounding oedema or tissue reaction Cluster analysis of involved arteries (left subclavian artery together with bilateral involvement of carotid and mesenteric arteries) Sensitivity 81−87% and specificity 74−84%	Same as Takayasu arteritis (symmetric subclavian artery with concomitant axillary artery vasculitis) Sensitivity 90−92% and specificity 85−98%

US = ultrasound; CTA = computed tomography angiography; MRA = magnetic resonance angiography; FDG-PET = fluorodeoxyglucose positron emission tomography.

OSR offers definitive excision of diseased tissue with favourable midterm outcomes when performed during remission, but carries high peri-operative morbidity and mortality rates.[685,686] While endovascular aortic repair is a less invasive alternative with a superior early outcome in selected patients with well controlled inflammation, it is associated with higher rates of persistent peri-aneurysmal fibrosis and late complications in abdominal inflammatory aneurysms.[674] Thus, given the heterogeneous clinical picture and lack of evidence, the choice of surgical method should be made on a case by case basis, taking into account the urgency, anatomy, fitness, and current inflammatory disease activity. Nonetheless, due to the surgical complexity of thoraco-abdominal involvement, an endovascular first strategy may be reasonable in appropriately selected patients.

Long term follow up after surgical intervention is critical, as up to 82% of patients develop disease related vascular complications within ten years, with nearly 50% requiring re-intervention.[687]

Recommendation 114	New	
Multidisciplinary team management is recommended for active inflammatory disease of the aorta and or its branches.		
Class	**Level**	**Reference**
I	C	Consensus

Recommendation 115	Changed	
High dose glucocorticoids combined with a disease modifying antirheumatic drug are recommended as first line treatment for active inflammation of the aorta and or its branches.		
Class	**Level**	**Reference**
I	C	Consensus

Recommendation 116	Unchanged	
Surgical treatment should be considered in selected patients with inflammatory arteritis of the thoraco-abdominal aorta and its branches, taking into consideration aneurysm size, evidence of end organ ischaemia, and inflammatory disease activity.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 117	New	
Elective endovascular repair should be considered as the first line surgical approach for patients with inflammatory arteritis of the thoraco-abdominal aorta.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 118	New	
Long term post-operative surveillance is recommended for patients treated for inflammatory aortitis, given the high risk of delayed complications and disease progression.		
Class	**Level**	**Reference ToE**
I	C	Espitia *et al.* (2023)[687]

10.4. Mycotic aortic aneurysm

Mycotic aortic aneurysm, also known as infective native aortic aneurysm or infectious aortitis, refers to infection induced degeneration of the aortic wall, leading to aneurysm formation.[8,98,688,689] The incidence is estimated at up to 2% in the Western population.[690,691] Infection may spread haematogenously, from adjacent structures, or via septic emboli, as seen in endocarditis or advanced human immunodeficiency virus (HIV) infection.[8,690] Gram positive cocci, particularly *Staphylococcus aureus* (29% of cases), are the most common pathogens in Western countries, while *Salmonella* predominates in Asia (60 — 70%). Other pathogens include *Pneumococcus*, *Escherichia coli*, *Candida*, *Aspergillus*, *Mycobacterium tuberculosis*, and *Treponema pallidum*, particularly in immunocompromised patients. Cultures remain negative in 20 — 30% of cases, limiting early pathogen identification.[689]

Clinical presentation typically includes fever $\geq 38^{\circ}$C, localised pain, fatigue, sepsis, or shock, often in the context of concurrent infection or immunosuppression. Rupture is present in up to 25% at diagnosis.[688] Elevated inflammatory markers (C reactive protein [CRP], leukocytes) and positive blood or tissue cultures support the diagnosis. Blood sampling should follow endocarditis protocols, with at least three sets (aerobic and anaerobic) from separate sites, repeated every 24 — 48 hours until infection is controlled. If open surgery is performed, aortic tissue samples should be collected for microbiological culture.

CTA is the imaging modality of choice for diagnosing mycotic aortic aneurysms owing to its high availability, even in urgent settings.[688] Key findings include saccular, multilobular, or eccentric aneurysms, peri-aortic gas, fluid, soft tissue mass, and rapid expansion (Fig. 15).[692] FDG-PET and white blood cell scintigraphy can aid in identifying peri-aortic inflammation,[693] while TOE is helpful for assessing aortic root and valve involvement.[98]

Diagnosis of a mycotic aortic aneurysm is based on a combination of (1) clinical presentation, (2) laboratory tests and microbiology, and (3) radiological findings (Table 14). In addition, the presence of peri-aortic infection during surgery is diagnostic.[8] A recent Delphi consensus statement proposed a diagnostic algorithm for mycotic aortic aneurysm, based on a combination of the criteria, with a definite diagnosis when all three criteria are met and no differential diagnosis being more probable, or when intra-operative finding of pus or abscess in the aneurysm wall, or positive microbiological culture or histology from guided aspiration from aneurysms with clinical suspicion of

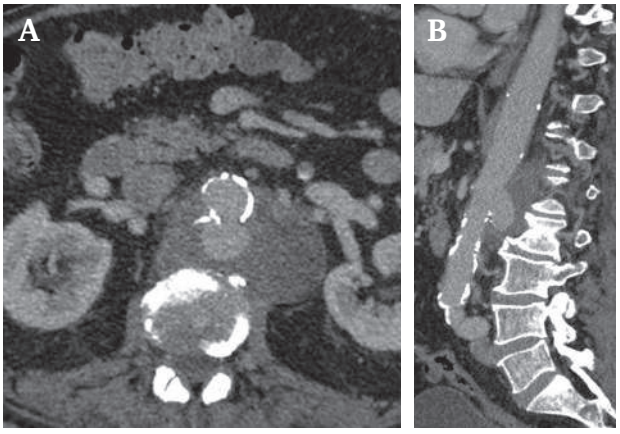


Figure 15. Paravisceral mycotic aneurysm. (A) Axial computed tomography angiography (CTA) image demonstrating asymmetrical posterior extension of the aneurysm, with inflammatory involvement of the back aortic wall. (B) Sagittal reconstruction showing pronounced posterior protrusion of the paravisceral aneurysm, consistent with mycotic degeneration and localised wall destruction (artist and copyright Carlota F. Prendes, Uppsala, Sweden).

Table 14. Suggested diagnosis criteria for mycotic aneurysm.

Clinical presentation	Abdominal and or back pain, fever ≥38°C, sepsis or shock Presence of concurrent infection or immunosuppression
Laboratory tests	Elevated inflammatory markers such as C reactive protein, procalcitonin, or total white blood cell count consistent with ongoing infection Microbiology: blood or peri-operative aneurysm wall or peri-aortic tissue cultures with growth of common causative pathogens. 16S ribosomal RNA polymerase chain reaction on tissue sample from aneurysm wall to show the presence of bacterial genome
Radiological findings	Computed tomography angiography or magnetic resonance imaging: presence of one or multiple aneurysms with morphological features (saccular, eccentric, or multilobular) associated with mycotic abdominal aortic aneurysm, signs of peri-aortic infection (peri-aortic mass or peri-aortic gas), and or signs of rapid expansion Molecular imaging: 18-fluorodeoxyglucose positron emission tomography or white blood cell scintigraphy with evidence of increased peri-aortic inflammatory activity or increased uptake within the aneurysm wall
Surgical findings	Presence of peri-aortic infection

mycotic aortic aneurysm; a probable diagnosis when 2/3 criteria are met and no differential diagnosis being more probable; and not a probable diagnosis when <2/3 criteria met.[688]

Early diagnosis, prompt administration of systemic antibiotics, and timely surgical intervention is key in the management of mycotic aortic aneurysms. Empirical antibiotic therapy targeting *S. aureus* and Gram negative rods (e.g., *Salmonella* spp.) should be initiated immediately after cultures have been secured. Antibiotic therapy should be tailored based on microbiological findings as soon as possible; if cultures remain negative, empirical treatment should be continued. The duration of antibiotic therapy (ranging from four to six weeks to lifelong), depends on pathogen type, surgical approach, and patient immune status.[689] Antibiotics alone are associated with poor outcomes and should be reserved for palliative care in patients unfit for intervention.[98]

The optimal timing of the surgery remains debated. However, due to the unpredictable and aggressive natural course of mycotic aortic aneurysms, with rapid expansion and high rupture rates of up to 44% at presentation, urgent repair should be considered regardless of aneurysm size.[690,694] Although some reports suggest favourable outcomes with delayed intervention following initial systemic antibiotic therapy, these findings may be influenced by selection bias, and delaying surgery without strict surveillance carries significant risk due to the high potential for growth and rupture.[695]

The supporting evidence on surgical treatment of mycotic aortic aneurysms is limited to retrospective case series and small cohorts with short follow up and is subject to selection bias, with OSR often reserved for fitter patients and EVAR used in high risk or anatomically challenging cases.[696] No direct head to head comparative studies between OSR and endovascular repair exists. Despite this, OSR with *in situ* reconstruction using synthetic grafts or allografts, combined with extensive debridement and prolonged antimicrobial therapy, has long been regarded as the gold standard for definitive and durable treatment.[697–699] However, OSR of mycotic TAA or TAAA is highly invasive, requiring thoracotomy and aortic cross clamping, and is associated with significant peri-operative morbidity and mortality, particularly in patients with chronic kidney disease or ASA score ≥ 3.[98,699] Due to the high risk of re-infection, long term antimicrobial therapy is essential.[8,697] Reported five year survival following OSR is approximately 60%.[698]

Over the past two decades, endovascular repair has been used increasingly to treat mycotic aortic aneurysms, offering high technical success and acceptable early outcomes, particularly in frail or unstable patients.[689] While concerns persist about leaving infected tissue in place and the risk of re-infection, endovascular treatment avoids the trauma of open surgery and may serve as a bridge or as definitive therapy in those unfit for OSR.[695,700] Cohort and registry studies suggest a survival advantage in the early period after endovascular treatment, with similar five year survival compared with OSR.[691,697] A Swedish nationwide study of 52 patients with mycotic TAA (67% treated with TEVAR, 15% with FBEVAR, and 14% with hybrid repair, 25% were ruptured) reported 30 day survival of 92% and five

year survival of 71%, with infection related complications in 17%, most occurring within the first year and often fatal.[690] A Swedish nationwide propensity matched analysis of 132 patients with mycotic AAA showed a significant early survival benefit for EVAR, with no late infection or aneurysm related outcome disadvantages.[701] Similarly, a meta-analysis of 963 patients reported a lower early mortality rate with EVAR (*vs.* OSR) for paravisceral and infrarenal mycotic AAAs, although no long term difference was observed.[690] However, Japanese registry data and recent meta-analyses indicate a higher recurrent infection rate after EVAR (OR 2.8; RR 2.4), although overall mortality and re-intervention rates were similar to OSR.[696,702]

In summary, due to the variability in clinical presentation, anatomic complexity, and causative pathogens, combined with limited supporting evidence, an individualised approach is recommended. Endovascular repair is a valid alternative to OSR for mycotic TAA and TAAA, and is often preferable, as patients are frequently unfit for major open surgery.[98,703] Regardless, long term clinical and radiological surveillance on an individual basis is advocated.[704,705] Finally, given the rarity and complexity of mycotic TAA and TAAA, its management should be centralised to high volume centres with available multidisciplinary expertise (see Chapter 2).

Recommendation 119 **New**

Multidisciplinary management at highly specialised aortic centres is recommended for patients with mycotic thoracic and thoraco-abdominal aortic aneurysms.

Class	Level	Reference
I	C	Consensus

Recommendation 120 **New**

Urgent surgical repair of mycotic thoracic and thoraco-abdominal aortic aneurysms should be considered irrespective of aneurysm size.

Class	Level	Reference
IIa	C	Consensus

Recommendation 121 **New**

An individualised treatment strategy (open surgical repair, endovascular repair, conservative, or palliative management) for mycotic thoracic and thoraco-abdominal aortic aneurysms should be considered, based on urgency, anatomic considerations, surgical risk, causative pathogens, and patient preferences.

Class	Level	Reference
IIa	C	Consensus

Recommendation 122 **New**

Individualised antibiotic therapy for mycotic thoracic and thoraco-abdominal aortic aneurysms should be considered, with treatment duration ranging from four to six weeks to lifelong, depending on microbiology, surgical approach, and immune status.

Class	Level	Reference
IIa	C	Consensus

10.5. Coarctation of the aorta

Coarctation of the aorta (CoA) is defined as a narrowing of the thoracic aorta at the ductus arteriosus insertion, just distal to the LSA (juxtaductal) (Fig. 16), but may also occur proximal (pre-ductal) or distal (post-ductal) to this site, and can involve other aortic segments including the arch and abdominal aorta. CoA is among the most prevalent congenital cardiovascular anomalies, with an incidence of 3 — 4 per 10 000 live births and a male predominance (2:1). It is frequently associated with other congenital defects, including bicuspid aortic valve (60%), aortic arch anomalies (18%), subaortic stenosis (6%), intracranial aneurysms, and other cardiovascular malformations.[706,707]

The clinical presentation varies with the severity of aortic narrowing, involvement of arch vessels, and collateral development. Most adults experience mild symptoms such as lower limb claudication; less commonly, headache, epistaxis, exertional dyspnoea, dizziness, abdominal angina, or heart failure may occur. Typical findings include upper extremity hypertension, lower limb hypotension, and delayed or diminished femoral pulses. In some cases, AD or aneurysm formation may be the initial manifestation.

Diagnosis is confirmed with imaging, typically CTA or MRI. Both modalities effectively assess stenosis severity, collateralisation, aneurysms, and associated anomalies.[708] MRI avoids radiation and allows flow quantification, while CTA offers superior spatial resolution, faster acquisition, and fewer artefacts, particularly in the presence of metallic implants.[709] Although TTE is effective for diagnosing CoA in infants, its utility in adults is limited by body habitus and anatomy. It can estimate CoA severity using velocity ratios and is useful for assessing left ventricular function and identifying associated anomalies such as bicuspid aortic and mitral valve defects. Catheter angiography remains the gold standard for assessing peak pressure gradient in CoA but is primarily reserved for patients undergoing catheter based interventions.[710]

The prognosis of untreated CoA is poor, with historical data showing an up to 75% mortality rate in middle aged adults due to heart failure, aortic syndromes, coronary disease, endocarditis, or intracranial bleeding. Even after successful CoA treatment, persistent hypertension affects over half of adults, contributing to reduced survival and QoL.[711]

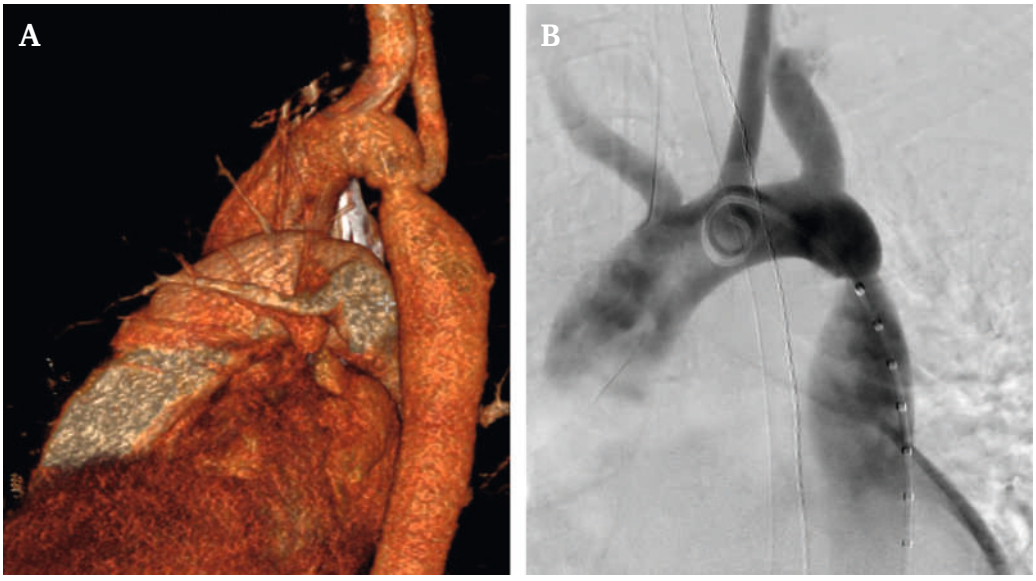


Figure 16. Coarctation of the aorta at the isthmic level. (A) 3D computed tomography angiography (CTA) reconstruction showing a discrete narrowing of the aortic isthmus just distal to the origin of the left subclavian artery, with associated post-stenotic dilatation. (B) Intra-operative angiography confirming the focal stenosis at the same anatomical level, demonstrating the characteristic contour and calibre change of aortic coarctation (artist and copyright Carlota F. Prendes, Uppsala, Sweden).

Treatment of CoA is indicated in patients with upper limb hypertension at rest or with exercise, or in the presence of left ventricular hypertrophy and either a non-invasive SBP gradient >20 mmHg between the upper and lower limbs or an invasive gradient across the coarctation with impaired left ventricular function or significant collateral flow.[712]

OSR has long been the standard treatment for adult CoA, with a low mortality rate ($<1\%$) but a risk of SCI.[713] Techniques such as cardiopulmonary bypass or LHB are used to reduce the SCI risk, and deep hypothermic circulatory arrest may be required for arch involvement. Surgical options include end to end anastomosis, prosthetic patch aortoplasty, subclavian flap aortoplasty, extra-anatomic bypass, and interposition graft. Due to high rates of patch aneurysms ($20-40\%$) and risk of left arm claudication after subclavian flap aortoplasty, interposition grafts and extra-anatomic bypasses are the preferred surgical approaches in adults.[713]

The first transcatheter balloon angioplasty for CoA was reported in 1982,[714] but high rates of re-coarctation (up to 80%) and aneurysm formation limited its use. Stenting prevents vascular recoil, fixes intimal flaps to the aortic wall, and reduces the risk of dissection and aneurysm formation. Adult CoA stenting has demonstrated high technical success and a low 30 day mortality rate (1%).[715,716] In the Coarctation of the Aorta Stent Trial (COAST), 104 patients treated with Cheatham Platinum balloon expandable bare metal stents showed excellent outcomes, with 13% requiring redilation and no surgical conversions.[717] An RCT of 92 patients found no differences between Cheatham Platinum balloon expandable stents and uncovered nitinol self expanding stents at one and three year follow up.[718] Meta-analyses report an 8% re-intervention rate (mostly endovascular) and 2% mortality rate at 2.5 years.[716] While early experience primarily involved bare metal stents, covered stents have gained favour due to a lower risk of AD and rupture.[719] The COAST II trial reported a 92% success rate in high risk patients treated with covered stents, with no aortic wall injuries or deaths, although access related complications were more frequent due to larger sheath sizes.[720] An RCT of 120 patients found no significant difference in re-coarctation rates between bare and covered stents, although pseudoaneurysms were more common with covered stents. Overall, current evidence does not support the superiority of one stent design over another; selection should be guided by individual patient anatomy and clinical factors. Covered stents are especially preferred in smaller CoA diameter, native CoA, and in the presence of pseudoaneurysms.[721] While in cases involving arch vessels or concomitant cardiovascular anomalies, such as aberrant subclavian artery, bare metal stents may be preferred to preserve flow to adjacent branches.

In a single centre study of 110 adults and adolescents with CoA, open, endovascular, and hybrid approaches all achieved 100% technical success, with similar unplanned re-intervention rates (12%).[722] A multicentre registry of 350 patients (including children >10 kg) found that surgery and stenting achieved better early gradient reduction than balloon angioplasty. Stenting had fewer complications but a higher re-intervention rate than surgery.[723] Meta-analytic data confirm high technical success, 1% 30 day mortality, and 8% re-intervention at 29 months for endovascular repair, supporting its safety and durability.[716] The choice of

treatment should consider aortic anatomy, patient age, lesion length, previous repairs, and centre expertise. Adults with native, focal CoA are particularly well suited to stenting.[724] Endografting has been shown to be feasible for pseudoaneurysms after open repair of coarctation. Endografting is also a feasible treatment option for pseudoaneurysms following open CoA repair.[725] A post-stent pressure gradient of <20 mmHg, and preferably <10 mmHg, is considered desirable for haemodynamic efficacy.[723,726]

Recommendation 123		Changed
Surgical or endovascular treatment of aortic coarctation is recommended in adults with upper limb hypertension or left ventricular hypertrophy when a systolic pressure gradient* ≥ 20 mmHg is present.		
Class	**Level**	**Reference**
I	C	Consensus

* Cuff blood pressure difference between upper and lower limbs, or catheter measured across the coarctation.

Recommendation 124		Changed
Endovascular treatment should be considered as first line therapy for adult patients with aortic coarctation, with stent choice (balloon expandable *vs.* self expanding, bare metal *vs.* covered stents) based on individual anatomic and clinical factors.		
Class	**Level**	**Reference**
IIa	C	Consensus

10.6. Aberrant subclavian artery (arteria lusoria) and Kommerell's diverticulum

An aberrant subclavian artery, also known as arteria lusoria, is a congenital vascular anomaly in which usually the right subclavian artery abnormally arises distal to the LSA, rather than from the brachiocephalic artery (Fig. 17). It characteristically courses behind the oesophagus and trachea (80%), and less frequently between the oesophagus and trachea (15%) or in front of the trachea (5%).[727] Right aberrant subclavian artery represents the most common variant of aortic arch anatomy, occurring in approximately 0.1 – 3.3% of the population,[728] with a reported female : male predominance ranging from 2:1 to 3:1. Male patients, however, appear to have a higher incidence of aberrant subclavian artery related complications and undergo surgical intervention more frequently.[727,729–732] In the largest multicentre study to date, the mean age at the time of surgical intervention was 57 years.[732]

Enlargement at the origin of the aberrant subclavian artery, referred to as Kommerell's diverticulum (KD), is a common finding. In a study of 312 patients with aberrant subclavian artery, KD was present in 84% of individuals with a left sided aberrant subclavian artery and in 56% of those with a right sided aberrant subclavian artery.[729] Other studies have reported an even higher prevalence, with KD

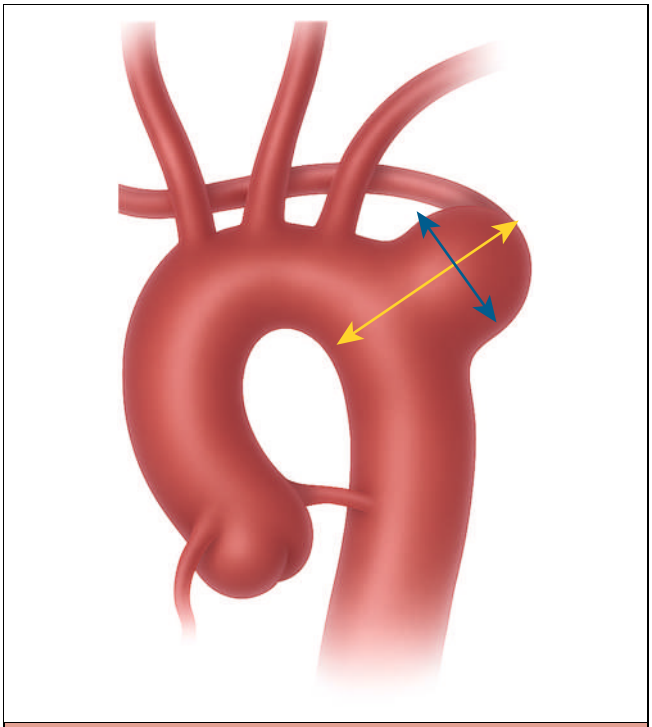


Figure 17. Suggested measurement of the aberrant subclavian artery or Kommerell's diverticulum in two planes: (1) the combined distance between the edges of the diverticulum and the adjacent aorta (yellow arrow); and (2) the transverse diameter of the aberrant right subclavian artery measured 1 cm distal to the ostium of the vessel (blue arrow). Adopted from Erben *et al.*[731]

identified in up to 100% of patients with left sided aberrant subclavian artery combined with right sided aortic arch.[733]

Aberrant subclavian arteries may be associated with other aortic arch anomalies, congenital cardiac malformations, and genetic syndromes.[728,734] A left aberrant subclavian artery typically occurs in the setting of a right sided aortic arch, and both right and left aberrant subclavian arteries may be associated with a complete vascular ring.[727,728,735] An increased prevalence of aberrant subclavian arteries has been reported in individuals with trisomy 21 and Turner syndrome.[734,735] Furthermore, patients with aberrant subclavian artery appear to have a higher risk of developing TAA and AD.[736] In an analysis of 21 336 CT scans performed for any indication on patients aged 50 – 85 years, 603 (2.8%) had arch anomalies. The prevalence of thoracic aneurysm was associated with aberrant left subclavian artery combined with right sided arch (33%), followed by bovine arch (13%), and aberrant right subclavian artery (8.2%).[737]

Although aberrant subclavian artery and KD are frequently asymptomatic, reported in 60 – 80% of cases, they may cause clinical symptoms such as dysphagia or dyspnoea due to extrinsic compression of adjacent structures, particularly the oesophagus and trachea.[729] In addition, thromboembolic events originating from the KD may result in upper limb ischaemia or cerebrovascular events, including stroke. When clinically suspected, the diagnosis is confirmed with CTA or MRI.[727] Measurement

of aberrant subclavian artery and KD has been suggested to be performed in two planes: (1) the combined distance between the diverticulum and the adjacent aorta; and (2) the transverse diameter of the aberrant right subclavian artery measured 1 cm distal to the vessel ostium (Fig. 17).[731,738–740]

Screening for associated cardiac malformations should be considered in patients with an aberrant subclavian artery and or KD, as concomitant heart defects may influence both treatment strategy and long term follow up.[734] In pre-natal diagnosis of aberrant subclavian artery and or KD cases, a comprehensive evaluation for additional structural anomalies and soft markers,[741] and in pre-natal or early post-natal settings genetic screening should be considered, particularly in the presence of associated structural abnormalities or characteristic phenotypes.[734]

The natural history of aberrant subclavian artery remains poorly understood due to the rarity of the condition; however, complications such as dissection or rupture have been reported, although rarely.[731,732] In the largest series to date, the Vascular Low Frequency Disease Consortium (VLFDC) analysed 285 patients undergoing treatment for aberrant subclavian artery and or KD, the most common indication for treatment was symptoms (59%), followed by size of the aberrant subclavian artery and or KD (38%). Rupture as the indication for treatment was documented in only 4.2% of the cohort and occurred at a mean diameter of 4.4 cm (range 1.8 – 10.0 cm).[732] Patients with incidentally detected, asymptomatic, non-aneurysmal aberrant subclavian artery may be considered for serial surveillance imaging with MRI or CTA, suggestively every five years. For aberrant subclavian artery dilation or KD, more frequent imaging is warranted. Symptomatic aberrant subclavian artery and or KD is exceedingly rare among children and young adults; in the VLFDC cohort, the mean age at treatment was 57 years (standard deviation 17 years).[732] Considering this, initiating surveillance at age of 40 years is reasonable.

Surgical treatment should be considered in all patients with an aberrant subclavian artery and or KD who present with symptoms attributable to compression of adjacent structures, such as the oesophagus or trachea. Prophylactic surgical treatment may also be justified for large, asymptomatic aneurysmal aberrant subclavian artery and or KD to prevent rupture or dissection. However, the optimal size threshold for prophylactic intervention remains uncertain due to limited supporting evidence. Earlier EACTS/ESVS consensus statements recommended surgery at an aberrant subclavian artery diameter $\geq$3.0 cm or when the combined distance between the edges of the diverticulum and adjacent aorta reached $\geq$5.5 cm, respectively,[201] while more recent guidelines from EACTS/STS (2024), American Heart Association/American College of Cardiology (ACC/AHA) (2022), and SVS (2020) advocate treatment at a slightly lower threshold of $\geq$3.0/$\geq$5.0 cm.[6,31,98] Nonetheless, based on the most recent VLFDC data, the GWC does not consider it justified to lower the threshold for prophylactic surgical treatment of aberrant subclavian artery and or KD.

Comparative analysis of surgical techniques is limited by the paucity of high quality data, publication bias, and heterogeneity in reporting standards. Data from the VLFDC suggest that favourable outcomes can be achieved regardless of the surgical technique employed. In this cohort, hybrid repair was the most commonly performed approach (59%), followed by OSR (36%).[732] Similar observations have been reported in previous systematic reviews and meta-analyses of cohort studies, which consistently found no clear superiority of one surgical technique over another. Therefore, current evidence does not support strict recommendations favouring any particular treatment approach.[27,742,743] The choice of surgical approach and technique should be individualised based on patient condition, indication for treatment (including symptom type, presence of AD, aneurysm), anatomic characteristics, and patient preference. Given the procedural complexity, patients with aberrant subclavian artery and or KD should be referred to high volume aortic centres with expertise in managing complex aortic pathology. Careful pre-operative planning is essential, as aortic arch anomalies, such as aortic origin of vertebral arteries, a short or separate innominate artery, or a bovine arch, may impact the treatment strategy.[729,736]

Hybrid repair is the most commonly reported and often preferred treatment approach.[732,742,744] The most frequent hybrid strategy involves TEVAR combined with carotid–subclavian bypass and coil embolisation of the aberrant subclavian artery. More extensive cervical debranching may be necessary to obtain a suitable LZ more proximally. Alternatively, custom made endografts may be used to land in the mid arch, typically incorporating a scallop for the left common carotid artery and a fenestration for the LSA, or in the ascending aorta, with inner branches to all supra-aortic vessels.[745]

OSR can be performed using various approaches and typically involves proximal ligation of the aberrant subclavian artery to relieve symptoms, most often via a thoracotomy, followed by subclavian artery revascularisation by either bypass or transposition. In patients with a concomitant KD, TAA, or dissection, arch and or DTA replacement may be required.[727,732] In cases with associated vascular rings, particularly in younger patients with a left sided aberrant subclavian artery, decompression should include division and resection of related structures such as the ligamentum arteriosum. For extensive arch or DTA pathology, concomitant aortic repair with a frozen elephant trunk may be considered to facilitate distal TEVAR.[4] Total endovascular repair of aberrant subclavian artery and or KD has been described in small case series and case reports, using custom made branched or fenestrated thoracic stent grafts, PMEGs, and PG techniques.[732,743,746,747] While early outcomes appear promising, demonstrating satisfactory aneurysm exclusion and symptom relief, these results must be interpreted with caution due to limited follow up, small sample sizes, and a high risk of publication bias.

Recommendation 125	New	
Patients with an aberrant subclavian artery and or Kommerell's diverticulum should be considered for evaluation of associated structural abnormalities.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 126	New	
An incidentally detected, non-aneurysmal, asymptomatic aberrant subclavian artery may be considered for serial imaging surveillance at five year intervals, beginning at age 40 years.		
Class	**Level**	**Reference**
IIb	C	Consensus

Recommendation 127	New	
Surgical treatment should be considered in patients with an aberrant subclavian artery and or Kommerell's diverticulum who present with symptoms attributable to compression of adjacent structures, such as the oesophagus or trachea.		
Class	**Level**	**Reference**
IIa	C	Consensus

Recommendation 128	New	
Surgical treatment for asymptomatic aberrant subclavian artery and or Kommerell's diverticulum may be considered when the transverse vessel diameter* is ≥ 3.0 cm or when the combined distance between the edges of the diverticulum and the adjacent aorta* is ≥ 5.5 cm.		
Class	**Level**	**Reference**
IIb	C	Consensus

* See Figure 17.

Recommendation 129	New	
The choice of surgical repair technique (open, hybrid, or endovascular repair) of an aberrant subclavian artery and or Kommerell's diverticulum should be considered individualised based on the treatment indication, anatomy, surgical risk, underlying pathology, and patient preferences.		
Class	**Level**	**Reference**
IIa	C	Consensus

11. THE PATIENTS' PERSPECTIVE

Literature focusing on the patients' perspective regarding diseases of the aorta in the chest is scarce. Informed consent for treatment addresses the most relevant potential risks and questions that may arise in patients who receive a particular choice of therapy. Yet the advantages and disadvantages of the various treatment options, including conservative treatment, should be made readily available for patients in addition to the formal consent process. Ideally, this information should come from healthcare professionals, based on expert agreement, rather than from other less reliable sources.

Furthermore, the assessment of quality of life after both treatment modalities is under represented in the literature and demands further research, enabling a more appropriate pre-operative evaluation of the patient's suitability for a specific kind of treatment.

The aim behind patient participation in healthcare decision making can be classified as democratisation and improving the quality of decisions. Patient engagement, as recommended, may also improve the validity of clinical guidelines.[748]

This document is designed to support both healthcare professionals and patients dealing with aortic dissections and aneurysms, conditions where the body's main blood vessel, the aorta, becomes enlarged in the chest (thoracic aortic aneurysm) and possibly also the upper abdomen (thoraco-abdominal aortic aneurysm). The European Society for Vascular Surgery (ESVS) has developed detailed guidelines for doctors and specialists to help ensure the best possible care. This is the main part of this document. The following part provides the same information in a simpler format, making it easier to understand for patients and others without medical training.

11.1. Information for patients

Some of the aspects that are presented in this chapter have been very adequately addressed in a similar chapter in the current ESVS abdominal aortic aneurysm guideline published in 2024 by Wanhainen *et al.*[8]

What is a thoracic or thoraco-abdominal aortic aneurysm? A descending thoracic aortic aneurysm or a thoraco-abdominal aortic aneurysm is an abnormal ballooning of the main artery in the body, in the chest or starting in the chest and extending into the abdomen (Fig. 18). These aneurysms are unusual before the age of 60 years, except in some rare inherited diseases. They are more common in people who have smoked and in people suffering from high blood pressure. They are also more common in men than in women. Most aneurysms do not cause any symptoms. Patients with an aneurysm usually do not realise they have one until it is found by a doctor as a result of other medical tests (by coincidence, so to speak). Fortunately, this is the case in most patients, but sometimes the first symptoms occur when an aneurysm ruptures (when the wall bursts).

What is an aortic dissection? A dissection is a tear within the different layers of the wall of the aorta, usually quite sudden. The aorta may "dissect" because of high blood pressure over a longer period. This tear in the lining of the aorta can spread into branches affecting blood flow to areas of the body and or weaken the aorta leading to aneurysm formation or "ballooning" over time. In addition, trauma may lead to a dissection or more extensive damage

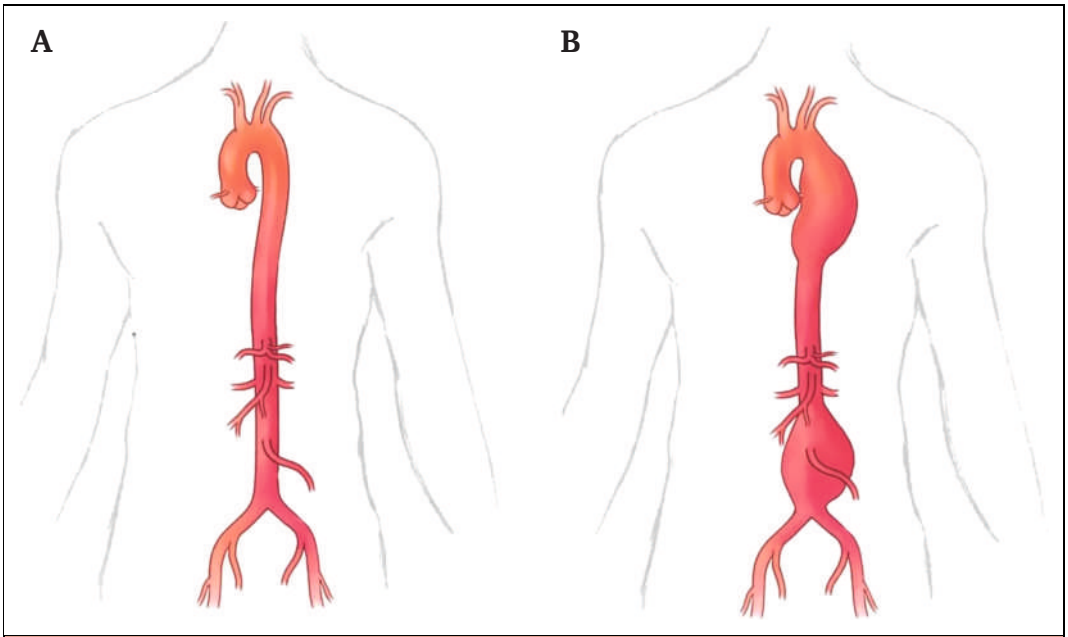


Figure 18. Image showing (A) the path of the normal aorta and (B) an aneurysm in the thoracic aorta and the abdominal aorta.

causing rupture. If the tear starts at the level of the main artery to the left arm, it is referred to as a "type B dissection". If the tear starts closer to the heart, it is referred to as "type A dissection", an acute situation that needs urgent treatment by cardiac surgery teams. Aortic dissection can also be classified as acute (happened recently) or chronic (long ago). An acute type B dissection can be a life threatening emergency and needs to be treated urgently in a hospital by conservative (medication on the ICU), endovascular (the use of stents), or open surgical therapy.

How is a thoracic aortic aneurysm or dissection diagnosed? Physical examination is not reliable for diagnosing aortic diseases, and the part of the aorta in your chest cannot be seen on ultrasound. If someone is suspected of having a dissection or aneurysm in the chest, the best way to confirm the diagnosis is by using computed tomography scanning (a CT scan). This involves the injection of dye into a vein in your body so that the aorta can be seen clearly on the scan. CT scans are most used when an operation to repair an aneurysm is being considered, or if your doctor wants to make sure your aneurysm has not burst. A doctor may suspect a burst aneurysm if someone who is known to have an aneurysm collapses or develops sudden and severe chest, abdominal, or back pain. Nevertheless, most aneurysms in the chest are diagnosed incidentally when patients are being evaluated for other conditions.

What about screening for thoracic aortic aneurysms? These conditions are uncommon and so there are no screening programmes. Thoracic aortic aneurysms are usually detected by investigation for other conditions or symptoms, as mentioned above, or in people with a positive family history who might be deemed to be at higher risk than the general population.

What happens if I am diagnosed with an aneurysm? If you are diagnosed with a thoracic aortic aneurysm, you will be told whether it is small (between 3 cm and 5.9 cm) or large (6 cm or bigger). The size of an aneurysm is usually measured by CT scan. When your aneurysm is small, it is very unlikely to cause you any problems, but you may be advised to have the aneurysm monitored by scans at certain time intervals. In particular, the risk of rupture or bursting is usually very low. Regardless, your doctor or healthcare professional will give you advice regarding your lifestyle such as smoking cessation, adequate blood pressure control, medication, healthy living, and diet. The frequency of monitoring (repeat CT scans) depends on several factors, but in particular the size of the aneurysm.

What happens if I am diagnosed with a dissection? If you are diagnosed with a thoracic aortic dissection and there are signs of rupture or reduced blood supply to vital organs or the legs, emergency surgery may be needed to repair the tear in the vessel wall and restore blood flow. In most cases, however, this is not necessary. Instead, treatment usually involves blood pressure lowering medications and regular CT scans to monitor the aorta throughout life. If the aorta later becomes enlarged (aneurysmal), treatment will follow the same approach as for aneurysms.

If I have an aneurysm, what is the risk of it bursting? If your aneurysm is small, the risk of it bursting is extremely small. The risk of aneurysm rupture increases as the size of the aneurysm increases. The estimated average growth rate of descending thoracic and thoraco-abdominal aortic aneurysms is < 2 mm in diameter per year, increasing with aortic size at the time of diagnosis. Most ruptures occur with aneurysms above 6.0 cm. Women or people of small stature are at higher risk of rupture at smaller diameter, including below 6.0 cm in some cases.

What can I do to stop an aneurysm progressing? A healthy lifestyle including changes to diet, regular physical activity, smoking cessation, and adequate blood pressure control are the main pillars to try to reduce the growth rate. These measures do not prevent the need to monitor the size of the aneurysm.

If I have an aneurysm, will it affect other parts of my body or my general health? For further information, please see the abovementioned guideline relating to the management of aneurysms in the abdomen, which are much more common. This guideline is available online for free. The development of an aortic aneurysm may be related to "atherosclerosis" (narrowing of blood vessels). If you are diagnosed with an aneurysm, it is therefore important to check for cardiovascular risk factors such as high blood pressure, high cholesterol, diabetes, and smoking. Your doctor may also recommend tests of your heart, lungs, and other blood vessels.

What happens if I have a small aneurysm and it gets bigger? If your aneurysm grows and becomes a large aneurysm, your doctor is likely to recommend an operation to repair it, depending on your age and your general condition. For many patients this will not happen in their lifetime. We recommend that for men, if their thoracic aneurysm grows to a diameter of 6.0 cm or more, they should be referred to a vascular surgeon for consideration of surgery. For women, who are often smaller in body size, a lower (5.5 cm) threshold may be considered. If a patient is suffering from a hereditary vascular disease such as Marfan syndrome, treatment can be recommended a bit earlier.

How is an operation to repair an aneurysm performed? An operation for a thoracic aortic aneurysm can be done endovascularly or by open surgery. The choice of method is determined by anatomy and patient related factors, but usually the endovascular technique is preferred due to its lower invasiveness.

An endovascular operation is carried out through small cuts in the groin, and sometimes also in the arms. Using Xray control, a graft material attached to a supporting "stent" is passed up from the arteries in the groin to reline the diseased aorta from the inside. These stent grafts may include pre-formed holes, so called fenestrations, or side arms (branches), to enable connection with the important branches of the aorta. Not everyone can have an endovascular aneurysm repair. Much depends on what the aorta looks like on the CT scan.

An open operation to repair a thoracic aortic aneurysm is performed through a large cut in the chest and sometimes also the abdomen. The aorta is identified and the blood flow through the aorta temporarily stopped. The surgical technique includes maintaining blood flow to the important structures in the lower body, including the bowel and kidneys, via a temporary bypass. The flow in the aorta itself is stopped using clamps, and a prosthetic graft is used to replace the affected aorta. The blood flow through the aorta can then be restored.

What are the advantages and disadvantages of open and endovascular repair? The main advantage of endovascular repair is that it is a much less invasive procedure, compared with open repair, as the whole chest does not need opening. This usually results in a shorter time in hospital around the time of the operation, a better chance of survival, and a lower risk of serious complications. The main disadvantage of endovascular repair is that after surgery you will need to be monitored with further scans to make sure problems do not arise relating to the stent grafts. Sometimes the sealing is not perfect over time, which can lead to the formation of so called "endoleaks". These mean there is further blood flow into the aneurysm from a variety of sources. This can in some cases cause the aneurysm to expand again. Some patients need additional surgery in their lifetime to prevent further aneurysm expansion and rupture. The monitoring performed after surgery usually requires CT scanning. The use of radiation and contrast solution (dye) can increase the risk of causing cancer and kidney disease over a lifetime. The ESVS recommends that after repair of a thoracic or thoracoabdominal aortic aneurysm, whether by endovascular or open surgery, patients should be offered regular follow up examinations to look for the effectiveness of the repair and to check the other blood vessels.

What happens if I am not fit enough to have an operation to repair my aneurysm? In some patients neither an open nor an endovascular procedure is possible. Conservative therapy consists of optimal antihypertensive therapy and healthy lifestyle including smoking cessation, adequate physical activity, and healthy diet as mentioned above. It is the task of your vascular surgeon to discuss all options and advise the best option for you. If the aneurysm is small and or the risk of treatment too high, the best option might be conservative treatment with medication.

What happens if an aneurysm bursts? This situation is a life threatening medical emergency and urgent help or treatment in a hospital is required. In this case, the only chance is rapid treatment, open or endovascular, in an experienced large volume centre, to stop the bleeding. This procedure is, however, very complex, with a high risk of not surviving, even in the best hands.

What rare causes are there for thoracic aortic aneurysm? Most aneurysms are caused by a combination of an individual's genetic predisposition and environmental factors, especially smoking, that damage the structure of the aortic wall causing weakness. In some rare cases an aneurysm can be caused by other factors. It is difficult to recommend treatments for these rare aneurysms because we generally know less about diseases that are uncommon. Some genetic conditions cause aneurysms. These are usually treated by experts in clinical genetics in combination with surgeons, if there is a need to repair the aorta. In such cases, the choice of surgical technique should be individualised.

Most rare aneurysms that occur later in life are due to infection, inflammation, or form as a result of other diseases of the aorta. The treatment for these aneurysms can be different from the usual sort of aneurysm and the recommendations above may not apply. An experienced

vascular surgeon should be able to discuss these with their patients.

12. UNRESOLVED ISSUES

A fundamental challenge in managing DTA and TAbdAo disease is the persistent lack of high quality evidence. These conditions are relatively rare, heterogeneous, and involve highly complex management, which limits the feasibility of large, robust studies. Consequently, much of the available data still derive from single centre, retrospective reports with inherent selection and reporting biases. In recent years, multicentre consortia have emerged, enabling larger pooled analyses with somewhat reduced bias. However, these efforts largely involve a small group of high volume aortic centres collaborating in various constellations. As a result, much of our current knowledge is repeatedly drawn from approximately the same cohort of 2 000 patients who have undergone OSR and a similar number treated with complex endovascular repair, with these datasets being re-analysed across multiple publications. This limited and repetitive evidence base creates uncertainty and variation in clinical decision making, often leaving treatment thresholds and strategies to rely heavily on expert opinion rather than robust data. The generalisability of conclusions based on the available data, derived from a few academic high volume aortic centres, can also be questioned.

The field urgently awaits data from well designed prospective registries and RCTs, and several such initiatives are already underway. In the meantime, the 2026 guidelines aim to mitigate this uncertainty by emphasising individualised, multidisciplinary care in experienced high volume aortic centres, supported by structured SDM with patients. Furthermore, it is strongly encouraged to actively participate in national and international registries to expand the collective evidence base and improve future guideline development.

Specific unresolved issues include the following:

- Centralisation: DTA and TAbdAo pathologies should be managed in high volume, specialised aortic centres by experienced and dedicated multidisciplinary teams, particularly in complex scenarios such as those associated with genetic aortopathy, pregnancy, or infection. There is a well recognised volume outcome relationship, and although defining the optimal threshold is challenging, a minimum of 20 operations per year is proposed as a reasonable benchmark. This threshold is probably conservative and may need to be higher, but additional data are required before more definitive recommendations can be made. Centralising the management of DTA and TAbdAo pathologies is further justified by the need to keep up with the rapid medical and technological advances and to ensure access to the full spectrum of contemporary treatment options. Potential drawbacks of centralisation should be addressed, including ensuring effective transfer networks and sustaining teaching and learning in non-centralised emergency departments.
- Assessment of rupture risk: a major limitation of current practice is the reliance on aortic diameter as the primary predictor of dissection or rupture. There is a clear need for more personalised risk stratification tools and a deeper understanding of the underlying pathophysiology to better identify patients at the highest risk. Several RCTs evaluating primary TEVAR *vs.* medical management for uncomplicated ATBAD are ongoing and the results are highly anticipated. Based on limited evidence, the principal threshold diameter for considering repair in CTBAD, DTAA, and TAAA remains set at 6 cm. However, the optimal diameter threshold for intervention remains uncertain. While RCTs are unlikely to be feasible, better prospective follow up data on small DTAA and TAAA could help refine these estimates. It is likely that future thresholds will need to be stratified based on additional factors such as ASI, underlying pathology, genetic predisposition, and other individual risk factors, thus highlighting the need for more personalised risk assessment tools that incorporate factors beyond diameter.
- Assessment of procedural risk: equally as important as identifying patients at risk of aortic related complications which may justify prophylactic aortic repair, is determining which patients can safely tolerate surgery. Better tools are needed to identify those at risk of procedure related complications. Beyond traditional measures of comorbidity, factors such as functional capacity and QoL play a critical role. In recent years, frailty has emerged as a particularly relevant concept for patients with DTAA and TAAA, who often require highly complex and high risk surgical interventions. This underscores the importance of thorough multidisciplinary assessment and structured SDM to ensure that the potential benefits of intervention outweigh the risks for each individual patient.
- Medical treatment: evidence guiding secondary preventive medical treatment and post-operative medical therapy in patients with DTAA and TAAA remains limited and is often extrapolated from studies into other aortic or cardiovascular pathologies that may not fully reflect the unique characteristics of this patient population. As a result, important questions remain unanswered regarding the optimal management of specific frequently encountered conditions. In particular, better data are needed on initial blood pressure management of ATBAD, how to effectively treat shaggy aorta to reduce embolic risk, how to manage floating thrombi in the thoracic aorta, and how to balance antithrombotic therapy following complex endovascular repairs such as FBEVAR. These areas represent clear opportunities for prospective studies, and RCTs would be both feasible and highly valuable to establish evidence based protocols tailored to patients with DTAA and TAAA. Generating such targeted evidence could significantly improve patient outcomes, reduce peri-operative and late complications, and optimise long term survival and QoL. However, the rarity and heterogeneity of these

pathologies limit the feasibility of large scale RCTs, underscoring the importance of collaborative multicentre registries and pragmatic study designs to advance knowledge in this field. International registries and research consortia could play a pivotal role in overcoming these limitations by pooling data from high volume centres, standardising outcome reporting, and enabling large scale analyses that would otherwise be impossible at single institution level.

- Durability of complex endovascular repair: although complex endovascular repair has become the preferred treatment for most DTA and TAbdAo pathologies with suitable anatomy, its long term durability remains the major source of uncertainty. This concern is particularly pronounced in younger patients, where life expectancy may exceed the proven lifespan of current devices, as well as in cases requiring FBEVAR, which involves more extensive sealing zones and multiple branch connections. While short and midterm results are encouraging, robust data on outcomes beyond five to ten years are scarce. To better define the true durability of these techniques, larger cohorts with standardised reporting and substantially longer follow up are essential. Such data would not only guide patient selection but also inform the evolution of device design, surveillance strategies, and potential indications for earlier re-intervention. Centralised follow up in high volume aortic centres, combined with participation in national and international registries, will be crucial to systematically capture these long term outcomes and build a stronger evidence base for future practice. Moreover, patients treated with complex endovascular repair are recommended for lifelong imaging surveillance to detect device related complications or disease progression at an early stage, ensuring timely intervention when necessary.
- Genetic aortopathy: the expanding role of endovascular techniques in patients with genetic aortopathies, such as MFS, LDS, and vEDS, requires careful evaluation. While these approaches offer less invasive alternatives to OSR, their long term durability and safety in patients with inherently fragile connective tissue remain uncertain. Concerns include progressive disease in untreated aortic segments, higher risks of device related failure, and challenges in achieving durable proximal and distal sealing zones. Prospective data and longer term follow up are needed to clarify patient selection criteria, optimise procedural strategies, and determine whether endovascular repair can provide outcomes comparable to open surgery in this high risk population. Collaborative multicentre registries and dedicated studies will be essential to establish evidence based recommendations for the use of endovascular repair in genetic aortopathies. The same applies to endovascular treatment of mycotic aneurysms, with the risk of late infection related complications.
- New technology: the development of endovascular technology is ongoing and evolving rapidly, with the constant introduction of new devices and techniques. This dynamic landscape necessitates careful and continuous monitoring of outcomes, safety, and durability. To achieve this, prospective registries are essential, providing real world data that can guide clinical practice, inform regulatory decisions, and support future guideline updates.
- Re-intervention: in the context of DTA and TAbdAo disease management, it is important to recognise that re-intervention after endovascular repair should not automatically be regarded as a failure but rather as an integral component of the overall treatment strategy. Complex aortic disease often requires a staged or stepwise approach, where secondary procedures, whether planned or unplanned, serve to maintain or restore durable exclusion of the aneurysm, optimise branch vessel patency, or address disease progression in untreated segments. This paradigm reflects the chronic nature of aortic pathology and the evolving capabilities of endovascular therapy, emphasising long term surveillance and timely re-intervention as essential elements of comprehensive patient care rather than indicators of initial treatment inadequacy. Such an approach underscores the need for lifelong imaging follow up and management in specialised high volume centres, where the expertise and infrastructure are available to ensure optimal outcomes over the patient's lifetime. It also highlights the importance of thorough pre-operative counselling and SDM, ensuring that patients understand the chronic nature of their disease, the likelihood of future interventions, and the commitment required for ongoing surveillance and care.
- Miscellaneous: several critical areas in the management of DTA and TAbdAo diseases remain insufficiently supported by evidence and warrant further research. High priority topics include the prevention and treatment of SCI and peri-operative stroke, as well as strategies for managing type II and undefined endoleaks, and dSINE. Additional uncertainty surrounds the indications for and optimal methods of LSA revascularisation, as well as the most effective approaches for FL management in chronic dissections. Beyond these, there is a lack of robust data on the natural history and appropriate follow up of non-aneurysmal, asymptomatic arteria lusoria, and the threshold at which intervention should be considered. Addressing these knowledge gaps through prospective studies, registries, and, where feasible, RCTs would substantially improve the evidence base and refine clinical decision making for these complex aortic pathologies.
- Technical aspects: important technical aspects of complex endovascular repair still require clarification and standardisation. These include the risk and management of bridging stent instability, optimal stent selection for various anatomic and pathological scenarios, and the choice between fenestrations and branches for target vessel incorporation. Further questions remain regarding the relative advantages of external *vs.* internal branch configurations and the role of adjunctive techniques, such as electrocautery, in optimising device

deployment. Addressing these unresolved technical issues through systematic evaluation, shared procedural experience, and dedicated research will be crucial for improving the safety, durability, and reproducibility of complex endovascular procedures. Progress in this field will also depend on close collaboration between clinicians, engineers, and industry partners to drive device innovation and develop solutions tailored to the unique challenges of DTA and TAbdAo repair.

- Cost and QoL: there is a notable lack of formal cost effectiveness analyses for the management of DTAA and TAAA, despite the substantial healthcare resources required for both open and complex endovascular repairs. FBEVAR procedures are significantly more expensive than standard EVAR, primarily due to the cost of the custom designed grafts and the potential for complications. A better understanding of the economic impact of different treatment strategies, including procedure costs, re-intervention rates, long term surveillance requirements, and complication management, is essential for guiding resource allocation and healthcare policy. In addition, more data are needed on the effects of both the disease itself and its various treatments on patients' QoL. Beyond survival, understanding functional outcomes, symptom burden, psychological well being, and long term patient reported outcomes is critical to fully assess the value of intervention. Prospective studies incorporating cost analyses alongside QoL assessments would provide a more holistic view of the true benefits and trade offs of DTAA and TAAA repair. Such evidence would also greatly enhance SDM, enabling patients and clinicians to weigh the risks, benefits, and overall impact of treatment in a more informed and individualised manner. Integrating cost effectiveness metrics and QoL data into national and international registries could provide a powerful platform to generate real world evidence, support health economic evaluations, and further refine patient centred treatment strategies.

ACKNOWLEDGEMENTS

Dr Nader Albin (Uppsala University, Sweden) for support in preparation of Chapters 2 and 7. Dr Joanna Hallman (Medical University of Gdansk, Poland) for support in preparation of Chapter 2. Dr Matthew Joe Grima (Uppsala University, Sweden and University of Malta, Malta), for support in preparation of Chapter 4. Prof. Yskert von Kodolitsch (German Aortic Centre, University Heart & Vascular Centre, University Hospital Hamburg-Eppendorf, Hamburg, Germany) for support in preparation of Chapter 5. Dr Paolo Spath (LMU University Hospital, Germany and University of Bologna, Italy) for support in preparation of Chapters 3.4 and 7. Dr Petroula Nana and Prof. Yskert von Kodolitsch (German Aortic Centre, University Heart & Vascular Centre, University Hospital Hamburg-Eppendorf, Hamburg, Germany) for support in preparation of Chapter 10. Mr T. Söderlund (Sweden) is acknowledged for contributing to proofreading and editing the updated plain English summary in Chapter 11. Dr Maysam Shehab (Tel Aviv University, Israel and Uppsala University, Sweden) is acknowledged for making the figures in Chapters 4, 10, and 11.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2025.12.050.

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