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CLINICAL PRACTICE GUIDELINE DOCUMENT

European Society for Vascular Surgery (ESVS) 2024 Clinical Practice Guidelines on the Management of Abdominal Aorto-Iliac Artery Aneurysms☆

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Objective: The European Society for Vascular Surgery (ESVS) has developed clinical practice guidelines for the care of patients with aneurysms of the abdominal aorta and iliac arteries in succession to the 2011 and 2019 versions, with the aim of assisting physicians and patients in selecting the best management strategy.
Methods: The guideline is based on scientific evidence completed 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 a modified European Society of Cardiology grading system, where the strength (class) of each recommendation is graded from I to III and the letters A to C mark the level of evidence.
Results: A total of 160 recommendations have been issued on the following topics: Service standards, including surgical volume and training; Epidemiology, diagnosis, and screening; Management of patients with small abdominal aortic aneurysm (AAA), including surveillance, cardiovascular risk reduction, and indication for repair; Elective AAA repair, including operative risk assessment, open and endovascular repair, and early complications; Ruptured and symptomatic AAA, including peri-operative management, such as permissive hypotension and use of aortic occlusion balloon, open and endovascular repair, and early complications, such as abdominal compartment syndrome and colonic ischaemia; Long term outcome and follow up after AAA repair, including graft infection, endoleaks and follow up routines; Management of complex AAA, including open and endovascular repair; Management of iliac artery aneurysm, including indication for repair and open and endovascular repair; and Miscellaneous aortic problems, including mycotic, inflammatory, and saccular aortic aneurysm. In addition, Shared decision making is being addressed, with supporting information for patients, and Unresolved issues are discussed.
Conclusion: The ESVS Clinical Practice Guidelines provide the most comprehensive, up to date, and unbiased advice to clinicians and patients on the management of abdominal aorto-iliac artery aneurysms.

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LIST OF ABBREVIATIONS

3D	Three Dimensional
AAA	Abdominal Aortic Aneurysm
ACS	Abdominal Compartment Syndrome
ACT	Activated Clotting Time
ACF	AortoCaval Fistula
AGI	Aortic Graft Infection
AGREE	Appraisal of Guidelines Research and Evaluation
AOB	Aortic Occlusion Balloon
AP	Anteroposterior
AUI	Aorto-Uni-Iliac
bEVAR	Branched EVAR
BP	Blood Pressure
CEUS	Contrast Enhanced Ultrasound
chEVAR	Chimney EVAR
CIA	Common Iliac Artery
CIN	Contrast Induced Nephropathy
COS	Core Outcome Set
CMD	Custom Made Device
COPD	Chronic Obstructive Pulmonary Disease
COVID-19	Coronavirus Disease 2019
CPR	CardioPulmonary Resuscitation
CRP	C Reactive Protein
CT	Computed Tomography
CTA	Computed Tomography Angiography
DSA	Digital Subtraction Angiography
DST	Decision Support Tool
DUS	Duplex Ultrasound
DVT	Deep Venous Thrombosis
EIA	External Iliac Artery
eGFR	Estimated Glomerular Filtration Rate
EJVES	European Journal of Vascular and Endovascular Surgery
ePTFE	Expanded PolyTetraFluoroEthylene
ERAS	Enhanced Recovery after Surgery
ESC	European Society of Cardiology
ESVS	European Society for Vascular Surgery
EVAR	EndoVascular Aneurysm Repair
EVAS	EndoVascular Aneurysm Sealing
FDA	the United States Food and Drug Administration
FDG	FluoroDeoxyGlucose
FEV1	Forced Expiratory Volume in one second
fEVAR	Fenestrated EVAR
FVC	Forced Vital Capacity
GEF	Graft Enteric Fistula
GSC	Guideline Steering Committee
GWC	Guideline Writing Committee
HR	Hazard Ratio
IAA	Iliac Artery Aneurysm
IAAD	Isolated Abdominal Aortic Dissection
IAH	Intra-abdominal Hypertension
IAP	Intra-abdominal Pressure
IBD	Iliac Branched Device
ICU	Intensive Care Unit
IFU	Instructions For Use
IgG4	ImmunoGlobulin G4
IHD	Ischaemic Heart Disease
IIA	Internal Iliac Artery
IMA	Inferior Mesenteric Artery
IMH	Intramural Haematoma
ITI	Inner to Inner
IVC	Inferior Vena Cava
IVUS	IntraVascular UltraSound
LELE	Leading Edge to Leading Edge
LMWH	Low Molecular Weight Heparin
LoE	Level of Evidence
MET	Metabolic Equivalent
MI	Myocardial Infarction
MRA	Magnetic Resonance Angiography
MRI	Magnetic Resonance Imaging
NAAASP	National Abdominal Aortic Aneurysm Screening Programme
OR	Odds Ratio
OSR	Open Surgical Repair
OTO	Outer to Outer
PAOD	Peripheral Arterial Occlusive Disease
PAU	Penetrating Aortic Ulcer
PET	Positron Emission Tomography
PMEG	Physician Modified EndoGraft
PROM	Patient Reported Outcome Measure
PTFE	PolyTetraFluoroEthylene
QoL	Quality of Life
rAAA	Ruptured Abdominal Aortic Aneurysm
RCT	Randomised Controlled Trial
SCI	Spinal Cord Ischaemia
SDM	Shared Decision Making
SMA	Superior Mesenteric Artery
SVS	Society for Vascular Surgery
T2EL	Type 2 EndoLeak
TAAA	Thoraco-Abdominal Aortic Aneurysm
UK	United Kingdom
US	UltraSound
USA	United States of America
vEDS	Vascular Ehlers—Danlos Syndrome
VQI	Vascular Quality Initiative
WBCS	White Blood Cell Scintigraphy

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WHAT'S NEW IN THE 2024 GUIDELINES?

Each section of the 2024 European Society for Vascular Surgery (ESVS) abdominal aorto-iliac artery aneurysm guidelines has been revised or rewritten. Compared with the previous version (2019), there are 160 recommendations, of which 59 are completely new (including seven Class I), and 49 recommendations have been regraded or significantly rephrased with a changed meaning to some extent. Only 52 recommendations have not been changed. This reflects the increase in knowledge about abdominal aortic aneurysm (AAA) and the rapid technical and medical developments in the field, with the urgent need to update information from the 2019 guidelines.

The 2024 ESVS guidelines benefit from 474 new references published between 2019 and 2023, including 16 primary or secondary analyses from randomised controlled trials (RCTs), 106 systematic reviews and or meta-analyses, and 84 studies based on vascular registries or quality initiative programmes. Nevertheless, only 10/160 (6%) recommendations are based on Level A evidence, of which five are Class I and two are Class III, and as many as 112 (70%) recommendations are limited to Level C evidence or consensus, illustrating the overall weak state of evidence that still prevails in the aortic field.

The section on quality control (Table 4) presents a newly defined core outcome set (COS) (consisting of six key patient related outcome measures) for elective AAA repair, developed through a European wide consensus survey involving all stakeholders including patient representatives. In section 2.3, the recommended minimum yearly caseload has been upgraded to at least 30 standard AAA repairs per centre (no less than 15 of each open and endovascular repair), and a consensus recommendation on a minimum yearly caseload of complex AAA repairs has been added. The updated chapter also addresses the importance of simulation based training.

Ultrasound (US) remains the recommended primary modality for the diagnosis and follow up of small AAAs, but it is still not possible to suggest one calliper placement over another. The background to this and the clinical consequences of different calliper placements are discussed at length in the updated Chapter 3. Against the background of the dramatically changed epidemiology, mainly the decreasing prevalence of AAA, a thorough re-evaluation of the screening recommendations has been made. In section 3.3 screening of high risk groups remains highly recommended (Class I, Level A), but the target groups are not defined in the recommendations as previously, but should be made depending on local conditions, such as prevalence of the disease, life expectancy and healthcare structure.

Sex specific surveillance intervals are specified in the updated Chapter 4, and a new recommendation has been issued to terminate continued surveillance when futile. The importance of cardiovascular risk factor management has been strengthened. Based on a comprehensive analysis of the available evidence it is not considered to be justified to restrict the use of fluoroquinolone antibiotics in patients with AAA as was previously suggested by the United States Food and Drug Administration (FDA) and European Medicines Agency (EMA). Similarly, this guideline advises against restricting exercise and sexual activity in patients with AAAs.

Section 4.4, on indications for repair has been significantly revised. In line with the evidence, a clear negative recommendation is now issued for the repair of AAA < 55 mm in men and < 50 mm in women. The diameter threshold for when repair can be considered is maintained at 55 mm for men and 50 mm for women; however, the recommendations have been downgraded due to the lack of supporting high quality evidence. Furthermore, in line with recent data, it is clarified that the diameter threshold for considering repair should preferably be based on the US measurement.

The section on intra-operative heparin administration and venous thromboprophylaxis has been updated in Chapter 5, and the use of prophylactic mesh reinforcement of midline laparotomies has been upgraded (Class IIa Level A) based on new RCT data. Following reports of failed endovascular aortic repair (EVAR) devices, this guideline advocates the use of devices with proven durability and advises against EVAR outside the manufacturer's instruction for use (IFU) in the elective setting. Long term follow up in prospective registers of updated devices based on established platforms is recommended as before, however with the increased requirement for 10 years of durability data. Due to the lack of evidence of a clinically relevant benefit, routine pre-emptive coiling of side branches or non-selective aneurysm sac embolisation before EVAR is not recommended. The impetus towards EVAR as the preferred treatment modality for AAA in most patients is retained as outlined in section 5.3.3.

In Chapter 6 the recommendation of using aortic balloon occlusion for proximal control is downgraded due to the uncertainty of its effect, while the recommendation for vacuum assisted open abdominal closure has been upgraded, and with the addition of mesh traction. Other news includes the need for proper stent graft oversizing, the role of anticoagulation in the emergency setting is discussed, a diagnostic process of colonic ischaemia after ruptured abdominal aortic aneurysm (rAAA) repair is presented, and the chapter now also covers treatment of aortocaval fistula.

Chapter 7 on follow up has undergone a thorough update. Recently published key studies warranted an update of treatment recommendations for aortic graft and stent graft infections. Several new and updated recommendations on the management of endoleaks are

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presented in section 7.4, an updated recommended follow up algorithm after EVAR is presented in section 7.4.2 (Fig. 6) and a suggested diagnostic step up for occult undetermined endoleaks described in section 7.4.3 (Fig. 7) where the option of conversion to open surgical repair (OSR) with stent graft explantation is highlighted.

Chapter 8 on complex AAAs has been expanded significantly to reflect advances in technology since 2019 and now covers the management of juxta- and pararenal AAAs as well as suprarenal AAAs and type 4 thoraco-abdominal aortic aneurysms (TAAAs). Treatment recommendations have been updated based on an increasingly comprehensive body of knowledge, including preliminary data from the most recent United Kingdom COMPlex AneurySM Study (UK COMPASS trial). Endovascular repair with fenestrated and branched endografts is considered to have some benefit and is advocated in patients with high surgical risk and complex anatomy. There are updated sections on preservation of renal function, prevention of spinal cord ischaemia, and new technologies, such as off the shelf branched devices, physician modified fenestrated endografts (PMEGs), parallel grafts, and *in situ* fenestration.

The diameter threshold for iliac aneurysm repair was raised from 30 mm in the ESVS 2011 guidelines to 35 mm in the ESVS 2019 guidelines, and now further, to 40 mm. The rationale underlying this decision is detailed in Chapter 9, where follow up intervals for small iliac aneurysm are also specified.

In the updated Chapter 11, it has been established how wall oedema should be assessed when measuring the diameter of inflammatory AAAs, which will have a major impact on the indication for repair. A new strong recommendation advocates preventive celiprolol treatment of all patients with vascular Ehlers—Danlos syndrome.

A new chapter (Chapter 11) on shared decision making (SDM) discusses the evidence for SDM in the AAA setting and provides specific recommendations for its application. In collaboration with patient representatives the Information for Patients section has been thoroughly updated in section 11.2, and the guidelines conclude with a list of unresolved issues, which highlight areas for future research in Chapter 12.

Table 1. New and updated recommendations included in the European Society for Vascular Surgery (ESVS) 2024 clinical practice guidelines on the management of abdominal aorto-iliac artery aneurysms compared with the previous 2019 guidelines. Numbers correspond to the numbers of the recommendations in the guideline document.

New Class I recommendations	
5.	The vascular surgery training curriculum should include simulation based training in open and endovascular aortic repair.
69.	All patients with an abdominal aortic aneurysm undergoing open surgical repair and high risk patients undergoing endovascular repair are recommended to have early post-operative monitoring in an intensive care or high dependency unit.
115.	Patients who have undergone endovascular abdominal aortic aneurysm repair are recommended for long term imaging follow up (regardless of initial risk stratification), to detect late complications and identify late device failure and disease progression.
142.	Patients with mycotic abdominal aortic aneurysms are recommended to be referred to high volume vascular surgical centres, for multidisciplinary management.
156.	Patients with vascular Ehlers—Danlos syndrome are recommended prophylactic treatment with celiprolol.
158.	For patients with abdominal aortic aneurysms with an underlying genetic cause, the threshold diameter for considering repair should be individualised, depending on the underlying genetics and anatomy.
159.	Shared decision making should be facilitated during conversations around abdominal aortic aneurysm screening, surveillance and the management of large asymptomatic abdominal aortic aneurysms being considered for repair
New Class IIa recommendations	
15.	Patients with small abdominal aortic aneurysms, who are either not expected to reach the diameter threshold for repair within their life expectancy, or are unfit for repair, or prefer conservative management, should be considered for discontinuation of surveillance.
26.	Prior to abdominal aortic aneurysm repair, routine imaging screening of the entire aorta, access and femoropopliteal arteries should be considered.
27.	Prior to endovascular abdominal aortic repair, detailed pre-operative procedure planning with computer tomography angiography, including the use of a dedicated post-processing software analysis, should be considered.
48.	All patients undergoing elective abdominal aortic aneurysm repair and deemed at risk of post-operative venous thromboembolism should be considered for thromboprophylaxis.
50.	For open abdominal aortic aneurysm repair, the choice of midline *vs.* transverse or transperitoneal *vs.* retroperitoneal abdominal incision should be considered based on surgeon preference and patient factors.
56.	For endovascular abdominal aortic aneurysm repair, device selection should be considered based on aorto-iliac anatomy and the availability of unbiased long term durability data.

Continued

Table 1-*continued*

76. Patients undergoing endovascular repair for ruptured abdominal aortic aneurysm in whom imaging was performed during permissive hypotension, should be considered for stent graft oversizing of up to 30%.
77. In ruptured abdominal aortic aneurysm repair, intra-operative administration of systemic anticoagulation with heparin should be considered once the rupture bleeding has been controlled.
78. Patients with a ruptured abdominal aortic aneurysm should be considered for post-operative deep vein thrombosis prophylaxis with low molecular weight or unfractionated heparin unless there are signs of ongoing bleeding or of a clinically significant coagulopathy.
84. For patients undergoing open or endovascular treatment for ruptured abdominal aortic aneurysm in whom colonic ischaemia is suspected, flexible sigmoidoscopy should be considered, to confirm the diagnosis.
91. For patients undergoing complete explantation of an infected aortic graft or stent graft, *in situ* reconstruction using biological graft material should be considered the preferred repair modality.
95. For patients with aorta or graft enteric fistula, adjuvant antifungal therapy should be considered, until fungal infection has been properly investigated.
96. For patients treated for aortic graft or stent graft infection deemed at high risk of re-infection or when complete graft removal is not achieved, long term culture specific antibiotic therapy should be considered.
99. For patients undergoing open repair of graft enteric fistula, assessment and management of the enteric defect by a gastrointestinal surgeon should be considered.
100. For patients treated for abdominal aortic aneurysm who are distressed by post-operative new onset sexual dysfunction, referral to specialised teams should be considered.
101. For patients with para-anastomotic aneurysm formation after previous abdominal aortic aneurysm repair, infection as underlying cause should be considered.
102. For patients with non-infectious aorto-iliac para-anastomotic aneurysm formation after previous abdominal aortic aneurysm repair, endovascular repair should be considered preferentially.
105. For patients with compromised proximal seal* after endovascular abdominal aortic aneurysm repair, proximal extension with fenestrated and branched devices should be considered in preference to other endovascular techniques.
108. Patients with persistent aneurysm growth after endovascular treatment attempt(s) to treat Type 2 endoleaks should be considered for elective open conversion with or without graft preservation.
117. For patients undergoing endovascular repair of complex abdominal aortic aneurysms, consideration should be given to limiting the aortic coverage to reduce the risk of spinal cord ischaemia, however without compromising the proximal sealing zone.
118. During endovascular aortic repair of complex abdominal aortic aneurysms, the use of intra-operative image fusion should be considered, to reduce radiation exposure, contrast volume, and operating time.
126. For patients undergoing endovascular repair of a complex abdominal aortic aneurysm a strategy to preserve renal function by dose reduction of iodine contrast media, withdrawal of nephrotoxic drugs and ensuring adequate hydration should be considered.
127. For endovascular repair of complex abdominal aortic aneurysm, preservation of large accessory renal arteries ($\geq$ 4 mm) should be considered.
133. Patients with target vessel obstruction after complex abdominal aortic aneurysm repair should be considered for prompt evaluation for possible revascularisation.
134. For patients with an iliac artery aneurysm (common iliac artery, internal iliac artery, and external iliac artery, or combination thereof), imaging surveillance using ultrasound should be considered; every three years for aneurysms 20 − 24 mm in diameter, every two years for aneurysms 25 − 29 mm in diameter, and yearly for aneurysm $\geq$ 30 mm, taking into account life expectancy, suitability for future repair, concomitant aortic dilatation, and patient preferences.
160. Use of decision support tools to assist patients in their decisions about the management of abdominal aortic aneurysms being considered for repair should be considered.

New Class IIb recommendations

44. Patients undergoing elective endovascular abdominal aortic aneurysm repair may be considered for locoregional anaesthesia in preference to general anaesthesia.
47. Intra-operative use of activated clotting time (ACT) may be considered during open and endovascular abdominal aortic aneurysm repair, to measure the effect of heparin in the individual patient and guide additional heparin administration.
51. Reconstruction of the left renal vein after its division during open abdominal aortic aneurysm repair may be considered if important collaterals have been sacrificed.
71. After endovascular repair of abdominal aortic aneurysm rupture into the inferior vena cava, subsequent endovascular closure of the aortocaval fistula may be considered in the presence of an endoleak associated with increased cardiac output, heart failure, or pulmonary embolisation.
89. Patients treated with endovascular abdominal aortic repair who present with symptomatic, evolving, or haemodynamically significant thrombus formation inside the stent graft may be considered for individualised intervention or escalation of antithrombotic therapy.

Continued

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Table 1-*continued*

92. For patients undergoing complete explantation of an infected aortic graft or stent graft, extra-anatomical reconstruction may be considered an alternative repair modality in frail patients, in cases with extensive infections, or with graft enteric fistula.

94. For selected high risk patients with an isolated (localised) aortic graft or stent graft infection not involving *Candida* and without enteric involvement, partial graft removal, rather than radical explantation, may be considered

104. Patients with compromised sealing zones* without visible endoleak after endovascular abdominal aortic aneurysm repair may be considered for intervention to improve seal, primarily by endovascular means.

106. For selected patients with compromised proximal seal* after endovascular abdominal aortic aneurysm repair, elective open conversion may be considered as an alternative to complex endovascular interventions, provided the surgical risk is acceptable.

119. During endovascular repair of complex abdominal aortic aneurysms the use of on table cone beam computed tomography imaging for completion control may be considered

128. For patients undergoing open or endovascular repair of complex abdominal aortic aneurysm, a policy of reactive (rescue) cerebrospinal fluid drainage may be considered preferable over routine prophylactic cerebrospinal fluid drainage.

131. After endovascular treatment for a complex abdominal aortic aneurysm, duplex ultrasound surveillance may be considered as an alternative to continued computed tomography angiography surveillance after the first post-operative year in selected patients.

132. Patients deemed at risk of bridging stent patency failure after endovascular treatment for complex abdominal aortic aneurysm may be considered for dual antiplatelet therapy in the early post-operative period.

New Class III recommendations

4. Centres treating complex abdominal aortic aneurysms should not have a yearly combined caseload of open and fenestrated and branched endovascular aortic repair of < 20.

18. Having a small abdominal aortic aneurysm is not a contraindication to using Fluoroquinolone antibiotics.

19. Restricting exercise or sexual activity in patients with small abdominal aortic aneurysms* is not indicated.

20. Men with an asymptomatic abdominal aortic aneurysm < 55 mm are not recommended for elective repair.

21. Women with an asymptomatic abdominal aortic aneurysm < 50 mm are not recommended for elective repair

41. Patients undergoing elective abdominal aortic aneurysms repair are not recommended to be on dual antiplatelet therapy or oral anticoagulants during the peri-operative period.*

49. For open abdominal aortic aneurysm repair, routine use of antimicrobial coated grafts to prevent aortic graft infection is not recommended.

57. Endovascular abdominal aortic aneurysm repair outside the manufacturer's instruction for use is not recommended in the elective setting.

63. For patients undergoing endovascular abdominal aortic aneurysm repair, routine pre-emptive embolisation of accessory renal arteries is not indicated.

64. For patients undergoing endovascular abdominal aortic aneurysm repair, routine pre-emptive embolisation of the inferior mesenteric artery and lumbar arteries, and non-selective aneurysm sac embolisation is not indicated.

88. For patients treated by endovascular abdominal aortic aneurysm repair who present with asymptomatic non-obstructive mural thrombus formation limited to the main body of the stent graft, intervention or escalation of antithrombotic therapy is not indicated.

124. Hybrid repair, by means of visceral and renal artery re-routing (bypassing) combined with endovascular exclusion of the aneurysm, is not recommended as the first line treatment for complex abdominal aortic aneurysms.

145. When measuring the diameter of inflammatory abdominal aortic aneurysms to determine the indication for repair, the peri-aortic inflammation or wall oedema should not be included.

Updated Class I recommendations

2. Centres or networks of collaborating centres treating patients with abdominal aortic aneurysms should be able to provide both endovascular and open aortic surgery *(downgraded to LoE C/Consensus)*

9. Computed tomography angiography is recommended for treatment planning once the anteroposterior diameter threshold for elective abdominal aortic aneurysm repair has been met on ultrasound, and for the diagnosis of rupture *(specified; once threshold diameter met on US)*

10. Aortic diameter measurement with computed tomography angiography is recommended using dedicated post-processing software analysis; with consistent calliper placement in an orthogonal plane perpendicular to the aorta *(upgraded to Class I)*

11. Ultrasound screening for early detection of abdominal aortic aneurysm is recommended in high risk populations* to reduce death from aneurysm rupture *(generic with unspecified target groups)*

16. All patients with abdominal aortic aneurysm should receive cardiovascular risk factor management; with smoking cessation*, blood pressure control*, statin and antiplatelet therapy*, and lifestyle advice (including exercise and healthy diet) *(upgraded to Class I)*

37. For patients with abdominal aortic aneurysms and concomitant symptomatic (within the last six months) 50 − 99% carotid stenosis, carotid intervention before elective abdominal aortic aneurysm repair is recommended *(upgraded to Class I and downgraded to LoE B)*

Continued

Table 1-*continued*

58. For newer generations of stent grafts for abdominal aortic aneurysm treatment based on existing platforms, such as low profile devices, long term follow up in prospective registries is recommended, to ensure device performance and procedural durability through 10 years *(long term specified to 10 years)*
61. For endovascular abdominal aortic aneurysm repair by a percutaneous approach, ultrasound guidance is recommended *(upgraded to Class I and LoE A)*
70. Patients with suspected ruptured abdominal aortic aneurysm should undergo prompt imaging of the thoraco-abdominal aorta and of the access vessels with computed tomography angiography *(rephrased)*
72. For patients with ruptured abdominal aortic aneurysm, a policy of permissive hypotension is recommended* *(downgraded to LoE C)*
80. For patients with a ruptured abdominal aortic aneurysm and suitable anatomy, endovascular repair is recommended as the first treatment option *(upgraded to LoE A)*
87. Patients operated on for an abdominal aortic aneurysm with new onset or worsening of lower limb ischaemia are recommended immediate evaluation of graft related problems, such as limb kinking or occlusion *(upgraded to LoE B)*
130. After endovascular treatment for complex abdominal aortic aneurysm, long term imaging surveillance is recommended; with computed tomography angiography within 30 days and one year and thereafter individualised *(rephrased)*
154. Patients with an abdominal aortic aneurysm with a suspected underlying genetic cause, such as early onset ($<$ 60 years) or positive family history of aneurysmal disease, or with physical features associated with monogenetic syndromes, are recommended genetic evaluation *(rephrased)*

Updated Class IIa recommendations

13. Men should be considered for imaging surveillance using ultrasound, every five years for a sub-aneurysmal aorta 25 − 29 mm in diameter, every three years for abdominal aortic aneurysms 30 − 39 mm in diameter, annually for aneurysms 40 − 49 mm, and every six months for aneurysms $\geq$ 50 mm, taking into account life expectancy, suitability for future repair, and patient preferences *(gender specific, including sub-aneurysms, downgraded to Class IIa)*
14. Women should be considered for imaging surveillance using ultrasound, every five years for a sub-aneurysmal aorta 25 − 29 mm in diameter, every three years for aneurysms 30 − 39 mm in diameter, annually for aneurysms 40 − 44 mm, and every six months for aneurysms $\geq$ 45 mm, taking into account life expectancy, suitability for future repair, and patient preferences *(gender specific, including sub-aneurysms, downgraded to Class IIa)*
22. Men with an abdominal aortic aneurysm $\geq$ 55 mm should be considered for elective repair *(downgraded to Class IIa and LoE C)*
35. Assessment of pre-operative nutritional status by measuring serum albumin should be considered prior to elective abdominal aortic aneurysm repair, with an albumin level of $<$ 2.8 g/dL as the threshold for pre-operative correction *(downgraded to Class IIa)*
40. Patients undergoing elective open or endovascular abdominal aortic aneurysm repair should be considered for continuation of established monotherapy with aspirin or thienopyridines (e.g., clopidogrel) during the peri-operative period *(downgraded to Class IIa)*
55. For open abdominal aortic aneurysm repair, prophylactic use of mesh reinforcement of midline laparotomies should be considered *(upgraded to Class IIa)*
60. For endovascular abdominal aortic aneurysm repair, the choice of percutaneous access or cut down should be considered based on patient factors and operator preferences *(rephrased)*
62. For patients undergoing endovascular abdominal aortic aneurysm repair, preservation of large accessory renal arteries ($\geq$ 4 mm) or those that supply a significant portion of the kidney ($>$ 1/3) should be considered, however without compromising adequate sealing *(upgraded to Class IIa)*
83. In the management of open abdomen following decompression for abdominal compartment syndrome after open or endovascular treatment of a ruptured abdominal aortic aneurysm, a vacuum assisted closure system with mesh mediated traction and early abdominal closure should be considered *(mesh mediated traction added)*
90. Patients with an aortic graft or stent graft infection should be considered for radical treatment with complete graft or stent graft explantation as first line treatment *(rephrased and downgraded to Class IIa)*
114. Patients who have undergone endovascular abdominal aortic aneurysm repair and have been stratified as low risk of complications* based on early post-operative computed tomography angiography should be considered for low frequency imaging follow up during the first five years *(upgraded to Class IIa)*
120. For patients with a complex abdominal aortic aneurysm and standard surgical risk, open or endovascular repair should be considered based on fitness, anatomy, and patient preference *(rephrased)*
121. For patients with a complex abdominal aortic aneurysm and high surgical risk, endovascular repair with fenestrated and branched technologies should be considered as first line therapy *(rephrased)*
122. Endovascular repair for a complex abdominal aortic aneurysm using parallel graft techniques should only be considered as an option in the emergency setting, or as a bailout, and ideally restricted to $\leq$ 2 chimneys *(upgraded to Class IIa)*
125. For patients undergoing open repair of a complex abdominal aortic aneurysm with a suprarenal clamp time $>$ 25 minutes, cold renal perfusion should be considered *(upgraded to Class IIa)*

Continued

Table 1-*continued*
129. For patients with a ruptured complex abdominal aortic aneurysm (or who are deemed urgent for any other reason), open surgical or endovascular repair (with an off the shelf branched stent graft, physician modified endograft, *in situ* fenestration, or parallel grafts) should be considered based on patient status, anatomy, and patient preferences *(rephrased and upgraded to Class IIa)*
135. Patients with an iliac artery aneurysm (common iliac artery, internal iliac artery, and external iliac artery, or combination thereof) should be considered for elective repair at a diameter of $\geq$ 40 mm *(larger threshold diameter and upgraded to Class IIa)*
136. The choice of surgical technique for iliac artery aneurysm repair should be considered based on individual patient and lesion characteristics *(rephrased and upgraded to Class IIa)*
148. Patients with an uncomplicated* penetrating aortic ulcer, isolated dissection, or intramural haematoma of the abdominal aorta should be considered for conservative management with best medical treatment and continued surveillance *(downgraded to Class IIa)*
149. Patients with pseudoaneurysm or complicated* penetrating aortic ulcer, isolated dissection, or intramural haematoma in the abdominal aorta should be considered for surgical treatment, preferably by endovascular means *(downgraded to Class IIa)*
Updated Class IIb recommendations
43. Patients undergoing elective open abdominal aortic aneurysm repair may be considered for peri-operative epidural analgesia or catheter based continuous wound analgesia, to maximise pain relief and minimise early post-operative complications *(downgraded to Class IIb and upgraded LoE to A)*
74. Haemodynamically unstable patients with a ruptured abdominal aortic aneurysm undergoing open or endovascular repair may be considered for aortic balloon occlusion under fluoroscopy guidance to obtain proximal control *(downgraded to Class IIb)*
75. Patients undergoing endovascular repair for a ruptured abdominal aortic aneurysm may be considered for a bifurcated device, in preference to an aorto-uni-iliac device, whenever anatomically suitable *(downgraded to Class IIb)*
85. Patients with a symptomatic non-ruptured abdominal aortic aneurysm may be considered for a brief period of rapid assessment and optimisation followed by urgent repair under optimal conditions (ideally during working hours) *(downgraded to Class IIb)*
116. Patients with complex abdominal aortic aneurysms may be considered for elective repair at a diameter of $\geq$ 55 mm in men and $\geq$ 50 mm in women, taking into account fitness for repair, aneurysm anatomy, and patient preferences *(gender specific threshold diameter)*
Updated Class III recommendations
3. Centres performing abdominal aortic aneurysm repair should not have a yearly total caseload of $<$ 30, and not less than 15 each by open and endovascular methods *(new numbers)*
31. Routine pulmonary function testing with spirometry or chest Xray prior to elective abdominal aortic aneurysm repair is not indicated *(downgraded to Class III)*
36. Routine screening for asymptomatic carotid stenosis and routine prophylactic carotid intervention for asymptomatic carotid artery stenosis prior to abdominal aortic aneurysm repair is not indicated *(upgraded to LoE B)*
54. In open abdominal aortic aneurysm repair routine re-implantation of the inferior mesenteric artery is not indicated, but should be reserved for selected cases of suspected insufficient pelvic organ perfusion and the risk of colonic ischaemia *(downgraded to Class III)*

LoE = level of evidence.
* For further information, see the corresponding recommendation box.

1. METHODOLOGY

1.1. Purpose of the guidelines

The ESVS has developed clinical practice guidelines for the care of patients with aneurysms of the abdominal aorta and iliac arteries, in succession to the 2011 and 2019 versions,[1,2] 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 aneurysms of the abdominal aorta and iliac arteries, such as vascular surgeons, 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 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).

The guideline is based on scientific evidence completed 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 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 in the evaluation and management of diseases of the abdominal aorta and iliac arteries become available.

The ESVS 2024 clinical practice guidelines on the management of abdominal aorto-iliac artery aneurysms are

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published in the European Journal of Vascular and Endovascular Surgery (EJVES), as an online open access publication, as well as being free to access via the ESVS website. They are also available on a dedicated ESVS Guideline App (https://esvs.org/blog/2022/09/16/new-and-improved-guidelines-app/).

1.2. Compliance with Appraisal of Guidelines Research and Evaluation II standards

Appraisal of Guidelines Research and Evaluation (AGREE) II reporting standards for assessing the quality and reporting of practice guidelines were adopted during preparation of the 2024 guidelines and a checklist is available (AGREE II checklist). 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, different insurance and healthcare provider structures, pricing levels and economic incentives, which makes costs largely incomparable.

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 abdominal aortic and iliac artery aneurysms (IAAs). The GWC comprised 16 vascular surgeons and one vascular pathologist, from 12 European countries.

The members of the GWC have provided disclosure statements of all relationships that might be perceived as real or potential sources of conflict of interest. These disclosure forms are kept on file at the headquarters of the ESVS. 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 *European Journal of Vascular and Endovascular Surgery* (*EJVES*). This document has been reviewed by 23 reviewers including 11 members of GSC and 12 external reviewers from 15 countries.

1.4. Methodology

1.4.1. Strategy. The GWC held a series of online conferences in June 2021 at which time topics and tasks were allocated, and monthly thereafter. Following preparation of the first draft, GWC members participated in a face to face meeting in Milan, Italy, in March 2022 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 (LoE) was secured via a majority vote of the GWC members. After several online follow up meetings, the WC was able to agree a final set of recommendations on 25 November, 2022. From December 2022 to August 2023, the document underwent three external review rounds. The final version of the guideline was submitted in September 2023.

1.4.2. Literature search and selection. Clinical librarians at the Uppsala University, Sweden, performed the literature search for this guideline systematically in PubMed (MEDLINE), Embase, and the Cochrane Library up to January 2022. Reference checking and hand search by GWC members added other relevant literature, including selected articles published up to August 2023. 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 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. The evidence used in each of the recommendations is detailed in the Table of Evidence (ToE).

1.4.3. Studies commissioned for the guideline. Six reviews and consensus documents were commissioned: (1) contemporary growth rates of small AAAs;[4] (2) prognostic impact of Type 1B endoleaks following EVAR;[5] (3) management of inflammatory aortic aneurysms;[6] (4) management of AAA with suspected genetic disease (work in progress); (5) management of patients treated with Nellix device (Endologix, Inc, Irvine, CA, USA);[7] (6) variability and reproducibility of AAA US measurement;[8] (7) development of a COS for elective AAA repair (work in progress).

1.4.4. Recommendations. The recommendations are graded according to a modified European Society of Cardiology (ESC) grading system, where the strength (class) of each recommendation is graded from I to III (Table 2) and the letters A to C mark the LoE (Table 3). In this modified system, approved by the ESVS GSC, RCT meta-analyses are level A; larger non-RCT meta-analyses are level B; while meta-analyses of small non-randomised studies are level C. Furthermore, pre-defined subgroup analyses of RCTs or large RCT subgroup analyses can be level A, while other subgroup analyses of RCTs should be considered level B.[3]

1.4.5. Limitations. These guidelines have important limitations which affect generalisability. The lion's share of the available evidence relates to men of white ethnicity in highly developed socioeconomic societies. Women specific

Table 2. **Class of recommendations from the European Society of Cardiology (ESC) evidence grading system.**

Class	Definition	Suggested wording
I	Evidence and or general agreement that a given treatment or procedure is beneficial, useful, effective	is recommended (should)
II	Conflicting evidence and or divergence of opinion about the usefulness/efficacy about the given treatment or procedure.	
IIa	Weight of evidence/opinion is in favour of usefulness/efficacy	should be considered
IIb	Usefulness/efficacy is less well established by evidence/opinion	may be considered
III	Evidence or general agreement that a given treatment or procedure is not useful/effective and in some cases may be harmful	is not recommended (is not indicated)

recommendations are given whenever possible, but usually with a lower LoE as these are generally under represented in studies on AAA. Aspects regarding other ethnicities are not covered, nor are the conditions of low and medium income countries or times of war or other situations that may limit health care resources, such as in a pandemic like COVID-19[9] which were considered to be outside the scope of this document. Other conditions that may require adaptation are long distances, inaccessibility of certain products, devices and apparatus, social deprivation and poverty. These limitations must be kept in mind when managing other target groups or when operating in other settings and environments.

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 reader is referred to the ToE or the cited reference.

1.4.6. The patient's perspective. A key aim of this guideline is to optimise SDM. This requires access to high quality 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 which takes his or her preferences, needs, and values into account.

In order to improve accessibility and interpretability for patients and the public, the plain English summaries for these guidelines were subjected to a lay review process. Information for patients was drafted for key sub-chapters, which was read and amended, by a vascular nurse specialist and at least one member of the public or a patient, before going to a patient focus group for their opinions.

Table 3. **Levels of evidence adapted from the European Society of Cardiology (ESC) evidence grading system.**

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, large non-randomised studies, or a meta-analysis of non-randomised studies
Level of Evidence C	Consensus opinion of experts and or small studies, retrospective studies, registries

2. SERVICE STANDARDS

This chapter discusses general recommendations concerning quality control, resource availability, centre volume, and experience, as well as time frames that apply to contemporary management and treatment of AAA. Whenever these requirements cannot be provided locally, patients should be transferred to an appropriate centre, taking into account the patient's preference.

2.1. Quality control

2.1.1. Vascular surgical quality registries. Continuous quality control is an important part of delivering excellent care to patients in vascular surgery, and this certainly holds true for aortic practice. Vascular surgical quality registries exist in several countries and allow for continuous assessment of aortic repair activity and its outcome in participating centres.[10] The role of quality registries in aortic surgery can assess changes in practice, e.g., introduction of screening or new endovascular techniques.[11–13] Population based prospective registries complement RCTs in providing pilot data early on as well as monitoring the generalisability of new treatment strategies and technologies at a later phase.[13,14] High quality and validated registries have a low risk of bias, reflect daily practice, and allow identification of regional or national variations in delivery of care.[15] Aggregated results from RCTs and prospective registries have the potential to guide local vascular surgeons as well as nationwide policy makers.[16]

A recent USA study effectively showed the strength of linking registries to routine claim data in identifying under performing EVAR devices and preventing harm. The early AFX Endovascular AAA System (Endologix, Irvine, CA, USA) had a complication rate (aortic re-intervention) nearly 10% higher in absolute terms than other devices within the first five years after surgery. Although conventional adverse event reporting to the United States FDA ultimately led to the device being recalled in the USA in 2017, the failure of the device could already be identified in 2013 in the linked registry claims surveillance data. Importantly, their study also found that safety outcomes soon after surgery were a poor predictor of a device's long term performance.[13]

Centres performing surgical treatment of AAA should therefore preferably participate in registries allowing continuous quality control assessment but internal and external validity of these registries is of the utmost importance.[17] Validation of quality registries should be performed regularly, with external validation against other data sources such as administrative or claims registries to ensure that registration of cases in the quality registry is not biased and that the registry provides representative and generalisable results. Internal validation requires assessment of key variables against another data source, e.g., patient records or population registry, to ensure reliability of data for analysis (e.g., comorbidity data and post-operative survival or complications).[18]

When using registry data to compare outcomes between centres, regions, or countries, adjustment for differences in case mix is necessary using available case mix adjustment methods.[19–23] While no single risk scoring system can be recommended for case mix adjustment, the need for harmonised data collection with explicit definition of the registered data such as pre-operative risk factors and post-operative outcomes is crucial for any registry based case mix adjustment. The ESVS Vascunet international registry collaboration is in the process of developing recommendations for a minimum dataset for quality registries in aortic surgery, which may serve as a baseline for establishment of local, regional and national registries (www.vascunet.org).

Recommendation 1		Unchanged
Centres performing aortic surgery are recommended to enter cases in a validated prospective registry to allow for monitoring of practice and outcomes.		
Class	**Level**	**References**
I	C	Consensus

2.1.2. Patient reported outcome measures.

When measuring outcome in registries, involvement of the patients' perspective through registration of patient reported outcome measures (PROMs) is valuable. In combination with clinical outcome measures,[24] PROMs support detailed evaluation of new surgical techniques or devices and help develop patient tailored treatment pathways. In a systematic review,[25] four PROMs were identified (Short Form 36, Australian Vascular Quality of Life Index, Aneurysm Dependent Quality of Life (AneurysmDQoL), and Aneurysm Symptoms Rating Questionnaire (AneurysmSRQ)), which had not undergone a rigorous psychometric evaluation within the AAA population. Further, the Aneurysm Treatment Satisfaction Questionnaire (AneurysmTSQ), containing 11 items, and the eight item SF-8 questionnaire have been suggested as post-operative PROMs for patients with AAA.[26–30] While no recommendation can be made regarding inclusion of a specific AAA PROM in vascular registries, further evaluation and refinement of these quality measurement tools and implementation of quality of life (QoL) aspects in vascular registries are warranted.

2.1.3. Core outcome sets.

Systematic reviews of intact AAA and rAAA repair have been consistent in demonstrating the large number and heterogeneity of outcome reporting in trials, registries, and other research studies, and with patient centred and long term outcomes poorly reported.[24] This has the effect of making clinically relevant comparisons between centres, trials, and pooling of results in meta-analyses difficult as well as patient involvement in decision making. To overcome these problems, the concept of COS has been introduced, which provide a minimum set of key outcomes, that all stakeholders, including patients, agree on. COS is developed by a defined process of systematic review, focus groups for under represented stakeholders, a Delphi consensus and an in person consensus.[31]

Within the framework of this guideline, the development of a COS for elective AAA repair was initiated (www.comet-initiative.org registration 1 582) to define the key patient related outcome measures for elective AAA repair through a Europe wide consensus survey, including patients, carers, family members, vascular nurses, vascular surgeons, trainees, interventional radiologists, anaesthetists, and industry partners. Following two rounds of a Delphi consensus (n = 98 and 96 participants with complete responses and 38 and 23 questions respectively) conducted in Greece, Italy, Malta, The Netherlands, Sweden, and the United Kingdom (UK), a consensus meeting was held on 29 June 2023 at the British Society of Endovascular Therapy annual meeting, which included representatives from Italy, Sweden, and the UK and all stakeholder groups.

Table 4 displays the six top scoring COS unanimously endorsed at the meeting. Other outcomes with strong but not unanimous support include; overall patient satisfaction with their treatment, thromboembolic events occurring as a consequence of repair (including limb and bowel ischaemia), re-intervention, retention of social functioning, stroke leading to permanent disability, and kidney damage leading to the need for long term dialysis.

Future work will need to identify the optimal methods for assessing QoL and cognitive functioning.

2.2. Resources and availability

The surgical management of AAA has changed over the past decades, with a shift from OSR as the primary surgical technique for elective and acute AAA repair to EVAR as the predominant strategy for AAA repair in several countries today.[10] The preferential use of EVAR for AAA repair is in line with the previous and current ESVS AAA guideline recommendations. Since > 70% of AAA repairs are performed with EVAR, it has resulted in reduced numbers of OSR, an important tool in management of patients with AAA anatomically unsuitable for standard or complex EVAR. Open surgery and endovascular techniques are complementary techniques for management of complications after aortic repair. Therefore, centres treating patients with AAA

Table 4. Core outcome sets for elective abdominal aortic aneurysm repair.

Post-operative death, 30 days after repair or in hospital if longer than 30 days
Secondary rupture of the aneurysm after repair
Overall quality of life, assessed before repair and after recovery period
Retention of cognitive function, assessed before repair and after recovery period
Long term survival, five years
For endovascular aortic repair only; continued aneurysm sac expansion after repair

should have the resources and expertise required to offer both open and endovascular aortic surgery when required, 24 hours a day and seven days a week.[32,33] Preferential treatment of patients with open surgery due to lack of know how in endovascular techniques within the centre, or complex experimental endovascular repair due to lack of know how when there is a reasonable open surgical option, is not acceptable.

Recommendation 2		Changed
Centres or networks of collaborating centres treating patients with abdominal aortic aneurysms should be able to provide both endovascular and open aortic surgery.		
Class	**Level**	**References**
I	C	Consensus

2.3. Surgical volume

The association between surgical volume (caseload) and outcome has been reported for a range of surgical procedures of varying complexity. In aortic surgery, multiple studies have established an association between higher annual caseload and improved peri-operative outcome.[34] This volume—outcome relationship applies to both elective and acute aortic repair.[35,36] While the association between increasing volume and lower peri-operative mortality has repeatedly been established for OSR, studies also suggest a volume—outcome relationship in standard and complex EVAR in terms of survival[37,38] and outcome of complications.[39]

The established volume—outcome relationship for AAA repair has been confirmed in various health care settings and organisations.[35,37,40] In an analysis of 31 829 procedures from the UK hospital episode statistics data 2011 – 2019, lower hospital annual volume was associated with higher 30 day emergency re-admission rates and a higher 30 day mortality rate after OSR.[35] This dataset also suggests an association between surgeons' caseload and outcome; however, this is harder to interpret in the modern era when AAA repair is performed by teams rather than individuals.[16] In Germany, from an analysis of 96 426 cases from the national Diagnostic Related Group statistics 2005 – 2013, hospital volume was inversely associated with in hospital mortality after OSR and EVAR.[37] Additionally, complication rates, length of stay, and use of blood products were lower in high volume hospitals. In an analysis performed by the International Consortium of Vascular Registries, involving data from > 170 000 AAA repairs from 11 countries between 2010 and 2016, the highest volume centres had a significant reduction in OSR mortality compared with the lowest volume quartile of centres (intact AAA repair: 3.6% *vs*. 6.0%; rAAA repair: 30.2% *vs*. 44.2%).[41] Further analysis of this multinational dataset suggests that this volume—outcome effect may be related to the ability to rescue patients with complications in high volume centres.[39]

The associations between volume and outcome have also been shown in rAAA repair. In nationwide studies from the UK, United States of America (USA), and Sweden, lower mortality was seen in hospitals with larger bed capacity, in teaching hospitals, and in hospitals with higher annual caseloads.[33,36,42,43] In a meta-analysis including data from 13 studies with a total of 120 116 patients, patients treated in low volume centres had a statistically significantly higher peri-operative mortality rate than those treated in high volume centres (OR 1.39; 95% CI 1.22 – 1.59), with a mortality difference in favour of high volume centres for both OSR and EVAR.[36] In a Vascunet study including 9 273 patients from 11 countries treated for rAAA, the peri-operative mortality rate was lower in centres with high caseload volume; 23% in centres doing > 22 repairs per year *vs*. 30% in centres with a caseload ≤ 22, $p < .001$.[44]

Some studies document that it is safe to transfer patients with rAAA to the nearest high volume specialised vascular centre and that such a policy may, in fact, decrease mortality.[45,46] Nationwide and regional surveys in the USA, however, suggest that this practice is not necessarily safe, since transfer was associated with a lower operative mortality but an increased overall mortality when including transferred patients who died without surgery.[47,48]

Surgeon speciality also has an impact on patient outcomes in AAA repair. The relationship between specialty and outcome is related to volume, as surgeons with specialties other than vascular surgery performing aortic repair are likely to have a very low caseload of aortic repairs.[49] In an analysis of elective AAA repairs performed in the USA based on National Inpatient Sample 1997, operative mortality was significantly lower, 2.2% when the operation was performed by vascular surgeons, compared with 4% by cardiac surgeons and 5.5% by general surgeons.[50,51] The likelihood of receiving EVAR rather than OSR was higher when vascular surgeons were involved compared with general and cardiac surgeons.[52] There is, however, no comparative study between vascular surgeons and interventional radiologists, who today represent the two specialities that perform most AAA repairs, and it is important to acknowledge that several centres perform EVAR procedures in a multidisciplinary team setting. Although, no specific recommendation on the specialty is made, the GWC advocates that AAA surgery should be done under the leadership of a vascular surgeon.

In summary, based on the current evidence of a volume—outcome relationship in AAA repair, it is justifiable to recommend a set minimum surgical volume for aortic centres. The specific volume threshold for such a recommendation has however been a matter of debate, and various threshold levels have been suggested by different organisations, often adjusting for local circumstances and political implications in terms of centralisation. Geographic and epidemiological factors, including population density and patient transfer possibilities, are factors that will necessarily affect local decisions regarding availability of aortic services and centralisation. These decisions may override the need for centralisation to maintain volume in geographically remote areas. A minimum volume threshold is however applicable to most centres offering aortic surgery in normal geographic conditions.

In a recent analysis of multinational registry data, the optimal threshold for the volume—outcome relationship after open AAA repair is an annual caseload of 13 − 16 OSR/year, with a peri-operative mortality rate of 4.6% for centres with < 13 cases/year, *vs.* 3.1% for centres with ≥ 13 cases per year.[53,54] It is important to note, however, that only 23% of > 1 000 centres in the 11 countries included in this analysis met the ≥ 13 procedures/year volume threshold, with significant variation between nations (Germany 11%; Denmark 100%). This suggests that there is a need for reorganisation of aortic services to ensure that a minimum volume threshold for OSR is met.

There are only limited data on the volume—outcome relationship for complex AAA repair[38,55,56] (see Chapter 8). However, due to the general relationship between surgical caseload and outcome together with the complexity involved in planning, treating, and following these patients, it is strongly recommended that complex aortic repair should only be performed in centres with a minimum yearly caseload of at least 20 complex repairs. The recommendations regarding the preferred technique for repair of complex aneurysms is defined in a Chapter 8. Unusual and complex aortic disease entities, such as explantation procedures, graft and stent graft infections (see section 7.2.2), mycotic AAA (see section 10.1), and AAAs associated with genetic syndromes (see section 10.5) should be managed by multidisciplinary teams in specialised high volume centres.

Recommendation 3			Changed
Centres performing abdominal aortic aneurysm repair should not have a yearly total caseload of < 30, and not less than 15 each by open and endovascular methods.			
Class	Level	References	ToE
III	B	Landon *et al.* (2010),[32], Gray *et al.* (2020),[35] Kontopodis *et al.* (2021),[36] Trenner *et al.* (2018),[37] D'Oria *et al.* (2021),[39] Sawang *et al.* (2020),[40] Scali *et al.* (2019),[41] Zettervall *et al.* (2017),[57] Trenner *et al.* (2020)[58]	

Recommendation 4		New
Centres treating complex abdominal aortic aneurysms should not have a yearly combined caseload of open and fenestrated/branched endovascular aortic repair of < 20.		
Class	Level	References
III	C	Consensus

2.4. Training in aortic surgery

The paradigm shift in aortic repair with declining OSR numbers has important implications for training. In the USA, the number of open AAA repairs fell by almost 80% over the period 2003 to 2013.[59] In 2014, almost half of senior vascular surgical trainees in the USA were exposed to fewer than five OSRs.[60] Therefore, training in a safe environment using simulated aortic models for OSR and EVAR of AAA has become increasingly important.[61−63] In an ESVS survey about technical procedures that should be included in a future simulation based curriculum in vascular surgery, OSR and EVAR were among the top 10 candidate procedures.[64]

Simulation based training of OSR has been shown to have the greatest impact on junior trainee performance.[63,65,66] For EVAR, generic and patient specific simulation based training results in reduction in peri-operative errors, and overall increased procedure efficiency.[62,67] In endovascular repair of ruptured AAA, simulation based training streamlines the treatment process of unstable patients.[68] Dedicated faculty who instruct trainees, as well as feedback and assessment tools are required to ensure effective simulation based training.[63,69]

With decreasing numbers of open aortic procedures and reduced trainee exposure, OSR may require additional training rotations through centres with higher volumes. Postgraduate fellowships in complex endovascular and open aortic surgery may support the establishment of future generations of aortic surgeons.

Recommendation 5			New
The vascular surgery training curriculum should include simulation based training in open and endovascular aortic repair.			
Class	Level	References	ToE
I	B	Maguire *et al.* (2020),[63] Robinson *et al.* (2013),[65] Lawaetz *et al.* (2021), Desender *et al.* (2016),[67] Desender *et al.* (2017),[70] Saratzis *et al.* (2017)[71]	

2.5. Pathway for treatment

Once the indication for elective repair has been reached in a patient under surveillance, adequate pathways to ensure safe and timely care of the patient are required at the centre performing the planned surgical intervention. The waiting time from decision to repair until the procedure is completed is one aspect of the AAA surgical management which should take into account the risk of rupture, primarily related to the AAA size.[72] Waiting time is also affected by healthcare organisation, availability of resources, and competing health care priorities, as underlined by the COVID-19 pandemic.[73]

There are limited data concerning a reasonable waiting time for treatment once the indication for repair has been reached. In the EVAR 2 trial, a RCT evaluating the long term outcomes in physically frail patients with AAA treated by either early EVAR or no intervention, about 5% ruptured after randomisation but before attempted surgery. The median aortic diameter was 64 mm and the median time between randomisation and repair was eight weeks.[74] Similarly, in an analysis of ruptures occurring during waiting time for complex EVAR, the three month rupture risk was estimated at 6.1% in a cohort of 235 patients with mean aortic diameter of 63 mm.[75] This rupture rate indicates a possible upper limit on the waiting time for surgery. In a meta-analysis, including 11 studies with a total of 1 514 patients reporting follow up of untreated large AAA, the annual rupture rate was 3.5% in AAAs 55 – 60 mm, 4.1% in AAAs 61 – 70 mm, and 6.3% in AAAs > 70 mm.[72] In a contemporary study of the rupture rate of patients with large aneurysms under surveillance 2003 – 2017, the three year cumulative incidence of rupture for aneurysms 50 – 60 mm was 2.2%, *vs*. 6.0% for 61 – 70 mm, and 18.4% for > 70 mm, with a generally higher rupture rate among women compared with men with aneurysms of the same size.[76] In addition, there are psychological consequences of living with a large AAA, which seem to be reversible by surgery,[77,78] which underlines the need to keep the waiting time for referral and treatment to a minimum.

Although there is no strong evidence to support exact timings, it is reasonable to adopt a similar approach as for other potentially lethal diseases, such as malignant disease. A suggested upper limit for the total pathway from referral to treatment is eight weeks once the indication for repair has been reached. This applies, however, only to standard AAA cases, whereas for more complex aneurysms or comorbid patients a lengthier planning or work up time may be justified. Correspondingly, a shorter timeframe should be pursued for larger AAAs. Endovascular repair of complex aneurysms with fenestrated and branched EVAR is generally associated with a waiting time for planning and custom graft production, which in itself carries a risk of interval rupture.[75,79] Industry partners should be encouraged to secure rapid paths to device delivery, ensuring that there are no geographic or centre based biases for delivery time, and minimise manufacturing delays, to enable the total pathway threshold of eight weeks to be met. Measures should be taken to create pathways to minimise waiting time when complex endovascular procedures are planned for patients with large aneurysms. However, if the waiting time becomes too long, alternative treatment options and strategies should be explored.

Recommendation 6			Changed
Patients with an asymptomatic abdominal aortic aneurysm who have reached the size threshold at which repair is considered should receive a fast track pathway* to vascular surgical care.			
Class	Level	References	ToE
I	C	Parkinson *et al.* (2015),[72] D'Oria *et al.* (2022),[75] Lancaster *et al.* (2022),[76] Lindholt *et al.* (2000),[77] Hinterseher *et al.* (2013),[78] Scott *et al.* (2016)[80]	

* An eight week pathway is a reasonable upper limit from referral to elective treatment of an infrarenal AAA, while a shorter timeframe should be considered for larger (> 70 mm) AAAs and a lengthier planning or work up time may be justified for more complex aneurysms or comorbid patients.

3. EPIDEMIOLOGY, DIAGNOSIS, AND SCREENING

3.1. Epidemiology

Aneurysm, from the Ancient Greek word ἀνεύρυσμα, means a dilatation or widening of an artery, most commonly being fusiform in shape.[81] The general definition of an aneurysm is a permanent localised (focal) dilatation of an artery of ≥ 50% increase in diameter compared with the expected normal diameter of the artery in question,[82] while in clinical practice a fixed threshold diameter of 30 mm or more is used to define an AAA,[83] which usually is more than two standard deviations above the mean diameter for men.[84,85] A lower threshold might be more appropriate in women and some Asian populations.

AAA prevalence and incidence rates have decreased over the last 20 years, which has been attributed to the decline in smoking,[86,87] and cardiovascular risk management improvements with better blood pressure (BP) control and widespread use of statins and antiplatelets.[88]

Prevalence is negligible before the age of 55 – 60 years but increases steadily with age. In 1990, the global prevalence in 75 – 79 year olds was 2 423 per 100 000 population *vs*. 2 275 in 2010. At both time points the prevalence was highest in Australasia, North America, and Western Europe and lowest in Latin America and Central Asia.[89] Over the past decades a marked decline in the incidence has been observed. Population screening studies offer the best evidence regarding the contemporary prevalence of AAA. The prevalence in 65 year old men 2006 – 2009 was 1.7% in the Swedish Screening Programme with an additional 0.5% with an already known AAA[87] and 1.3% in the UK National Screening Programme 2009 – 2013.[90] Most recently (2020 – 2021) both national screening programmes report a prevalence < 1%. In contrast, a program in the USA only offering screening to current and ex-smokers reported a prevalence of over 5%.[91] A corresponding 20 – 50% decline in rAAA hospital admissions and incidence of rAAA repair has been reported from several countries in Europe and the USA over the last two to three decades, despite an ageing population.[11,92–94] There have not been any population based studies of the incidence of AAA in men aged 70 years or older in the last 20 years.

A systematic review of population based studies in female cohorts, published between 2000 and 2015 indicates that the pooled prevalence of AAA in women over 60 years was 0.7%,[95] up to fourfold less in women than in men. The studies in this review also used a threshold of 30 mm aortic diameter to diagnose AAA, but women have smaller normal aortic diameters than men.[96] However, alternative definitions based on either normal aortic diameter or body surface area have not been validated at a population level. Similar issues relate to the diagnosis of AAA in specific ethnic groups with smaller aortic diameters than those of white ethnicity.

Smoking is the strongest risk factor for AAA, with an odds ratio (OR) of > 3 for the association,[87] and higher in women.[97,98] A screening and validation study of USA veterans between 50 and 79 years old (n = 114 419) noted the

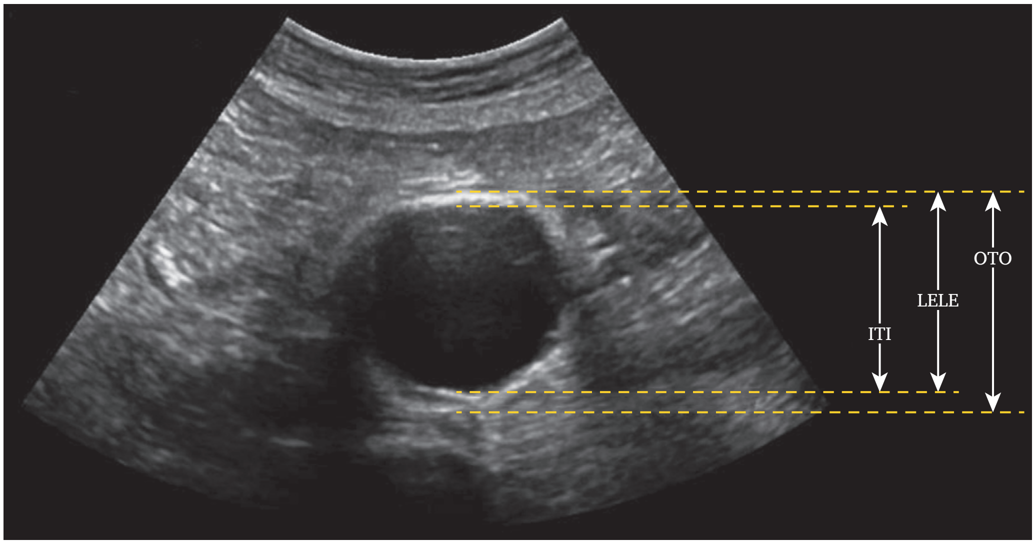

Figure 1. Calliper placement for measurement of aortic diameter. ITI = inner to inner; LELE = leading edge to leading edge; OTO = outer to outer.

highest prevalence of AAA $\geq$ 30 mm of 5.1% in smoking men of white ethnicity.[99]

Other risk factors include atherosclerosis, hypertension, ethnicity, and family history of AAAs.[87,90,98–100] Unique twin registry studies from Sweden and Denmark suggest that the heritability may be as high as 70%.[101,102] The risk of developing AAA in a person with diabetes, especially type 2 diabetes, is about half compared with a person without diabetes.[103,104] AAA is one of only 12 cardiovascular disorders where the incidence does not increase with increasing socio-economic deprivation.[105]

The natural history of a small AAA is progressive growth in the majority of patients. With progressive aneurysm enlargement, the risk of AAA rupture increases. This rupture risk has been estimated as $<$ 1% at one year for 50 mm diameter AAAs, at four years for 40 mm AAAs, and at eight years for 30 mm diameter AAAs.[106]

3.2. Diagnosis

Intact AAAs are usually clinically silent. Symptoms or signs, if present, are mainly pain or tenderness on palpation, localised to the AAA or radiating to the back or to the genitals. Symptoms may be related to complications, either by compression of nearby organs (inferior vena cava, duodenal obstruction, lower limb oedema, ureteral obstruction) or distal embolism. Symptoms of inflammatory AAA are discussed in section 10.2.

For rupture the signs are usually more dramatic; haemodynamic collapse, pallor, abdominal and or back pain, abdominal distension. Symptoms of aortocaval fistulae are discussed in section 6.1.2.

Physical examination may reveal a pulsatile mass, and abdominal palpation has a sensitivity ranging from 33% to 100%, specificity from 75% to 100%, and positive predictive value from 14% to 100%. Detection rates are affected by aortic diameter, experience of the clinician, and body habitus of the patient.[99,107–109] Therefore, abdominal palpation is not reliable for the diagnosis of AAA.

3.2.1. Ultrasonography. Abdominal US and duplex ultrasound (DUS) are first line imaging tools for the detection and management of small AAAs, with high ($>$ 97%) sensitivity and specificity.[110–112] Measurements must be performed in a plane perpendicular to the aortic longitudinal axis, which will vary in the presence of aortic tortuosity.

Different diameters can be measured: anteroposterior (AP), transverse, or maximum in any direction. Intraobserver coefficients of repeatability for the AP and transverse diameters vary from 2 to 8 mm and from 3 to 15 mm, respectively,[113] which supports the use of the AP diameter as the principal measuring plane.

US measurement performed in diastole *vs*. systole, may result in a 2 mm lower diameter.[114] The use of a standardised US protocol including electrocardiogram gating and subsequent offline reading with minute calliper placement reduces variability.[115]

Calliper positioning determines which aortic boundaries are selected to define the diameter;[112] outer to outer (OTO), leading edge to leading edge (LELE), or inner to inner (ITI) (Fig. 1). Due to the lack of consensus on which method is preferable, all three methods are currently in use in different settings; ITI is used in the UK National Abdominal Aortic Aneurysm Screening Programme (NAAASP),[116,117] while the Swedish screening programme uses the LELE method.[118,119]

The existing literature diverges over which calliper placement to use.[116,118,120,121] A recent systematic review and meta-analysis including 21 studies showed that the different methods are quite equivalent in terms of intraobserver variability, while the interobserver variability was lower for the AP OTO calliper placement.[8] The clinical implication of this is however probably of little importance.

More important is the significant difference in crude diameter obtained, with ITI wall measurements being about 3 − 6 mm smaller than OTO wall measurements, and LELE measurements being intermediate.[116,118,120] This crude diameter has a major impact on who will get the diagnosis or not in a screening setting; compared with ITI, the prevalence increases by 31% using LELE and by 77% using OTO.[118]

OTO is thus more sensitive in diagnosing a diseased aorta, and aortas with sub-aneurysmal diameters (25 − 29 mm) based on OTO will have less risk of becoming clinically relevant later. Furthermore, OTO measurements cause the threshold for repair to be reached earlier, which is not desirable. ITI, on the other hand, has the advantage of providing the most relevant measure of threshold for repair with fewer unnecessary operations on small AAAs, and has proven to be safe in the UK screening programme.[117] With ITI, however, it is important to ensure a strict follow up schedule for sub-aneurysmal aortic dilatation, since these may be at a greater risk of becoming an AAA requiring repair (see section 4.1).

Given the variation of evidence and established routines, and the different and partly conflicting clinical effects of the different calliper placements it is not feasible to recommend one measurement method over the other. It is, nonetheless, important to use one method consistently within every clinical programme and to recognise its specific impact on the epidemiology and in clinical decision making. Insufficient attention to reporting standards (specifying plane and positioning of callipers) is an important cause of poor inter- and intra-observer reproducibility.[112] The acceptable standard for measurement repeatability is that the limits of agreement should be ±5 mm (meaning that the difference between measurements is < 5 mm for 95% of measurements).[112]

Recommendation 7			Unchanged
Ultrasonography is recommended for the first line diagnosis and surveillance of small abdominal aortic aneurysms.			
Class	Level	References	ToE
I	B	Concannon *et al.* (2014),[110] Rubano *et al.* (2013),[111] Long *et al.* (2012)[112]	

Recommendation 8			Changed
The anteroposterior plane with consistent calliper placement should be considered the preferred method for ultrasound abdominal aortic diameter measurement.			
Class	Level	References	ToE
IIa	B	Bissacco *et al.* (2023),[8] Long *et al.* (2012),[112] Grondal *et al.* (2012)[114]	

3.2.2. Computed tomography angiography.

Computed tomography angiography (CTA) plays a key role in assessing the extent of disease and therapeutic decision making and planning. CTA is also the recommended imaging modality for the diagnosis of rupture and is an important tool in follow up after repair.[122]

Several issues concerning measurement by US apply to CTA measurement, for example axial *vs*. orthogonal centreline diameters, changes with the cardiac cycle, and details of calliper placement.[123,124] When applying pre-defined methodologies, intra-observer reproducibility can be within the clinically accepted range (±5 mm) in 90% AAA measurements, but the interobserver reproducibility is poor, with 87% of comparisons being outside ± 5 mm.[123] This variability is of great clinical significance, since the number of patients considered for AAA repair, based on a diameter threshold, may vary from 11% to 24%, 5% to 20%, and 15% to 23% for three different radiologists.[123]

CTA provides several advantages for intervention planning: it provides a complete dataset of the thoraco-abdominal aorta and access vessels, which with dedicated post-processing software enables analysis in three perpendicular planes, construction of a centreline, and accurate diameter and length measurement. This reconstruction allows for pre-intervention planning for EVAR and three dimensional (3D) image fusion of CTA and angiography for real time peri-operative guidance. A prerequisite for a good reconstruction is CTA with ≤ 1 mm slice thickness. CTA provides additional information on patency and stenosis of arterial tributaries, position and or duplication of the left renal vein, neck morphology, and aortic wall integrity at the level of the neck, useful for endovascular and OSR planning.

There is often poor agreement between US and CTA diameters, particularly close to the treatment threshold. These differences are probably attributable to inadequate reporting standards with respect to specification of aortic axis, plane of measurement and calliper placement, although differences in instrumentation will also be contributory. Most often, this results in a larger diameter on CTA compared with US, and it has been reported that the mean AP CTA diameter was 4.2 mm larger than AP US diameter[125] and of 50 − 55 mm aortas, up to 70% of AAAs exceed 55 mm on CTA.[126] US is recommended for surveillance of small AAAs and CTA for pre-operative imaging, i.e., CTA should be performed when the size threshold at which repair is considered has been reached, as assessed by US (see section 4.3).

Not infrequently, an AAA is primarily detected on a CT (done for another reason). It is then often reasonable to base a repair decision on that measurement, instead of an US diameter as recommended. However, in the case of a borderline diameter, or in case of uncertainty regarding operability, it may be justified to verify the measurement with US, and base further decision making on the US diameter. Clinical judgment along with SDM with the patient should determine how such a situation is handled.

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Table 5. Summary of randomised trials of population based screening for abdominal aortic aneurysm in men.

Trial characteristics	Chichester, UK[127]	Viborg, Denmark[133]	MASS, UK[128,129]	Western Australia[134]
Number randomised	15 775	12 628	67 800	41 000
Sex	Men and women	Men	Men	Men
Age – y	65–80	65–73	65–74	65–79
Period recruited	1988–1990	1994–1998	1997–1999	1996–1998
Year published	1995	2002	2002	2004
Attendance rate – %	68	76	80	70*
AAA detection rate – %	4; 7.6 in men	4	4.9	7.2
Place of screening	Hospital	Hospital	Community	Community
Intervention policy	At 60 mm	At 50 mm measured as external diameter	At 55 mm measured as internal diameter	None
Mean follow up – y	4.1	13.0	13.1	12.8
AAA mortality, OR (95% CI) screened *vs.* not	0.59 men only (0.27–1.29)	0.31 (0.13–0.79)	0.58 (0.42–0.78	0.91 (0.68–1.21)
All cause mortality, OR (95% CI) screened *vs.* not	1.07 (men only) (0.93–1.22)	0.98 (0.95–1.02)	0.97 (0.93–1.02)	0.98 (0.96–1.01)

* As percentage of those alive when invitation for screening was sent: randomisation predated this invitation by several months in a large sector of subjects. MASS = Multicentre Aneurysm Screening Study; OR = odds ratio; CI = confidence interval.

Recommendation 9			Changed
Computed tomography angiography is recommended for treatment planning once the anteroposterior diameter threshold for elective abdominal aortic aneurysm repair has been met on ultrasound, and for the diagnosis of rupture.			
Class	**Level**	**References**	**ToE**
I	C	Long *et al.* (2012),[112] Oliver-Williams *et al* (2019),[117] Biancari *et al.* (2013)[122]	

Recommendation 10			Changed
Aortic diameter measurement with computed tomography angiography is recommended using dedicated post-processing software analysis; with consistent calliper placement in an orthogonal plane perpendicular to the aorta.			
Class	**Level**	**References**	**ToE**
I	C	Mora *et al.* (2014)[123]	

3.3. Screening for abdominal aortic aneurysm

There have been four randomised trials of population based screening for AAA in men in the UK, Denmark, and Australia[127–129] and one small trial of screening in women in the UK[130] (Table 5). The four screening trials in men have been summarised in a Cochrane Review and by the USA Preventive Services Task Force.[131] Overall there was a significant reduction in AAA specific mortality with the Cochrane review reporting the OR in favour of screening for men as 0.60 and the USA Preventive Task Force reporting an OR of 0.53. At the longest reported follow up from each trial, all cause mortality was statistically significantly lower in the groups invited to screening, risk ratio 0.987 (p = .03).[132] A Swedish nationwide study later confirmed the result from the RCTs in a contemporary population based setting.[119]

The principal harms of screening are associated with an increased rate of elective AAA repair (with its associated morbidity and mortality) and effects on QoL. The number of elective repairs increases approximately twofold in persons invited to screening, although this is partially offset by the reduction of emergency AAA repairs.[90,119,129] The high mortality rate associated with rupture combined with the observed low elective peri-operative risk[119,135] results in the number of men needed to screen of 667 and to treat with AAA repair of 1.5 to prevent one premature AAA related death.[119] A recent systematic review and meta-analysis pooling all available quantitative and qualitative studies with pre- and post-screening health related QoL (HRQoL) data demonstrated no significant impact on HRQoL from being under surveillance for a screen detected AAA.[136]

There are several limitations in translating the results of these screening trials to contemporary practice. The trials all started in the last century when the prevalence of AAA in men was 4 – 7% in the men screened and most repairs were done by OSR. Today the population prevalence of AAA in 65 year old men has decreased significantly in several European countries and EVAR has become the treatment modality of choice in elective and in emergency repairs. In addition, the incidental AAA detection rate may have increased with more widespread use of diagnostic imaging, and last but not least, life expectancy has increased substantially.

Contemporary evidence from two European countries with national AAA screening programs for older men (UK and Sweden) indicates that screening remains cost effective in these health economies and continues to be so provided the estimated lower AAA prevalence threshold is about

1%;[119,137,138] however, rates of AAA detection have now fallen below 1% in both Sweden and the UK (www.gov.uk). In some countries the AAA prevalence among 65 year old men remains within the range for screening to be highly cost effective.[139]

Furthermore, the optimal age of screening at which most lives are saved and which is cost beneficial has not been assessed formally and with the increasing life expectancy in Europe, screening at older ages might be of benefit.

Therefore, it is justified to revise the strong (Class I) recommendation from the 2019 guideline, which recommended that all 65 year old men should be offered screening. Although the RCTs are partially outdated, they still provide strong evidence that AAA screening of high risk groups is effective. However, the target population may have altered. Therefore, the GWC chooses to issue a more general recommendation on screening of high risk groups with maintained strength and LoE, while refraining from specifying the target population. The definition of a high risk group varies by local (country) conditions, such as prevalence of AAA, life expectancy, and healthcare structure, and this may change over time. Table 6 lists the potential for AAA screening in different risk populations based on AAA prevalence and if available analyses of the effect and benefit.

The dominant risk factor for AAA, apart from male sex and age, is smoking. It has been estimated that 75% of AAA cases are mainly attributable to smoking.[87,99] The USA Preventive Services Task Force has recommended AAA screening for men aged 65 – 75 years who have ever smoked, based on the strength of the association between smoking and AAA rather than evidence from RCTs.[140] With a recommended screening strategy targeting all men aged 65 years there is currently no need for targeting screening based on smoking status. However, in populations with a decreasing prevalence a more selective high risk screening strategy based on smoking status could be a more effective alternative than general screening.

There is limited evidence for screening in women, with the only RCT being underpowered.[130] Hence, based on the lower AAA prevalence in women[95,141] population screening has not been considered.[140] A discrete event simulation model with input parameters specifically for women was employed, and parameter uncertainty addressed by deterministic and probabilistic sensitivity analyses. The base case model adopted the same age at screening (65 years), definition of AAA ($\geq$ 30 mm), surveillance intervals, and AAA diameter for consideration of surgery (55 mm) as for men. The prevalence was low (0.43%) and operative mortality rates about twice that of men. The simulation model showed that the base case and all alternative scenarios (including screening at older ages, definition of AAA as 25 mm, intervention at lower diameter thresholds) resulted in minimal gain in quality adjusted life years and would probably not be cost effective. The authors suggest that population screening of women should not be considered at this time.[142] Canada remains the only country with a recommendation (weak) to screen women who have ever smoked.

Table 6. Potential for abdominal aortic aneurysm screening in different risk populations.

Risk group	Potential for screening	
	Men	Women
65 year old	+	–
65 year old former or current smoker	++	–
Non-white ethnicity	–	–
First degree relative with abdominal aortic aneurysm	+++	+++
Other peripheral aneurysms	+++	+++
Cardiovascular disease	–	–
Organ transplanted	++	++

+ indicates different degrees of suitability for screening and – indicates not suitable for screening.

Importantly, all screening RCTs were conducted in relatively advanced socioeconomic areas predominantly outside the largest cities and in persons of white ethnicity. Ethnicity studies from the UK, have reported a very low prevalence of AAA (0.2%) in subjects of Asian ethnic origin.[143] In the USA, the prevalence is lower in those of African American than in those of white ethnicity.[98] This suggests that those of non-White ethnicity may benefit less from universal screening.

The heritability of AAA has been estimated to be 70%,[101] and there are reports from several countries of an increased incidence of AAA among first degree relatives of patients with AAA.[100] In a Swedish population study, a family history of AAA increased the risk of AAA two fold[144] and in a large Swedish twin registry study there was a 24% probability that a monozygotic twin of a person with AAA will have the disease.[101] Family history of AAA is suggested to be associated with more rapid aneurysm growth and a higher rupture rate[145] and rupture may occur at smaller aneurysm diameter and at lower age.[146] In a health economic model based study evaluating targeting screening for AAA in siblings the absolute risk reduction in AAA deaths was five per 1 000 invited with 27 quality adjusted life years gained per 1 000 invited, and the probability of cost effectiveness was 99%.[147] AAA screening is recommended in all men and women aged 50 years and older with a first degree relative with an AAA.

Because of the high co-existence of AAA with other peripheral aneurysms (iliac, femoral, popliteal),[148,149] patients with peripheral aneurysms are routinely screened for AAA, and vice versa. In a study of 190 patients operated on for popliteal artery aneurysm, 39% developed a new aneurysm during a mean seven year follow up, of which 43% were AAAs.[148]

Some relatively small studies have indicated a high incidence of AAA in patients with other cardiovascular disease: carotid stenosis,[150] coronary heart disease,[151] and peripheral arterial occlusive disease (PAOD).[150] Concomitant AAA screening during US examination for other cardiovascular diseases has been suggested as a feasible strategy for targeted high risk screening.[152] The benefit of

AAA screening in patients with cardiovascular disease has not been assessed formally, and the higher occurrence of the disease among these patients may be counterbalanced by a lower life expectancy and higher operative risk in this subgroup.[153] This was confirmed in a recent UK study on individuals opportunistically screened for AAA during transthoracic echocardiograms or lower limb arterial duplex scans, demonstrating a high prevalence of AAA (7.1%). However, due to a high degree of comorbidity, which limits suitability for repair, and many screening detected AAAs being small with a slow growth rate never reaching threshold for repair, only 3.7% of the screen detected AAAs had been offered repair after median 7.6 years follow up.[154] Thus, evidence is lacking to support this strategy.[2]

The prevalence of AAAs in transplant recipients is reportedly high: 14 — 22% in heart and or lung,[155,156] 30% in liver, and 11% in kidney transplant recipients.[157] In addition, AAAs in transplant patients seem prone to rapid expansion and rupture (11 — 38%), possibly related to the immunosuppression to which the patients are exposed. Thus, in patients who have undergone a solid organ transplant, US screening for AAA is recommended. However, there are no data to suggest when and how often, but this should be determined on an individual basis, based on the organ transplanted and other risk factors.[157]

Recommendation 11		Changed
Ultrasound screening for the early detection of abdominal aortic aneurysm is recommended in high risk populations* to reduce death from aneurysm rupture.		
Class	**Level**	**References** **ToE**
I	A	Lederle *et al.* (2000),[99] Wanhainen *et al.* (2016),[119] Scott *et al.* (1995),[127] Ashton *et al.* (2002),[128] Thompson *et al.* (2009),[129] Lindholt *et al.* (2005),[133] Norman *et al.* (2004),[134] Cosford and Lend (2007),[158] Guirguis-Blake *et al.* (2014)[159]

* What can be considered a high risk group varies based on local conditions, such as disease prevalence, life expectancy, and healthcare structure, see Table 6.

3.4. Incidental detection

Diagnostic imaging used for the investigation of other pathologies including back or chest pain, abdominal and genitourinary symptoms may also detect an AAA. While US and CT scan are most commonly used, there are other imaging modalities including magnetic resonance imaging (MRI), echocardiography, CT colonography, and spinal imaging that may diagnose an AAA.[152,160–164] There is little information about the sensitivity and specificity of these imaging modalities for the diagnosis of AAA. There is also the worrying observation that many of these incidentally diagnosed AAAs are ignored and not referred to vascular surgeons.[142,165,166]

Recommendation 12		Unchanged
Patients with an incidentally detected abdominal aortic aneurysm should be referred to a vascular surgeon for evaluation, except for cases with very limited life expectancy.		
Class	**Level**	**References** **ToE**
I	C	van Walraven *et al.* (2010)[165]

4. MANAGEMENT OF PATIENTS WITH A SMALL ABDOMINAL AORTIC ANEURYSM

This chapter primarily addresses standard fusiform infrarenal AAAs. However, most of the recommendations herein also apply to complex AAAs, unless otherwise stated. Considerations specific to complex AAAs are discussed further in Chapter 8. For specific advice on mycotic (infected), inflammatory, and saccular AAA, pseudoaneurysms and genetic syndromes, see Chapter 10.

4.1. Surveillance of small abdominal aortic aneurysms

By far the most influential study of the natural course of small AAA 30 to 55 mm is the RESCAN study.[106] It assessed individual data collected from 18 different studies from Europe, Canada, the USA, and Australia with patients being included between 1983 and 2008. More than 15 000 individuals with a small AAA and a mean of four years of follow up were included. They estimated a mean AAA growth rate of 2.2 mm/year, independent of age and sex, which increased in smokers by 0.4 mm/year and decreased in patients with diabetes by 0.5 mm/year. Based on these observations, the RESCAN Collaborators suggested a three year surveillance interval for AAAs measuring 30 — 39 mm, yearly for 40 — 49 mm, and every six months for 50 — 54 mm.[106] This has gained wide acceptance and was adopted by the ESVS 2019 AAA guidelines.[2] At that time, a sex neutral surveillance regimen was given, not taking into account that women had a fourfold greater rupture risk, justifying more frequent surveillance.[106]

The safety of the RESCAN surveillance routine has also been demonstrated in the national UK screening program, where the AAA risk of rupture was as low as 0.03% per annum for men with 3.0 — 44 mm AAAs, 0.28% for 45 — 54 mm AAAs, and 0.40% for men with AAAs just below the referral threshold (50 — 54 mm).[117]

AAA prevalence has changed in the last two decade, partly due to a significant reduction in smoking in the population,[167,168] together with improvements of cardiovascular risk management with better BP control and widespread use of statins and antiplatelets,[88,169] resulting in an increased age of patients undergoing repair. At the same time, small AAAs are detected earlier, either incidentally or through population based screening programmes.[90,119] These changes have significantly reduced the detection rate of AAAs in 65 year old men targeted by screening programmes,[87,169] and could potentially also have affected the growth rate of small AAAs.[170–173] This entails uncertainty regarding today's natural history of small AAAs, which could have an impact on surveillance intervals of small AAAs and potentially also on the indication for repair. The GWC therefore commissioned a task force to carry out a systematic review and meta-analysis with the aim of evaluating the contemporary growth rate of small AAAs in view of the recent epidemiological changes. The analysis did not demonstrate any clinically meaningful changed growth rate of small AAAs contemporaneous with the changed AAA epidemiology,[4] suggesting that the RESCAN recommendations are still valid.

In the final follow up of MASS the long term protective effect of screening appeared to decline due to ruptures after eight or more years among men initially screened normal (< 30 mm). Approximately half of these ruptures occurred among those with sub-aneurysmal aortic diameters (25 – 29 mm) at the time of screening.[174] Later cohort studies have demonstrated that most eventually progress to an AAA of which a substantial proportion will reach the diameter threshold for consideration of repair.[138,175–177] In a Swedish population based cohort study including > 1 000 65 year old men with screen detected sub-aneurysmal aortic dilatation, 30% reached the 55 mm diameter within 10 years. The study also showed that a follow up policy with an US examination after five years can safely and effectively identify those sub-aneurysms at risk of becoming an AAA and reaching the diameter threshold for consideration of repair.[177] Although there is only limited evidence regarding the cost effectiveness of surveillance of persons with sub-aneurysmal aortic dilatation,[178,179] current knowledge justifies the recommendation to re-screen men with sub-aneurysmal aortic dimeters with a reasonable life expectancy after five years. Less than 5% of all men screened fall into this category, meaning that this will not require large resources.

In the context of patient specific health, surveillance of small AAAs not expected to reach the diameter threshold for when repair is considered within a reasonable timeframe for the patient to ever be subject to elective repair, or in patients not fit for repair, may not be necessary. Octogenarians with an AAA < 40 mm are significantly less likely than their younger counterparts to ever reach the threshold size for repair, and in the event of AAA growth much less likely to be a candidate for repair,[180] suggesting that surveillance of small AAAs in octogenarians is unlikely to be beneficial. If discontinuation of follow up is considered, the patient should be well informed, and consideration should be given to the patient's wishes. In a recent UK and Dutch study only 8% of conservatively managed patients with an AAA received a palliative care consultation, indicating a need for improvement.[181]

Due to reports of synchronous aneurysms in other vascular beds in patients with AAA, pre-operative screening of the thoraco-abdominal aorta with CTA and the femoropopliteal segment with US is advocated (see section 5.1.1.). Whether such screening should be initiated earlier, already in patients with small AAAs under surveillance, is unclear, and must be determined on a case by case basis.

Recommendation 13			Changed
Men should be considered for imaging surveillance using ultrasound, every five years for a sub-aneurysmal aorta 25 – 29 mm in diameter, every three years for abdominal aortic aneurysms 30 – 39 mm in diameter, annually for aneurysms 40 – 49 mm, and every six months for aneurysms ≥ 50 mm, taking into account life expectancy, suitability for future repair, and patient preferences.			
Class	**Level**	**References**	**ToE**
IIa	B	Prendes *et al.* (2023),[4] Bown *et al* (2013),[106] Svensjö *et al.* (2014),[138] Thompson *et al.* (2012),[174] Oliver-Williams *et al.* (2018),[175] Wild *et al.* (2013),[176] Thorbjornsen *et al.* (2021),[177] Hamel *et al.* (2018),[178] Sogaard *et al.* (2012),[179] Rockley *et al.* (2020)[180]	

Recommendation 14			Changed
Women should be considered for imaging surveillance using ultrasound every five years for a sub-aneurysmal aorta 25 – 29 mm in diameter, every three years for abdominal aortic aneurysms 30 – 39 mm in diameter, annually for aneurysms 40 – 44 mm, and every six months for aneurysms ≥ 45 mm, taking into account life expectancy, suitability for future repair, and patient preferences.			
Class	**Level**	**References**	**ToE**
IIa	C	Bown *et al.* (2013)[106]	

Recommendation 15		New
Patients with small abdominal aortic aneurysms who are either not expected to reach the diameter threshold for repair within their life expectancy, or are unfit for repair, or prefer conservative management, should be considered for discontinuation of surveillance.		
Class	**Level**	**References**
IIa	C	Consensus

4.2. Medical management of patients with small abdominal aortic aneurysms

4.2.1. Cardiovascular risk reduction. Patients with an AAA have a high risk of future cardiovascular events. A systematic review including 21 articles demonstrated a 3% annual risk of cardiovascular death in patients with a small AAA, with a high risk of ischaemic heart disease (IHD) (45%), myocardial infarction (MI) (27%), and stroke (14%).[182] More recently, results from the MASS trial including almost

27 000 men report a 2.2 hazard ratio (HR) of long term cardiovascular death for patients with a small AAA, while the contemporary risk of major cardiovascular events in more than 237 000 men with a small AAA in the English NHS AAA Screening Program (NAAASP) was increased with a HR of 2.9.[183]

A study evaluating medical treatment in more than 12 000 UK patients with a recorded diagnosis of AAA showed that five year survival rates improved significantly for those taking statins (68% *vs*. 42%), antiplatelet therapy (64% *vs*. 40%), or antihypertensive agents (62% *vs*. 39%) compared with patients with an AAA not taking these drugs.[88] More detailed analysis of the antihypertensive agents used indicated that diuretics may be less beneficial than other types.[88] Nevertheless, there is only one RCT evaluating long term effectiveness of antiplatelet, antihypertensive, or lipid lowering medication in cardiovascular event and mortality reduction in 227 patients with an AAA, and specifically evaluated metoprolol *vs*. placebo, without finding any significant results.[184,185]

Thus, it is recommended that all patients with an AAA receive cardiovascular risk factor management; with smoking cessation, BP control, and statin and antiplatelet therapy, as well as lifestyle advice (including exercise and a healthy diet). For specific target values, reference is made to the latest dedicated guidelines on cardiovascular risk reduction.[186] National guidelines may specify which antiplatelet drug, statin or antihypertensive agent(s) are recommended, and if so, these should be consulted.

The 2021 ESC Guidelines on Cardiovascular Disease Prevention in Clinical Practice classify patients with an AAA as having an established atherosclerotic cardiovascular disease with high or very high cardiovascular risk. Intensive risk factor treatment is recommended (Class I Recommendation) including (1) smoking cessation and lifestyle recommendations, including a healthy diet, and exercise; (2) antithrombotic therapy; (3) low density lipoprotein (LDL) cholesterol reduction $\geq$ 50% and $<$ 1.8 mmol/L ($<$ 70 mg/dL) using high intensity statin therapy; and (4) systolic BP $<$ 130 $-$ 140 mmHg. Additional intensified risk factor treatment may be considered, with lower treatment goals (systolic BP $<$ 130 mmHg, LDL cholesterol $<$ 1.4 mmol/L, or $<$ 55 mg/dL, and dual antiplatelet therapy).[186] Despite recommendations for statin and antiplatelet treatment in patients with an AAA, recent studies have drawn attention to the fact that both medications are only prescribed in about 60%, and in whom compliance is as low as 60%.[71,187] Furthermore, over 30% of patients diagnosed with an AAA in an English study continued smoking, despite the evidence that smoking is a key risk factor for AAA prevalence, AAA growth and AAA rupture.[71] The introduction of screening programmes and increased diagnosis of small AAAs provides an opportunity for improved cardiovascular risk prevention in these patients at risk.

Recommendation 16			Changed
All patients with an abdominal aortic aneurysm should receive cardiovascular risk factor management with smoking cessation*, blood pressure control*, statin and antiplatelet therapy*, and lifestyle advice (including exercise and healthy diet).			
Class	**Level**	**References**	**ToE**
I	B	Bahia *et al* (2016),[88] Bath *et al.* (2015),[182] Niebauer *et al.* (2021),[188] Bhak *et al.* (2015),[189] Robertson *et al.* (2017),[190] Wemmelund *et al.* (2014)[191]	

* For details regarding nicotine replacement therapy, specific drug choice, doses, and target values for medical treatment, reference is made to the latest dedicated guidelines on cardiovascular risk reduction.

4.2.2. Strategies to reduce the rate of aneurysm growth and rupture.

Medical management of AAA generally involves cardiovascular risk reduction, including antiplatelet, statin and antihypertensive therapy but does not aim to reduce AAA growth rates.

Several RCTs evaluating different drugs, such as antiplatelet drugs, angiotensin converting enzyme inhibitors, beta blockers, antibiotics, and mast cell inhibitors, have all failed to show any effect on AAA growth (Table 7)[192] and currently there is no specific drug therapy for small AAAs.

The role of statins on AAA growth reduction has not been formally evaluated, with a lack of RCTs looking specifically at statin effect on AAA growth rates. Nevertheless, observational data repeatedly suggest that statins may be associated with a reduction in AAA progression and rupture,[191,206,207] and their effects on cardiovascular mortality reduction have been repeatedly proven, and, therefore, they should be considered in all patients with AAAs. As a consequence, it is impossible to conduct a placebo controlled study to evaluate its possible effect on AAA growth.

Patients with diabetes have a slower AAA growth rate than patients without diabetes, and a number of recent experimental studies and observational data suggest a possible growth inhibitory effect of metformin, used to treat type II diabetes.[208,209] There are several ongoing RCTs evaluating the effects of metformin on AAA growth (ClinicalTrials.gov), but no data have been published.

Observational studies have consistently shown smoking to be associated with increased AAA growth and rupture rates. Smoking cessation appears to be associated with an approximate 20% reduction in growth rate, as well as halving the risk of aneurysm rupture.[189,210] RCTs have shown that smoking cessation is most effective when supported by drugs and counselling.[211] So, besides the positive effect on the general health, especially cardiovascular, smoking cessation also has important AAA specific

Table 7. Summary of randomised controlled trials evaluating medications to slow small aneurysm growth.

Author, year	Study name	Treatment evaluated	Included − *n*	Main findings
Wanhainen *et al.* (2020)[193]	TicAAA trial	Ticagrelor	139 patients, Ticagrelor ($n = 69$)	No difference in MR volume or MR and ultrasound diameter growth rates after 12 months follow up
Baxter *et al.* (2020)[194]	N-TA-3CT	Doxycycline	254 patients, Doxycycline ($n = 129$)	No difference in CT diameter growth rates after two year follow up
Golledge *et al.* (2020)[195]	TEDY trial	Telmisartan	210 patients, Telmisartan ($n = 107$)	No difference in ultrasound or CT diameter growth rates after two year follow up
Pinchbeck *et al.* (2018)[196]	FAME-2 trial	Fenofibrate	140 patients, Fenofibrate ($n = 70$)	No difference in growth rates after 24 week follow up
Kiru *et al.* (2016)[197]	AARDVARK trial	Perindopril and Amlodipine	227 patients, Amlodipine ($n = 72$) Perindopril ($n = 73$)	No difference in ultrasound diameter growth rates after two year follow up
Sillesen *et al.* (2015)[198]	The AORTA trial	Pemirolast	321 patients, 10 mg Pemirolast ($n = 80$) 25 mg Pemirolast ($n = 76$) 40 mg Pemirolast ($n = 84$)	No difference in ultrasound diameter growth rates after 12 month follow up
Meijer *et al.* (2013)[199]	Phast trial	Doxycycline	286 patients, Doxycycline ($n = 144$)	Doxycycline treatment associated with significant increased ultrasound diameter growth rates after 18 month follow up
Høgh *et al.* (2009)[200]		Roxithromycin	84 patients, Roxithromycin ($n = 42$)	No difference in ultrasound measured growth rates after mean 53 month follow up
Karlsson *et al.* (2009)[201]		Azithromycin	247 patients, Azithromycin ($n = 122$)	No difference in CT volume growth rates after 12 month follow up
Propanolol Aneurysm Trial Investigators (2002)[202]	Propranolol Aneurysm trial	Propranolol	548 patients, Propranolol ($n = 276$)	Patients with AAA did not tolerate propranolol well, no differences in ultrasound diameter growth rates after 2.5 year follow up
Mosorin *et al.* (2001)[203]		Doxycycline	34 patients, Doxycycline ($n = 17$)	No difference in ultrasound diameter growth rates after 18 month follow up
Vammen *et al.* (2001)[204]		Roxithromycin	92 patients, Roxithromycin ($n = 43$)	Roxthromycin treatment for four weeks associated with reduced ultrasound diameter growth rates after 1.5 years follow up ($p = .020$)
Lindholt *et al.* (1999)[205]		Propranolol	54 patients	Trial stopped after two years due to significant dropout rate

TicAAA = The efficacy of TICagrelor on AAA Expansion; N-TA(3)CT = Non-Invasive Treatment of Abdominal Aneurysm Clinical trial; TEDY trial = Telmisartan in the Management of AAA trial; FAME-2 trial = Fenofibrate in the Management of AAA 2; AARDVARK trial = Aortic Aneurysmal Regression of Dilation: Value of ACE-Inhibition on RisK trial; The AORTA trial = CRD007 for the Treatment of AAA; Phast trial = Pharmaceutical Aneurysm Stabilisation Trial Study Group; CT = computed tomography.

beneficial effects. Therefore, all patients with a small AAA are recommended to stop smoking and assistance should be provided to do so.

Recommendation 17			Unchanged
Patients with a small abdominal aortic aneurysm are recommended to stop smoking and should receive help to do this, to reduce the abdominal aortic aneurysm growth rate and risk of rupture.			
Class	**Level**	**References**	**ToE**
I	**B**	Sweeting *et al.* (2012),[210] Hartmann-Boyce *et al.* (2022)[211]	

4.2.3. Fluoroquinolone antibiotics in abdominal aortic aneurysm patients. In 2018, the US FDA and the Pharmacovigiliance Risk Assessment Committee (PRAC) of the EMA issued warnings[212,213] concerning an observed association between use of fluoroquinolones and an increased risk of AAA and dissections in the aorta, based on four observational studies.[214–217] All four studies defined the risk of AAA as differences in the number of registered non-specific AAA ICD codes between cohorts exposed to fluoroquinolones *vs.* not, or *vs.* another antibiotic. Collectively, the rate of AAA ICD codes was approximately doubled among those exposed to fluoroquinolones. The risk of significant residual confounding was, however, high in all studies. In particular, ICD codes for asymptomatic and symptomatic AAA disease were combined, while at the same time not controlling for imaging status, resulting in difficulty of differentiating between harmless incidental AAAs and harmful AAAs. Antibiotics used as controls for fluoroquinolones may have been associated with different indications and diagnostic work ups, resulting in potentially different rates of detection of incidental AAAs. Aortic dissection outcomes were presented combined with AAA, despite the fact that that the diseases have different aetiologies and natural histories, and fluoroquinolone exposed and controls had significantly different rates of cardiovascular risk factors. In summary, interpretation of the association between fluoroquinolone exposure and actual harm from AAA in these studies was unclear, despite a conformity in reported elevated risk of AAA.

To date, several additional studies analysing the AAA risk from fluoroquinolone exposure have been published. Some signal increased AAA risk from fluoroquinolone exposure,[218–224] others report no increased risk.[225–230] All are non-randomised, retrospective, and registry based, and most define risk of AAA as detection of any type of ICD code for AAA documented in close proximity to fluoroquinolone exposure not controlling for the most important confounders.

Two studies; an observational cohort study[225] and a nested case–control study,[226] have reported asymptomatic and symptomatic AAA disease outcomes separately, analysed cohorts of similar magnitude and type of infection, controlling for imaging status, using comparator antibiotics with similar indication profiles, as well as controlling for major cardiovascular risk factors, including smoking status. These studies reported no increased risk of AAA from fluoroquinolone exposure. A similar conclusion was reported in a recent combination cohort ($n = 3\,586\,207$) and case crossover ($n = 95\,198$) study. The associations between fluoroquinolone use and increase in risk of hospitalisation with aortic aneurysm or aortic dissection observed in the unadjusted cohort study analyses and relative to non-users in the case crossover study were lost after covariable adjustment and relative to comparator antibiotics (cephalosporin). This finding further supports that the reported associations between fluoroquinolone use and risk of hospitalisation with aortic aneurysm or dissection is due to confounding.[230]

Consequently, there is currently insufficient evidence to support that the presence of an AAA should be weighed into the decision to use fluoroquinolones or not in these patients.

Recommendation 18		New
Having a small abdominal aortic aneurysm is not a contraindication to using Fluoroquinolone antibiotics.		
Class	Level	References ToE
III	B	Gopalakrishnan *et al.* (2020),[225] Dong *et al.* (2020),[226] Brown *et al.* (2023)[230]

4.3. Physical activity and driving

In a RCT, exercise was considered to be safe in patients with small AAAs, and training for up to three years did not influence rate of AAA enlargement.[188,231] Moderate to high intensity interval exercise training was shown to be safe in patients with large AAAs ($\leq$ 70 mm) awaiting surgical repair, assuming strict adherence to safety guidelines (systolic BP $<$ 180 mmHg and or heart rate $<$ 95% of the maximum).[232] Thus, there are no data suggesting that exercise may be harmful to patients with small AAAs. On the contrary, it is important to acknowledge the positive effect of exercise on the general health of patients with small AAAs who have cardiovascular comorbidity.[233] Hence, it is not advisable to discourage these activities. Furthermore, retrospective single centre studies have shown that both spirometry based pulmonary function tests, cardiopulmonary exercise testing and dobutamine stress echocardiography can be performed safely in patients scheduled for AAA repair.[234,235] However, given the small number of large AAAs in these studies, it is not possible to draw any firm conclusions about the safety of patients with an aneurysm diameter $>$ 70 mm. Available information does not allow giving well founded advice on specific sports activities. Case reports have associated strength sports, such as heavy weightlifting, with aortic dissection, which is thought to be caused by the Valsalva manoeuvre resulting in an acute BP spike. Although the equivalent has not been reported for rupture of AAA, restraint with such vigorous sporting activities may be advisable for patients with a large AAA.

Likewise, it is important to point out the lack of evidence that sexual activity might be dangerous for patients with small AAAs.

A recent literature review found no available scientific literature regarding suitability to drive for patients with AAA.[236] The only available information is legislation in transport agency guidelines, which, however, consistently lack information about the basis for the recommendations given. There are discrepancies in the legislation regarding patient fitness to drive based on aneurysm size between countries, with some being more conservative in restricting patient ability to drive once an aneurysm is deemed borderline. For example, in New Zealand, Australia, Spain, and Germany, patients lose the ability to drive once an aneurysm reaches 55 mm whereas in the UK patients can drive until the aneurysm diameter reaches 65 mm, and in Canada, the AAA diameter threshold is based on sex (65 mm for men and 60 mm for women).[236] In Sweden a specific threshold of 55 mm is used only for professional drivers of heavy vehicles, such as buses and trucks, while revocation of driving licenses for cars, including taxis, and motorcycles is indicated in the case of considerable risk of sudden rupture without a specified threshold diameter (www.transportsyrelsen.se). In some countries, such as the UK, continued surveillance is warranted regardless of the patient's fitness for repair in order to assess suitability to continue driving. Due to the lack of evidence and inconsistent legislation between countries, the GWC refrains from issuing a recommendation on driving in patients with AAA but refers to local regulations.

Recommendation 19		New
Restricting exercise or sexual activity in patients with small abdominal aortic aneurysms* is not indicated.		
Class	Level	References ToE
III	B	Niebauer *et al.* (2021),[188] Myers *et al.* (2014)[231]

* Here defined as $<$ 70 mm diameter.

4.4. Indications for elective repair

The immediate decision about the size at which an aneurysm should be repaired is based on the balance between aneurysm rupture risk (which is fatal in > 80% cases)[237] and operative mortality risk of aneurysm repair. Today, with increased life expectancy, it also is necessary to consider the long term prognosis, including durability, surveillance, life expectancy, and the QoL after AAA repair. Furthermore, the patient's preference is of course key in the decision making (see Chapter 11).

The management of fusiform, degenerative aneurysms 40 − 55 mm in diameter has been effectively determined by four RCTs including two large multicentre RCTs of early open elective surgery *vs*. surveillance, the UK Small Aneurysm Trial (UKSAT)[238−241] and the American Aneurysm Detection And Management study (ADAM),[242] and two smaller trials of endovascular repair *vs*. surveillance, the Comparison of surveillance *vs*. Aortic Endografting for Small Aneurysm Repair (CAESAR) trial[240] and the Positive Impact of endoVascular Options for Treating Aneurysm early (PIVOTAL) study.[241] The consensus from these trials is that aneurysms < 55 mm in diameter should be managed conservatively, and has been summarised in a Cochrane review, showing that surveillance was safe in men.[243] This has been confirmed to be safe for men in two national screening programmes in England and Sweden.[90,117,119]

Despite this high quality evidence, AAAs in men are still repaired below the 55 mm diameter threshold in several countries particularly those with privately funded healthcare, many of these repairs breaching quality standards.[15,244] An administrative registry based analysis showed a significantly lower population aneurysm related mortality in the USA, where more than 40% of repairs were performed on small AAAs < 55 mm, as opposed to the UK, where the small AAA repair rate was less than 10%.[245] This paper has, however, been questioned for reasons relating to incidental detection rates, differences in coding systems, population structure, and total healthcare expenditure, as well as the indications for surgery and impact of population screening.[246−248]

Another topic of debate is which imaging modality and methodology should be used for decisions about repair. It is known that CTA measurements provide a larger diameter than US,[125,126] and it is common to perform a CTA at a diameter just below the diameter threshold at which repair is considered as measured by US, thereby probably obtaining a larger diameter which may unnecessarily warrant repair.

The risk of rupture of AAAs < 55 mm is very low in men and ranges between 0.3 and 0.8% per year.[238−241] The modality and methodology of measurement of the maximum diameter varies however and are sometimes not clearly defined (see also Chapter 3). The UKSAT used maximum AP diameter with US (unknown calliper placement) and the other trials used CTA; ADAM and CAESAR used maximum diameter centreline in any plane, while the PIVOTAL did not specify. RESCAN was an individual patient data meta-analysis and estimated the risk of rupture to be 0.6% per year for men until a diameter of 55 mm.[106] While absolute growth rates were similar for women and men there were marked differences in the absolute risks of rupture. Women had a fourfold greater rupture risk for all AAA sizes and reached a rupture risk of greater than 1% in a much shorter time than men. In a population based screening cohort study, the annual rupture rate for AAAs up to 60 mm was 0.8%.[249] Decisive data come from the NAAASP in the UK.[116] Screening units use US for surveillance and use the ITI AP diameter.[116,117] Rupture rates in men were 0.4% per year for diameters between 50 and 54 mm (which translates to CT diameters between 55 and 59 mm). Studies on rupture risk of small AAAs are displayed in Table 8.

Multiple papers have reported the mean AAA diameter at the time of rupture, which vary between 75 − 80 mm for men and 67 mm for women.[250−252] About 8 − 10% of rAAA operations are done for aneurysms with a diameter < 55 mm. This has been put forward as an argument for lowering the current diameter threshold at which repair is considered. This is, however, a misguided conclusion. Despite small AAA having a very low risk of rupture, their sheer numbers in the population (due to the normal distribution of aortic diameter) make them a sizeable proportion of all operations for rAAA. This is further underlined by a VASCUNET study, showing that the average diameter at repair varies between countries, but this does not translate to a reduced number of operations for rAAA.[253]

A recent prospective surveillance registry study, including 332 patients with large AAAs undergoing delayed repair for more than one year and 1 033 patients with large AAAs not undergoing repair, most often due to patient preference or comorbidity, reported a three year cumulative incidence of rupture of 3.4% for initial AAA size 50 − 54 mm (women only), 2.2% for 55 − 60 mm, 6.0% for 61 − 70 mm, and 18.4% for > 70 mm. Women with AAA size 61 − 70 m had a three year cumulative incidence of rupture of 12.8% compared with 4.5% in men ($p = .002$).[76]

In conclusion, in men the risk of rupture is very low (0.3 − 0.8% per year) for AAAs with a diameter below 55 mm measured with US, which translates to a diameter on CTA between 55 − 62 mm depending on which measurement methodology is used. Therefore, there is no need to lower the diameter threshold for repair or to perform a CTA when US measures the AAA diameter < 55 mm in men. On the contrary, based on the NAAASP data it has been suggested to raise the diameter threshold to 60 mm when based on CTA.[254] Although it is possible that the threshold should be raised in the future, the GWC does not believe there is sufficient support at this time. Nevertheless, the GWC has chosen to issue a new strong negative recommendation of elective repair of AAA < 55 mm, and to downgrade the recommendation on the threshold for considering repair in men (from Class I and LoE A to Class IIa and LoE C) due to the fact that the RCTs underlying this recommendation only showed that it is not worthwhile operating on AAAs < 55 mm, which only indirectly suggests that elective repair should be considered in AAAs larger than that, i.e., 55 mm. To further emphasize the lack of an evidence based distinct diameter limit for when elective repair should take place, we refrain from labelling it a threshold in the revised recommendation.

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Table 8. Studies on rupture risk of small abdominal aortic aneurysms.

Study	Recruitment	Modality	Measurement	Threshold	Ruptures
ADAM[242]	1992–2000	CTA	The diameter of the aneurysm was defined as the maximum external cross sectional measurement in any plane but perpendicular to any bend in the vessel	55 mm	0.6%/year
UKSAT[239,255]	1991–1998	US	Maximum anteroposterior diameter	55 mm	0.6%/year
CEASAR[240]	2004–2008	CTA	Diameter of the aneurysm was defined on computed tomography scan at the maximum external cross sectional measurement in any plane but perpendicular to the vessel axis	55 mm	2/178 (1.1%) after 24 and 52 months of follow up
PIVOTAL[241]	Not specified	CTA	infrarenal AAAs between 4.0 and 50 mm in diameter by computed tomography	55 mm	0.3%/20 months
NAAASP[116]	2009–2017	US	Inner to inner maximum anteroposterior diameter	55 mm	0.03% per annum (95% CI 0.02–0.05%) for men with small AAAs 0.28% (0.17–0.44%) for medium AAAs 0.40% (0.22–0.73%) for men with AAAs just below the referral diameter threshold (50–54 mm)
Scott[256]	1988–1995	US	The maximum aortic diameters in both the transverse and anteroposterior planes were recorded	60 mm	0.8%/year
RESCAN[106]	IPD meta-analysis	US	Inner to inner and outer to outer diameter	55 mm	0.64%/year at 50 mm (men) 2.97%/year at 50 mm (women)

CTA = computed tomographic angiography; US = ultrasound; IPD = individual participant data; ADAM = the American Aneurysm Detection And Management study; UKSAT = the UK Small Aneurysm Trial; CEASAR = the Comparison of surveillance vs. Aortic Endografting for Small Aneurysm Repair trial; PIVOTAL = the Positive Impact of endoVascular Options for Treating Aneurysm early; NAAASP = National Abdominal Aortic Aneurysm Screening Programme.

There is anecdotal evidence that rapid aneurysm growth (> 10 mm/year) is associated with a greater risk of rupture. Instances of presumed rapid aneurysm growth may be related to measurement errors and the first action should be to re-measure the aneurysm diameter.[257–259] In a prospective cohort study most small AAAs showed a linear growth, while non-linear growth patterns (staccato or exponential) were infrequent when a core laboratory was used to report AAA diameter. No patients with a baseline AAA diameter less than 42 mm exceeded the diameter thresholds at which repair is considered within two years, suggesting that continuing imaging follow up is safe regardless of growth pattern.[258]

The risk of rupture for a small AAA is about four times higher in women than men.[106,210,260] In the RESCAN meta-analysis the rupture rate for women with a 42 mm AAA was approximately the same as that of a man with a 55 mm AAA, suggesting a diameter threshold at which surgery is considered of 45 mm may be appropriate in women.[106,261] On the other hand, the operative mortality is higher in women than men for endovascular and open repair.[262–264] Therefore, good evidence about the diameter threshold for repair in women is lacking, but it may be prudent to consider aneurysm repair at lower diameters, closer to 50 mm measured with US.

Although the 55 mm limit continues to create debate and compliance varies, the evidence is convincing not to operate on AAAs < 55 mm in men, and it has been accepted that the diameter threshold for considering repair in women should be lowered.[243,263] Patient information on the safety of surveillance of small AAAs may improve adherence to this recommendation.

Recommendation 20			New
Men with an asymptomatic abdominal aortic aneurysm < 55 mm are not recommended for elective repair.			
Class	Level	References	ToE
III	A	Lederle *et al.* (2002),[238] Powell *et al.* (2007),[239] Cao *et al.* (2011),[240] Ouriel *et al.* (2010)[241]	

Recommendation 21		New
Women with an asymptomatic abdominal aortic aneurysm < 50 mm are not recommended for elective repair.		
Class	Level	References
III	C	Consensus

Recommendation 22			Changed
Men with an abdominal aortic aneurysm ≥ 55 mm should be considered for elective repair.			
Class	Level	References	ToE
IIa	C	Oliver-Williams *et al.* (2019),[117] Filardo *et al.* (2015)[265]	

Recommendation 23			Changed
Women with an abdominal aortic aneurysm ≥ 50 mm may be considered for elective repair.			
Class	Level	References	ToE
IIb	C	Bown *et al.* (2013),[106] Sweeting *et al.* (2012),[210] Brown and Powell (1999),[260] Grootenboer *et al.* (2010),[262] Ulug *et al.* (2017)[264]	

Recommendation 24			Unchanged
Patients with small abdominal aortic aneurysms showing rapid growth (≥ 10 mm/year) should be considered for re-measurement of the aneurysm diameter as the first measure.			
Class	Level	References	ToE
IIa	C	Kurvers *et al.* (2004),[257] Sharp *et al.* (2003),[259] Olson *et al.* (2022)[266]	

There are a significant number of patients with AAA who are not considered suitable for repair (including EVAR) because of comorbidities or limited life expectancy.[90,135,264,267] Among 3 026 men referred for possible intervention within the NAAASP, 8% were declined repair for medical reasons.[135] There has been only one RCT to assess whether EVAR provided a survival benefit for patients too physically compromised to undergo OSR, the EVAR 2 trial.[268] This trial showed that in these physically frail patients although EVAR prevented death from aneurysm rupture, operative mortality was high (7%) and it did not offer any benefit in terms of overall survival up to 12 years, with two thirds of both randomised groups being dead within five years.[269,270]

However, there is likely to be a sliding scale for assessing fitness for repair as the aneurysm enlarges, with lower barriers for fitness in abdominal aneurysms $>$ 70 mm in diameter. Therefore, patients should be kept under surveillance and referred to other relevant specialities to optimise their physical fitness.

In these patients with a high burden of cardiovascular comorbidities, strategies to reduce cardiovascular risk are crucial especially since data suggest that statins may reduce the risk of rupture of small and large AAA[191,206,271] and that the risk of rupture is increased two fold in current smokers.[210]

Recommendation 25			Changed
Patients with an abdominal aortic aneurysm who have reached the diameter threshold for repair, but who initially are deemed unfit for repair should be considered for continued surveillance, referral to other specialists for optimisation of their fitness status and then reassessment.			
Class	Level	References	ToE
IIa	C	Salata *et al.* (2018),[206] Greenhalgh *et al.* (2010),[268] Sweeting *et al.* (2017)[270]	

5. ELECTIVE ABDOMINAL AORTIC ANEURYSM REPAIR

This chapter focuses on non-ruptured infrarenal AAAs for cases that are amenable to elective treatment by a standard, commercially available stent graft, or by OSR using an infrarenal aortic clamp in elective circumstances. For ruptured and symptomatic non-ruptured AAA see Chapter 6, and for juxta- and suprarenal AAAs Chapter 8.

5.1. Pre-operative management

5.1.1. Vascular anatomy assessment. Dedicated aortic imaging is crucial to determine an appropriate repair strategy and to optimally plan pre-operatively. As the presence of synchronous aneurysms in other vascular beds may influence surgical decision making, screening of the entire aorta with CTA and the femoropopliteal segment with US is advocated.[149]

The feasibility of EVAR and its early and long term success depend on reliable baseline assessment of aortic morphology including landing zones for fixation and sealing, and correct measurements for appropriate stent graft selection (Table 9).[272] Several criteria have been established that define patient suitability for EVAR according to the IFU defined by the device manufacturers.[273]

Although there is no RCT on the best imaging modality, the consensus is that CTA with thin slices ($\leq$1 mm), including multiplanar and curved 3D vascular reconstructions is the preferred pre-operative imaging modality.[274]

Recommendation 26		New
Prior to abdominal aortic aneurysm repair, routine screening imaging of the entire aorta, access and femoropopliteal arteries should be considered.		
Class	Level	Reference
IIa	C	Consensus

Recommendation 27		New
Prior to endovascular abdominal aortic repair, detailed pre-operative procedure planning with computer tomography angiography, including the use of a dedicated post-processing software analysis, should be considered.		
Class	Level	References
IIa	C	Consensus

5.1.2. Operative risk assessment and optimisation. In a Cochrane Review, operative 30 day or in hospital mortality with EVAR was lower than with OSR in patients fit for surgery (1.4% *vs.* 4.2%, OR 0.33).[275] Mortality risk was higher in women, compared with men, for OSR (OR 1.49) and more so for EVAR (OR 1.86).[263] The ESC guidelines grade OSR as a high risk intervention (defined as carrying a risk of cardiovascular death or MI of 5% or more within 30 days), whereas EVAR is graded as an intermediate risk

Table 9. Imaging evaluation for planning of infrarenal abdominal aortic aneurysm repair.

1. Proximal neck to be cross clamped or used as landing zone, including diameter and length, angulation, shape, presence, and extent of calcification and atherothrombosis
2. Iliac arteries to be cross clamped or used for access and landing zone, including: patency; diameter and length; angulation and tortuosity; extent of calcification and atherothrombosis; patency of internal iliac arteries and pelvic circulation; presence of iliac artery aneurysms
3. Access vessel and lower limb run off vessels and circulation
4. Anatomy and patency of visceral arteries and presence of accessory renal arteries
5. Concomitant aneurysms in visceral arteries or thoracic aorta
6. Presence of shaggy aorta (extensive atheromatous degeneration of the aorta with irregular parietal thrombi and ulcerated plaques, which can potentially lead to athero-embolic events
7. Other: Venous anomalies, including position and patency of the inferior caval and left renal vein; organ position, including pelvic or horseshoe kidney; signs of concomitant disease potentially altering prognosis and, thereby, indication for repair

intervention with a cardiac risk between 1% and 5%.[276] This section provides a broad overview of the relevant factors that should be taken into account in the pre-operative evaluation of patients undergoing aortic repair.[277]

There is extensive guidance on operative risk assessment and reduction which should be consulted for in depth information.[276–283]

As a minimum, all patients should undergo a medical history and clinical examination, functional assessment, full blood count, electrolytes and renal function, and electrocardiogram. Additional testing depends upon the individual circumstances of the patient as described below.

A UK RCT has shown that a period of pre-operative supervised exercise training is beneficial to patients undergoing open or endovascular aortic surgery by reducing cardiac, respiratory and renal complications post-operatively, as well as reducing the length of hospital stay.[284] Furthermore a contemporary study of a 24 week community exercise programme RCT demonstrated improved cardiopulmonary exercise testing parameters for those randomised to exercise.[285] However a contemporary Cochrane review concluded that due to low certainty evidence pre-habilitation might slightly reduce cardiac and renal complications compared with standard care but uncertainty remained about the impact on 30 day mortality, pulmonary complications, the need for re-intervention, or post-operative bleeding.[286]

Smoking cessation prior to both EVAR and OSR has been demonstrated to reduce respiratory complications and one year mortality rate in a recent large series reported from the Vascular Quality Initiative of Society for Vascular Surgery (SVS-VQI)[287] (please also see section 4.2.1 Recommendation 16 and section 4.2.2 Recommendation 18).

5.1.2.1. Assessment and management of cardiac risk. Cardiac complications are estimated to cause up to 42% of peri-operative deaths after non-cardiac surgery[288] and the level of cardiac risk should be assessed clinically.[289]

In cases with active cardiovascular disease, such as unstable angina, decompensated heart failure, severe valvular disease, and significant dysrhythmia, further specialist assessment and management are required before AAA repair planning.

In the absence of active cardiovascular disease, clinical cardiovascular risk factors (Table 10) and the patient's functional capacity (Table 11) should be assessed.[290–292] Functional capacity is estimated by the patient's ability to perform activities of daily living, assessed by metabolic equivalent (MET), which is estimated as the rate of energy expenditure while sitting at rest. By convention 1 MET corresponds to 3.5 mL O_2/kg/min.[293]

Patients capable of moderate physical activities (Table 11), such as climbing two flights of stairs or running a short distance (MET $\geq$ 4), will not benefit from further testing. Patients with poor functional capacity (MET $<$ 4) and or with significant clinical risk factors should be referred to a specialist for cardiac work up prior to AAA repair. Although poor capacity alone is only weakly associated with impaired outcomes after aortic repair,[296] cardiac prognosis is good if functional capacity is high, even in the presence of stable IHD or other risk factors.[297] Cardiac work up includes non-invasive evaluation of left ventricular dysfunction, heart valve abnormalities and stress induced myocardial

Table 10. Risk factors for cardiac, respiratory, and renal complications after abdominal aortic aneurysm repair according to Gupta *et al.*, Lee *et al.*, and Inagaki *et al.*[291,292,294]

Predictors of cardiac complications	Predictors of pulmonary complications	Predictors of renal complications
Age	Age $\geq$ 60 year	Pre-existing renal insufficiency
History of symptomatic ischaemic heart disease	Pre-existing chronic obstructive lung disease	Congestive heart disease
History of congestive heart failure	Congestive heart failure	Chronic obstructive lung disease
History of symptomatic cerebrovascular disease	Serum albumin level $\leq$ 3.5 g/dL	Peripheral arterial occlusive disease
Creatinine clearance $<$ 60 mL/min or serum creatinine $>$ 170 μmol/L	FEV1 $<$ 70% of expected	Diabetes mellitus
Diabetes mellitus	FVC $<$ 70% of expected	Arterial hypertension
Functional status in terms of independent living	FEV1/FVC $<$ 0.65	
American Society of Anaesthesiology Class 3/4		

FEV1 = forced expiratory volume in one second; FVC = forced vital capacity.

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Table 11. Functional capacity estimation based on physical activity, according to Ainsworth *et al.*[295]	
Activity level	**Example of activity**
Poor (MET < 4)	Eating, getting dressed, light housework (washing dishes, cooking, making bed)
Moderate (MET 4–7)	Climbing two flights of stairs, walking up a hill, jogging < 10 minutes, heavy housework (scrubbing floor or moving furniture), hand mowing lawn, shovelling snow by hand
Good (MET 7–10)	Tennis, bicycling in moderate pace, leisure swimming, jogging > 10 minutes
Excellent (MET > 10)	Strenuous sports such as uphill mountain bicycling, football, basketball, karate, running 10 km/h or more

MET = metabolic equivalent.

ischaemia. Invasive coronary angiography, by contrast, should follow the same indications as in a non-surgical setting and not be used routinely for peri-operative risk assessment before aortic surgery.[276]

Biomarkers (e.g., troponin T and B type natriuretic peptide) should not be used routinely in pre-operative risk stratification, but may be considered selectively in high risk patients,[276] for example with poor functional capacity or suspected relevant IHD.

Two RCTs have demonstrated that patients with stable coronary artery disease do not benefit from prophylactic revascularisation before vascular surgery,[298,299] even considering those with left main stem and triple vessel disease, or those with a left ventricular ejection fraction below 35%. Therefore, pre-operative coronary revascularisation should not be performed prophylactically but be reserved for patients with unstable coronary artery disease, acute MI, or those considered with a prohibitive coronary risk of AAA repair.[276,280,298]

For patients undergoing interventional coronary revascularisation before AAA repair, the risk of in stent thrombosis is highest during the first six weeks after coronary stenting, and dual antiplatelet therapy should not be discontinued during this period of time. If bare metal stents have been used, reduction to antiplatelet monotherapy may be considered after six weeks. In contrast, if drug eluting stents have been used, dual antiplatelet therapy should not be discontinued for 3 – 12 months depending on the specific drug eluting stents used.[300] Therefore, elective AAA repair should usually be delayed if possible if dual antiplatelet therapy needs to be stopped for surgery. Alternatively, EVAR may be performed under dual antiplatelet therapy if AAA repair cannot be postponed.

Patients with heart failure (New York Heart Association Functional Classes III and IV: marked activity limitation due to symptoms, and severe symptoms at rest respectively) should be optimised pharmacologically under expert guidance. Elective aortic repair should be deferred whenever possible until heart failure has been assessed and treated appropriately. A careful multidisciplinary meeting should evaluate the risk benefit of treatment for each patient individually.[301]

Aortic valve stenosis is the most relevant valvular heart disease in the context of AAA repair, because it increases the risk associated with blood loss, volume shifts, and dysrhythmia. Patients with severe aortic valve stenosis (defined as mean gradient > 40 mmHg, valve area < 1 cm^2, and peak jet velocity > 4.0 m/s) should be considered for aortic valve replacement prior to elective AAA repair.[276,280,298,302] Transfemoral transcatheter aortic valve implantation can be performed simultaneously or sequentially,[303] but the optimal approach and timing of transcatheter aortic valve implantation is largely unexplored and must be determined on a case by case basis.

Applicable guidelines should be consulted for specific guidance on peri-operative management of patients with coronary, congestive, and valvular heart disease.[276,280,304]

Recommendation 28		Unchanged
Routine referral for pre-operative cardiac work up, coronary angiography, cardiopulmonary exercise testing, and routine coronary revascularisation in patients with stable coronary artery disease, is not indicated prior to abdominal aortic aneurysm repair.		
Class	**Level**	**Reference**
III	C	Consensus

Recommendation 29		Unchanged
Patients with poor functional capacity (defined as metabolic equivalents < 4) or with significant clinical risk factors (unstable angina, decompensated heart failure, severe valvular disease, and significant dysrhythmias), are recommended to be referred for cardiac work up and optimisation prior to elective abdominal aortic aneurysm repair.		
Class	**Level**	**References**
I	C	Consensus

Recommendation 30		Unchanged
For patients on dual antiplatelet therapy after percutaneous coronary intervention, delaying abdominal aortic aneurysm repair until after reduction to monotherapy may be considered. Conversely, performing endovascular aortic repair under dual antiplatelet therapy may be considered if repair cannot be postponed.		
Class	**Level**	**References**
IIb	C	Consensus

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5.1.2.2. Assessment and management of pulmonary risk. Pulmonary complications including atelectasis, pneumonia, respiratory failure, and exacerbation of underlying chronic lung disease may increase peri-operative morbidity and length of hospital stay to a similar extent as cardiac complications in patients after non-cardiac major surgery. Risk assessment strategies have been published previously[281,282] and certain risk factors indicate patients at risk (Table 10).

Pulmonary function testing with spirometry has not been shown to be superior to clinical evaluation in predicting post-operative pulmonary complications. Therefore, routine pulmonary function testing with spirometry is not recommended, but should be reserved for patients at risk of pulmonary complications.[281]

Routine chest Xray prior to AAA repair is redundant since CT of the entire aorta (including the chest) has usually been done and does not improve the pre-operative risk stratification and is not recommended.

In patients with suspected compromised respiratory function on clinical evaluation, respiratory work up and optimisation is recommended prior to AAA repair.

Smoking cessation should be encouraged in every AAA patient (see Chapter 4).

Recommendation 31			**Changed**
Routine pulmonary function testing with spirometry or chest Xray prior to elective abdominal aortic aneurysm repair is not indicated.			
Class	Level	References	ToE
III	C	Smetana *et al.* (2006)[282]	

Recommendation 32			**Unchanged**
Patients with risk factors for pulmonary complications or a recent decline in respiratory function should be referred for respiratory work up and optimisation prior to elective abdominal aortic aneurysm repair.			
Class	Level	References	ToE
I	C	Boden *et al.* (2018)[305]	

5.1.2.3. Assessment and optimisation of kidney function. Post-operative impairment of kidney function is a known predictor of increased morbidity and long term mortality,[278,306] and patients with pre-existing renal insufficiency, congestive heart disease, chronic obstructive pulmonary disease (COPD), PAOD, diabetes mellitus, or arterial hypertension are at particular risk[307,308] (Table 10). In the context of open or endovascular AAA repair pre-existing renal dysfunction is one of the most important predictors of peri-operative morbidity and mortality.[309,310]

Patients undergoing AAA repair should have their serum creatinine measured to assess pre-operative kidney function (i.e., estimated glomerular filtration rate (eGFR) according to the Modification of Diet in Renal Disease Study Group or Cockcroft and Gault formula). Although there are no established criteria about the level of renal dysfunction that requires referral to specialist renal services, an eGFR below < 60 mL/min/1.73 m^2 has been regarded as renal compromise, and < 30 mL/min/1.73 m^2 to be severe and therefore warrant urgent referral.

Patients with severe renal insufficiency (i.e., Chronic kidney disease Stages 4 or 5; eGFR < 30 mL/min/1.73 m^2) should be evaluated by a nephrologist to optimise the renal function before elective aortic repair. Recent data from the VQI database suggests that in patients with chronic kidney disease Stage 5, elective EVAR may need to be reserved for AAAs $\geq$ 70 mm unless there are other concerning anatomical characteristics demonstrated, due to a higher than expected one year mortality rate.[311] Patients with mild to moderate renal failure (i.e., chronic kidney disease Stages 2 or 3; eGFR < 60 but > 30 mL/min/1.73 m^2) should be adequately hydrated before AAA repair, especially when intra-arterial contrast media will be used.[310] Currently, no clear effective strategies besides appropriate hydration to prevent post-operative acute kidney injury after AAA repair have been demonstrated.[312] Hence, urine output should always be monitored peri-operatively.

Recommendation 33			**Unchanged**
Assessment of pre-operative kidney function by measuring serum creatinine and estimating glomerular filtration rate is recommended prior to elective abdominal aortic aneurysm repair, with referral to a nephrologist in case of severe renal impairment (estimated glomerular filtration rate < 30 mL/min/1.73 m^2).			
Class	Level	References	ToE
I	C	Castagno *et al.* (2016),[309] Saratzis *et al.* (2016)[310]	

Recommendation 34		**Unchanged**
Patients with renal impairment should be adequately hydrated before elective abdominal aortic aneurysm repair.		
Class	Level	References
I	C	Consensus

5.1.2.4. Assessment and optimisation of nutritional status. Nutritional status is an important determinant of peri-operative mortality and morbidity. In an observational analysis of 15 000 patients undergoing AAA repair, 30 day mortality and incidence of re-operations and pulmonary complications increased with hypoalbuminaemia after both open (n = 4 956) and endovascular (n = 10 046) AAA repair.[294] Therefore, nutritional status should be assessed before aortic surgery for risk stratification. An albumin level of < 2.8 g/dL should be considered as severe malnutrition and is associated with significantly worse outcomes.[294] In this situation, nutritional deficiencies should be corrected before elective OSR and elective EVAR, even though efficacy has not been assessed by RCT in patients with AAA. Referral to a medical dietician may be advisable and should be considered depending on the degree and quality of nutritional deficiency.

Recommendation 35			Changed
Assessment of pre-operative nutritional status by measuring serum albumin should be considered prior to elective abdominal aortic aneurysm repair, with an albumin level of < 2.8 g/dL as the threshold for pre-operative correction.			
Class	**Level**	**References**	**ToE**
IIa	**C**	Inagaki *et al.* (2017)[294]	

5.1.2.5. Carotid artery assessment. Among more than 15 000 patients operated on for AAA in the US National Quality Improvement Program database the peri-operative stroke risk was 0.8% after OSR and 0.5% after EVAR.[313] The prevalence of internal carotid artery stenosis is high among patients with AAA because of similar risk factors. In the Second Manifestations of ARTerial Disease (SMART) study 8.8% of all patients with AAA had an asymptomatic internal carotid artery stenosis of at least 70%.[314] There is, however, no association between asymptomatic carotid artery stenosis and peri-operative stroke after non-cardiac surgery.[315]

Thus, current evidence does not support routine pre-operative screening[315,316] or routine carotid intervention for asymptomatic carotid stenosis prior to AAA repair,[317] which is in agreement with the ESVS 2023 Carotid guidelines recommending against (Class III) routine carotid screening in (neurologically) asymptomatic patients, or prophylactic carotid endarterectomy or carotid stenting prior to major non-cardiac surgery in patients with asymptomatic 50 – 99% carotid artery stenosis.[318]

In a large Danish nationwide cohort study in patients with a history of stroke undergoing elective, non-cardiac surgery, the rate of peri-operative stroke was 11.9% if operations were performed within three months of the stroke, declining to 4.5% three to six months after the stroke and 1.8% six to 12 months after the event *vs*. 0.1% in patients with no history of stroke.[319] Thus, patients with recently symptomatic internal carotid artery stenosis (less than six months) may require appropriate management of the carotid artery stenosis prior to AAA repair to reduce overall stroke risk, which is consistent with the ESVS 2023 Carotid guidelines where a strong (Class I) recommendation to perform carotid revascularisation prior to elective non-cardiac surgical procedures was issued.[318]

Recommendation 36			Changed
Routine screening for asymptomatic carotid stenosis and routine prophylactic carotid intervention for asymptomatic carotid artery stenosis prior to abdominal aortic aneurysm repair is not indicated.			
Class	**Level**	**References**	**ToE**
III	**B**	Sharifpour *et al.* (2013),[313] Sonny *et al.* (2014),[315] Axelrod *et al.* (2004),[316] Ballotta *et al.* (2005)[317]	

Recommendation 37			Changed
For patients with an abdominal aortic aneurysm and a concomitant symptomatic (within the last six months) 50 – 99% carotid stenosis, carotid intervention before elective abdominal aortic aneurysm repair is recommended.			
Class	**Level**	**References**	**ToE**
I	**B**	Jorgensen *et al.* (2014),[319] Rothwell *et al.* (2003)[320]	

5.1.2.6. Assessment of frailty and sarcopenia. Frailty is defined as decreased reserve and resistance to stressors due to cumulative declines across multiple physiological systems. In a systematic review, including 22 cohort studies and one RCT, overall frailty, assessed as functional status, was found to be associated with a significantly increased 30 day mortality risk after AAA repair (OR 5.1), while central muscle mass predicted long term all cause mortality after AAA repair (HR 2.1).[321] Sarcopenia is defined as the progressive and widespread loss of skeletal muscle mass and muscle function, often measured as psoas muscle mass. In a systematic review and meta-analysis, including 1 440 patients from seven observational cohorts, a significant link was found between sarcopenia and death after AAA repair (HR 1.7) and a subgroup analysis including only patients who underwent EVAR showed a marginal survival benefit for patients without low skeletal muscle mass (HR 1.9).[322] Whether the use of frailty score or measurement of sarcopenia adds anything beyond already established risk assessments, such as functional status and cardiovascular status, has however not yet been confirmed and more evidence is required before these tools can be used in the decision making process.[322]

5.2. Peri-operative management

5.2.1. Peri-operative best medical treatment. RCTs on newly initiated beta blockers within 24 hours of vascular surgery either demonstrated no advantage in low risk patients,[184,323] or showed increased all cause mortality, hypotension, and stroke, despite reduced rates of peri-operative MI.[324] A recent Cochrane review on pharmacological treatment of patients with AAA did not identify any new data on beta blockers and suggested the quality of evidence was insufficient to draw robust conclusions.[190] In a meta-analysis, including 32 000 patients from three RCTs, five retrospective cohort studies, and three prospective cohort studies, beta blockers did not improve peri-operative outcomes in vascular and endovascular surgery.[325] Patients who already take an appropriate dose of beta blockers should continue this treatment.

Multiple observational studies have suggested that patients who take statins have lower rates of MI and stroke after vascular surgery,[326,327] and two RCTs confirmed that peri-operative statin use (mean 30 – 37 days) reduced adverse cardiovascular events after vascular surgery.[328,329] These findings have been corroborated in a recent meta-analysis, which reported short term survival benefits following AAA repair for patients taking statins.[207]

Antiplatelet monotherapy with aspirin or thienopyridines (e.g., clopidogrel) does not pose an excessive bleeding risk during AAA repair.[330–332] In a sub-study of the Peri-operative Ischaemic Evaluation 2 (POISE-2) RCT, including 265 patients having AAA repair, peri-operative withdrawal of chronic aspirin therapy did not increase cardiovascular or vascular occlusive complications. Although a protective effect of peri-operative antiplatelets is uncertain, evidence is lacking for the need to withdraw antiplatelet monotherapy prior to EVAR or OSR for AAA.

Certain circumstances may necessitate continuation of dual antiplatelet, but mostly in high risk patients, in whom the balance of risks of AAA repair should be considered carefully.[333] Experience of dual therapy including more potent antiplatelet agents, such as prasugrel and ticagrelor, and AAA repair is very limited but is probably associated with a higher risk of serious bleeding and should be avoided. Warfarin and direct oral anticoagulants should be discontinued at least five days and two days respectively, prior to surgery to mitigate the risk of excessive bleeding. Depending on the indications for their use, anticoagulation may be bridged during the peri-operative period using a short acting agent such as low molecular weight heparin (LMWH) or unfractionated heparin. In general, applicable guidelines should be consulted for specific guidance on antiplatelet and or anticoagulant therapy during the peri-operative period of AAA repair.[334–336]

Contemporary data from the VQI in the USA have identified that combined statin and antiplatelet therapy at discharge following elective repair of AAA by either OSR or EVAR was associated with a long term survival benefit, particularly for those with a history of atherosclerotic cardiovascular disease.[337]

Recommendation 38			Unchanged
Initiation of beta blockers is not recommended prior to abdominal aortic aneurysm repair.			
Class	Level	References	ToE
III	A	Yang *et al.* (2006),[184] Brady *et al.* (2005),[323] Devereaux *et al.* (2008)[324]	

Recommendation 39			Unchanged
Patients undergoing elective abdominal aortic aneurysms repair should start statin treatment pre-operatively (ideally at least four weeks before surgery) and continue indefinitely post-operatively.			
Class	Level	References	ToE
I	A	Xiong *et al.* (2022),[207] De Martino *et al.* (2015),[326] Lindenauer *et al.* (2004),[327] Durazzo *et al.* (2004),[328] Schouten *et al.* (2009),[329] Risum *et al.* (2021)[338]	

Recommendation 40			Changed
Patients undergoing elective open or endovascular abdominal aortic aneurysm repair should be considered for continuation of established monotherapy with aspirin or thienopyridines (e.g., clopidogrel) during the peri-operative period.			
Class	Level	References	ToE
IIa	B	Burger *et al.* (2005),[330] Stone *et al.* (2011)[339]	

Recommendation 41		New
Patients undergoing elective abdominal aortic aneurysms repair are not recommended to be on dual therapy or oral anticoagulants during the peri-operative period.*		
Class	Level	References
III	C	Consensus

* See also Recommendation 31.

5.2.2. Antibiotic prophylaxis. Multiple RCTs have shown the benefits of systemic broad spectrum antibiotic prophylaxis during arterial reconstruction.[340,341] Contemporary data from the SVS-VQI confirms that prophylactic antibiotics reduce surgical site infections and in hospital mortality following EVAR.[287] Therefore, peri-operative intravenous antibiotic prophylaxis is recommended prior to both open and endovascular AAA repair, with the choice of agent based on local institutional guidelines.

An association between dental status and prosthetic valve endocarditis has been described,[342] which is why routine examination of dental status prior to major cardiac surgery with implantation is advocated by some. However, the evidence for the benefit of this routine is insufficient and there are divergent recommendations among professional societies. The incidence of aortic graft or stent graft infection is significantly lower than for prosthetic valve endocarditis[341,343] and the corresponding correlation with dental status is missing. Although it is reasonable to remedy an established or suspected dental infection before AAA repair, there is a lack of support for routine dental examination before aortic repair.

Recommendation 42			Unchanged
All patients undergoing open or endovascular abdominal aortic aneurysm repair should receive peri-operative systemic antibiotic prophylaxis.			
Class	Level	References	ToE
I	A	Eldrup-Jorgensen *et al.* (2020),[287] Stewart *et al.* (2007)[340]	

5.2.3. Anaesthesia and post-operative pain management. Multimodal pain therapy, including the use of a non-opioid regimen should be instituted to maximise the efficacy of pain relief, while minimising the risk of side effects and complications.[344] This approach may include the use of epidural analgesia, patient controlled analgesia, or placement of catheters for continuous infusion of local anaesthetic agents into the wound.

For open AAA repair, a Cochrane review including 1 498 patients from 15 trials[345] demonstrated that post-operative epidural analgesia provided better pain management compared with systemic opioid based analgesia including reduced rates of MI, faster endotracheal extubation with reduced incidence of post-operative respiratory failure, and shorter stays on the intensive care unit (ICU). However, there was no difference in 30 day mortality. In contrast, a retrospective study from the USA, investigating 1 540 patients undergoing elective AAA surgery, found improved survival and significantly lower morbidity and mortality rates if general anaesthesia was combined with epidural anaesthesia.[346] These findings however were not supported by a more recent retrospective study based on data from the National Surgical Quality Improvement Program (NSQIP) in the USA of 2 145 patients undergoing OSR, which reported no survival benefit or reduction in major complications from combined epidural and general anaesthetic for OSR and furthermore increased blood transfusion requirements.[347]

There is a wealth of evidence supporting the use of catheter based continuous wound analgesia in cardiothoracic, orthopaedic, general, urological, and gynaecological surgery, but there are no published data specific to aortic surgery.[348]

There are no RCTs comparing various methods of anaesthesia for EVAR in AAA. The international multicentre Endurant Stent Graft Natural Selection Global Post-Market Registry (ENGAGE) study examined the outcomes of 1 231 patients undergoing EVAR under general (62% of patients), regional (27%), and local (11%) anaesthesia. The investigators concluded that the type of anaesthesia had no influence on peri-operative mortality or morbidity.[349] A contemporary meta-analysis reported the benefits of local anaesthesia in EVAR to be less clear during elective than rAAA.[349] Locoregional anaesthesia, however, appeared to reduce procedure time, ICU admissions, and post-operative hospital stay[350–353] and data from the UK's National Vascular Registry including 9 783 patients receiving an elective, standard infrarenal EVAR showed a lower 30 day mortality rate after regional *vs.* general anaesthesia.[354]

While the data regarding the preferred method of anaesthesia in elective EVAR are limited, the GWC find it to be appropriate, in the light of current evidence and the proven benefit of local anaesthesia in ruptured EVAR, to issue a weak recommendation favouring locoregional anaesthesia over general anaesthesia in elective settings.

Recommendation 43		Changed
Patients undergoing elective open abdominal aortic aneurysm repair may be considered for peri-operative epidural analgesia or catheter based continuous wound analgesia, to maximise pain relief and minimise early post-operative complications.		
Class	**Level**	**References** ToE
IIb	A	Guay *et al.* (2016),[345] Mungroop *et al.* (2019)[348]

Recommendation 44		New
Patients undergoing elective endovascular abdominal aortic aneurysm repair may be considered for locoregional anaesthesia in preference to general anaesthesia.		
Class	**Level**	**References** ToE
IIb	C	Liu *et al.* (2021)[350] Cheng *et al.* (2019),[353] Dovell *et al.* (2020)[354]

5.2.4. Intra-operative imaging. The technical success of EVAR relies on accurate intra-operative imaging. For standard EVAR, regular digital subtraction angiography (DSA) is usually sufficient to ensure correct stent graft deployment and to detect the presence of endoleaks.

Other intra-operative imaging modalities, such as image fusion, cone beam CT, and intravascular ultrasound (IVUS) may also be of value in standard EVAR, in reducing radiation dose and in identifying endoleaks and stent graft compression and kinks, however they are mainly used in complex EVAR and are discussed further in Chapter 8.

5.2.5. Radioprotection measures. It is essential that clinicians who work with radiation understand the risks involved (for patients, themselves, and other healthcare personnel) and the measures that can minimise this risk and the radiation dose.[355–358] Radiation during EVAR has been shown to cause acute deoxyribonucleic acid damage in operators[359] as well as chronic deoxyribonucleic acid damages, including chromosomal aberrations that may herald genomic instability and predisposition to malignancy,[360] and research has highlighted the benefit of wearing full protective shielding.[359]

Adherence to the ALARA (as low as reasonably achievable) principle[361] has been demonstrated to reduce radiation exposure during EVAR,[362] and operators should know and apply the ALARA principle to protect the patient, themselves and team members.[363]

Furthermore, modern fixed imaging systems have been shown to reduce radiation doses to both patients and providers[364] and EVAR performed with a mobile C arm should be avoided.[363] A contemporary review has also supported the use of hybrid operating rooms and modern imaging equipment to improve imaging quality and reduce radiation exposure.[365]

Table 12 summarises radiation safety measures recommended during EVAR of AAA. For more information and detailed recommendations regarding radiation safety and protection, please consult the ESVS 2023 radiation safety guidelines.[363]

5.2.6. Cell salvage. Intra-operative red blood cell salvage involves aspiration, washing, and filtration of patient blood during an operation to minimise blood loss by re-transfusion. Cell salvage has been shown to reduce the need for the intra-operative use of allogeneic blood during elective open AAA repair.[366,367] Contemporary data has also suggested the use of cell salvage reduces one year mortality rates after OSR.[287]

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Table 12. Summary of radiation safety measures during endovascular abdominal aortic aneurysm repair.[363]

Maintain distance from the radiation source
Limit fluoroscopy pulse rate, time of exposure, use of digital subtraction acquisitions, steep C arm angulations, and magnification
Position the image intensifier close to the patient and put the table high, with a well collimated beam
Diligent use and appropriate positioning of lead shields, including personal shields (personalised apron, thyroid, shins and goggles) and mobile shields
Use advanced imaging techniques (e.g., image fusion)
Use of hybrid operating room, with a fixed imaging system (in preference over a mobile system)

Recommendation 45 — Unchanged

Intra-operative use of cell salvage and re-transfusion should be considered during open abdominal aortic aneurysm repair.

Class	Level	References	ToE
IIa	B	Eldrup-Jorgensen *et al.* (2020),[287] Marković *et al.* (2009),[366] Pasternak *et al.* (2014)[367]	

5.2.7. Intra-operative heparin administration. To minimise the risk of thrombosis due to stasis, heparin is administered systemically before cross clamping, or locally, during OSR or at onset of EVAR. Although, a systematic review found limited evidence for the efficacy of heparin in AAA repair,[368] it is a general vascular surgery principle. Accepted doses range between 50 and 100 IU/kg,[368] and heparin efficacy may be tested using an activated clotting time (ACT) test to ensure adequate anticoagulation.[369] In a multicentre registry study (measuring the ACT during non-cardiac arterial procedures), including 186 patients undergoing non-cardiac arterial procedures, a standardised dose of 5 000 IU heparin did not provide adequate anticoagulation, resulting in thromboembolic complications in 9% of patients.[370] A weight based dose with a starting dose of 100 IU/kg was more appropriate to reach adequate anticoagulation levels, with a target ACT $\geq$ 200 seconds resulting in the lowest frequency of thromboembolic complications (4.3%). To limit bleeding complications, a target ACT of 200 – 220 seconds seemed optimal.[371] Additional relevant information is anticipated from the ongoing ACT Guided Heparinisation During Open Abdominal Aortic Aneurysm Repair (ACTION-1) trial, an RCT investigating whether ACT guided heparinisation results in safe and more optimal coagulation than 5 000 IU as a single bolus during open AAA repair.[372]

Once peripheral perfusion has been re-established, protamine sulphate may be administered to reverse heparinisation based on ACT test and the presence of diffuse bleeding or oozing. There are, however, no data regarding the role of protamine specifically in AAA repair.

Recommendation 46 — Unchanged

In elective open or endovascular abdominal aortic aneurysm repair intra-operative administration of intravenous heparin (50 – 100 IU/kg) is recommended.

Class	Level	References	ToE
I	C	Wiersema *et al.* (2012)[368]	

Recommendation 47 — New

Intra-operative use of activated clotting time (ACT) may be considered during open and endovascular abdominal aortic aneurysm repair, to measure the effect of heparin in the individual patient and guide additional heparin administration.

Class	Level	References	ToE
IIb	B	Wiersema *et al.* (2012),[368] Doganer *et al.* (2022),[371] Doganer *et al.* (2021)[373] Doganer *et al.* (2020),[374] Roosendaal *et al.* (2022)[375]	

5.2.8. Venous thrombosis prophylaxis. Venous thromboembolism (VTE) is an important cause of post-operative morbidity and mortality after major surgery, often caused by immobilisation and old age. Consequently, routine VTE prophylaxis is recommended after major abdominal and orthopaedic surgery. There is, however, a paucity of literature that addresses the effectiveness of VTE prophylaxis specifically in the AAA repair setting. In a retrospective single centre study the incidence of symptomatic VTE was 4% after OSR and 0% after EVAR, with a reduced risk after chemoprophylaxis.[376] A meta-analysis on the effect of VTE prophylaxis in patients undergoing vascular surgery, including eight OSR and EVAR publications, was only able to demonstrate a non-statistically significant trend towards lower VTE risk among patients receiving VTE prophylaxis (RR 0.7). The authors suggested a selective VTE prophylaxis strategy, based on the risk of development of post-operative VTE, in patients undergoing major vascular surgery.[377] Similarly, a recent systematic review and meta-analysis of RCTs assessing the role of thromboprophylaxis after vascular surgery, including eight studies with a total of 3 130 patients, demonstrated a non-significant trend towards a lower risk of post-operative deep venous thrombosis (DVT) (RR 0.3, $p = .060$) and pulmonary embolism (RR 0.17, $p = .17$) among patients receiving VTE prophylaxis. There was no difference for bleeding outcomes between anticoagulants and placebo, and there was no significant difference in outcomes when LMWH was compared directly with unfractionated heparin.[378] In summary, it is recommended to evaluate the risk of post-operative VTE in all patients undergoing elective AAA repair, and in patients deemed at risk, thromboprophylaxis should be considered. Local hospital routines should be consulted regarding drug choice, dose and duration.

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Recommendation 48			New
All patients undergoing elective abdominal aortic aneurysm repair and deemed at risk of post-operative venous thromboembolism should be considered for thromboprophylaxis.			
Class	Level	References	ToE
IIa	C	Toth *et al.* (2020),[377] Haykal *et al.* (2022)[378]	

5.3. Techniques for elective abdominal aortic aneurysm repair

5.3.1. Open repair

5.3.1.1. Types of grafts. Textile polyester material, specifically polyethylene terephthalate, commonly known by its brand name Dacron, has been the most frequently used material for 70 years. Different manufacturers employ different kinds of sealing impregnation (i.e., gelatin, albumin, etc.) to obtain zero graft porosity. Expanded polytetrafluoroethylene (ePTFE) has also been used for aorto-iliac reconstruction. There are no data to suggest that one graft is superior to another.

Vascular grafts with antimicrobial substances such as silver with or without Triclosan or rifampicin are available. A recent systematic review and meta-analysis identified only six studies on antimicrobial coating strategies such as antibiotics ($n = 3$) and silver ($n = 3$), with only three comparing coated with uncoated grafts (two antibiotic and one silver). Two RCTs reported on the protective effect of rifampicin soaked grafts on graft infection and showed no significant effect in the early (two months; OR 0.69, 95% CI 0.29 – 1.62) or late (two years; OR 0.73, 95% CI 0.23 – 2.32) post-operative periods. A retrospective cohort study focusing on the effect of silver coated grafts did not reveal any advantage (OR 0.19, 95% CI 0.02 – 1.64).[379] Thus, there is no evidence supporting the routine use of these grafts, or soaking grafts in rifampicin, to prevent or to reduce aortic graft infection (AGI).

Recommendation 49			New
For open abdominal aortic aneurysm repair, routine use of antimicrobial coated grafts to prevent aortic graft infection is not recommended.			
Class	Level	References	ToE
III	B	Mufty *et al.* (2022)[379]	

5.3.1.2. Incision and approach. A midline incision through the linea alba from the xiphoid to the pubis is the most widely used technique because of its flexibility and the option to access all abdominal organs with relative ease. Alternative accesses include the transverse subcostal incision below the ribcage allowing good access to the juxtarenal, suprarenal, and coeliac portions of the aorta, and the left retroperitoneal approach providing access in more proximal aneurysm disease, inflammatory aneurysms, or hostile abdomen. A RCT on an AAA population showed a lower incidence of hernia after transverse incision than vertical incision.[380] A Cochrane review however found no clinically important difference between midline and transverse incisions in general abdominal surgery,[381] which was confirmed in a later RCT.[382] With very low certainty evidence from five small RCTs, a recent Cochrane systematic review showed no major differences between the transperitoneal and the retroperitoneal route for elective open AAA repair in terms of mortality, rates of complications including haematoma or chronic wound pain, aortic cross clamping time, and operating time.[383] A recent systematic review and meta-analysis showed no significant differences in incisional hernia rate between transverse abdominal and vertical midline incisions, and between midline transperitoneal and retroperitoneal incisions, in patients undergoing OSR.[384] Repair of midline incisional hernia might be easier than the retroperitoneal incision. Therefore, the decision about the incision should be driven by surgeon preference and patient factors.

For infrarenal AAA repair, the proximal landmark for exposure is the left renal vein, which often has to be mobilised to facilitate exposure of the aorta just below the renal arteries. Sometimes, left renal vein division may be needed to gain adequate exposure and facilitate the subsequent proximal anastomosis. Retrospective single centre reports suggests that ligation of the left renal vein is associated with increased levels of acute kidney injury in the early post-operative phase but does not affect long term renal function or mortality.[385–389] Left renal vein ligation should be performed close to the inferior vena cava in order to preserve left renal vein tributaries including inferior adrenal, phrenic, gonadal, and lumbar veins to preserve the venous return from the left kidney. When kept intact, these tributaries usually allow left renal vein ligation to be performed without significant left kidney dysfunction.[390,391] Thus, routine reconstruction of the left renal vein after its division during open abdominal aortic aneurysm repair is not indicated but may be considered in selected cases when important collaterals have been sacrificed.[392,393]

Recommendation 50			New
For open abdominal aortic aneurysm repair, the choice of a midline *vs.* transverse or transperitoneal *vs.* retroperitoneal abdominal incision should be considered based on surgeon preference and patient factors.			
Class	Level	References	ToE
IIa	B	Mei *et al.* (2021)[383]	

Recommendation 51			New
Reconstruction of the left renal vein after its division during open abdominal aortic aneurysm repair may be considered if important collaterals have been sacrificed.			
Class	Level	References	ToE
IIb	C	Pandirajan *et al.* (2020)[386]	

5.3.1.3. Open surgical aortic reconstruction. The proximal anastomosis should be sutured as close as possible to the renal arteries even in long necks, to prevent later aneurysm development in the remaining infrarenal aortic segment. On a histological level, advanced matrix degradation may also be present in seemingly healthy necks, leading to proximal aneurysm formation or anastomotic false aneurysm formation. Furthermore, the orientations of the medial collagen fibres near the origin of the renal arteries provide improved mechanical properties.[394,395]

The proximal end to end anastomosis is usually performed with a non-resorbable monofilament running suture (4-0 — 2-0). Pledgets (e.g., prosthesis, bovine pericardium, Teflon, etc.) may be employed to reinforce the suture line if the tissue is friable. The distal anastomosis is performed in a similar fashion (5-0 — 2-0), after sufficient flushing of both iliac arteries and the graft to prevent distal embolisation.

Bifurcated grafts should be tailored to maintain sufficient main body length to facilitate endovascular re-intervention in the future. At least one internal iliac artery (IIA) should be preserved or re-implanted when possible, to provide sufficient perfusion of pelvic organs and to reduce the risk of buttock claudication and colonic ischaemia.[396—399] Suture ligation of the inferior mesenteric artery (IMA) should be performed at its origin from the aneurysm sac to preserve left colic collaterals. There is no evidence in the literature to support routine re-implantation of a patent IMA, but it may be considered in selected cases of suspected insufficient visceral perfusion with risk of colonic ischaemia, for example if the superior mesenteric artery (SMA) is occluded and the IMA is an important collateral or in diseased IIAs. Often, the need is only recognised intra-operatively. If in doubt, re-implantation should be performed using a small Carrel patch of aortic wall around the origin of the IMA to reimplant it end to side to the graft or one of its limbs, or through a bypass.[400,401]

The cross clamping time should be as short as possible to minimise lower body ischaemia, cellular damage and metabolic injury. Coordination with the anaesthesia team is particularly important at the time of declamping. The distal circulation should be checked and if necessary promptly corrected.

Recommendation 52			Unchanged
For open abdominal aortic aneurysm repair, it is recommended to perform the proximal anastomosis as close as possible to the renal arteries to prevent later aneurysm development in the remaining infrarenal aortic segment.			
Class	**Level**	**References**	**ToE**
I	C	Lipski *et al.* (1998),[394] Cao *et al.* (2009)[395]	

Recommendation 53			Unchanged
For open abdominal aortic aneurysm repair, it is recommended to preserve the blood flow to at least one internal iliac artery to reduce the risk of buttock claudication and colonic ischaemia.			
Class	**Level**	**References**	**ToE**
I	C	Björck *et al.* (1997),[396] Marconi *et al.* (2015),[397] Björck *et al.* (2000),[398] Becquemin *et al.* (2008)[399]	

Recommendation 54			Changed
In open abdominal aortic aneurysm repair routine re-implantation of the inferior mesenteric artery is not indicated but should be reserved for selected cases of suspected insufficient pelvic organ perfusion and the risk of colonic ischaemia.			
Class	**Level**	**References**	**ToE**
III	C	Killen *et al.* (1999),[400] Senekowitsch *et al.* (2006)[401]	

5.3.1.4. Abdominal closure. Incisional hernia is a well known complication of laparotomy and requires treatment in 7 — 26% of patients.[402—404] In addition to post-operative wound complications, smoking, COPD, and obesity, AAA repair is an independent risk factor for the development of incisional hernia.[384,405]

The closure technique is crucial to reduce the rate of wound complications in midline incisions. Fascial closure with small bites and a suture length to wound length ratio greater than four to one significantly reduces the risk of incisional hernia and is a generally recommended surgical technique.[384,406,407]

Two systematic review and meta-analysis showed that sublay (retromuscular) or onlay prophylactic mesh reinforcement of midline laparotomies significantly reduces the risk of incisional hernia after OSR. There was, however, no clear effect on the frequency of re-operation.[384,408] In the recently published five year follow up results of the PRIMAAT (Prevention of Incisional Hernia by prophylactic mesh augmented reinforcement of midline laparotomies for abdominal aortic aneurysm treatment) RCT including 120 patients, the cumulative incidence of incisional hernia in the no mesh group was 33% after 24 months and 49% after 60 months, compared with none in the mesh group. In the no mesh group 22% underwent re-operation within five years due to an incisional hernia.[409] Thus, it is reasonable to consider the prophylactic mesh reinforcement technique using a permanent synthetic mesh in patients with AAA who undergo OSR.[408,410] Adjunctive post-operative complications may occur, such as infection, seroma and need for re-intervention depending on the technique used.[410]

Updated guidelines for the closure of abdominal wall incisions have recently been published by the European and American Hernia Societes.[406,411]

Recommendation 55			Changed
For open abdominal aortic aneurysm repair, prophylactic use of mesh reinforcement of midline laparotomies should be considered.			
Class	Level	References	ToE
IIa	A	Nicolajsen *et al.* (2020),[384] Indrakusuma *et al.* (2018),[408] Dewulf *et al.* (2022),[409] Jairam *et al.* (2017)[410]	

5.3.2. Endovascular repair. Unlike OSR, a stent graft is meant to seal the sac from the inside of the aneurysm, while the aneurysm wall is left untouched. The paradigm is therefore changed from replacing the aneurysm to excluding it from the systemic circulation. Therefore, the anchoring segments need to provide both sufficient sealing and fixation. Most devices rely on some degree of oversizing of the stent graft to guarantee sealing and fixation. The degree of oversizing required, which ranges from 10% to 25%, varies between different devices.

Most stent grafts now adopt a modular design with two or three separate components including an aortic bifurcated main body and one or two iliac limbs. This has several important advantages. With a relatively limited stock, devices can be tailored precisely to the diameters and lengths of the vessels of the individual patient. Moreover, taking advantage of the overlap between components gives a degree of flexibility in planning.

Additional features that are specific to individual types of device include the option to reposition the proximal portion of the device during deployment, the presence of proximal bare stents for suprarenal fixation, and hooks or barbs for additional fixation. There are no data that convincingly favour any of the above features or one particular EVAR device over another.[412] Comparative studies are lacking and given the rapid technological development, even within the same brand, device specific studies are rapidly outdated. Pending further evidence, local preference and experience should therefore guide device selection. Consideration should also be given to the availability of unbiased performance and long term durability data.

There are several anatomical requirements specific to individual stent grafts which are specified in their respective IFU. Figure 2 shows various anatomical metrics of relevance for EVAR device planning, and Table 13 summarises the range of anatomical requirements according to the latest available IFU of the stent grafts which are either FDA approved or have a CE mark: AFX 2, Ovation iX and Alto (Endologix, Irvine, CA, USA), Altura and Aorfix (Lombard Medical, Oxfordshire, United Kingdom), Anaconda (Terumo Aortic, Vascutek Ltd, Inchinnan, United Kingdom), Endurant II (Medtronic Cardiovascular, Santa Rosa, CA, USA), Excluder C3 and Excluder Conformable stent graft (W.L. Gore and Associates, Flagstaff, AZ, USA), E-vita and E-tegra (Jotec/Artivion, Hechingen, Germany), Incraft (Cordis Corporation, Bridgewater, NJ, USA), Treo (Terumo Aortic, Bolton Medical Inc., Sunrise, FL, USA), and Zenith Alfa and Zenith Flex (Cook Medical, Bloomington, IN, USA).

In a multicentre observational study on 10 228 patients, only 41% were treated within device IFU with an associated high rate of AAA sac enlargement.[413] A retrospective analysis of 4 587 patients (13.9% treated outside IFU), collated from the ENGAGE and the Global Registry for Endovascular Aortic Treatment (GREAT), reported more Type IA endoleaks in patients treated outside IFU, but similar results when comparing other outcomes.[414] A systematic review on 17 observational studies including 4 498 patients, with 40% of patients treated outside IFU, reported similar aneurysm related outcomes between groups but a higher overall mortality when treating outside IFU. Outside IFU the use of devices may have medicolegal implications in some countries, in such a way that the manufacturer's liability for device quality no longer applies. Instead, responsibility is assumed by the operating surgeon, centre, or hospital.[415] And, with today's access to proven fenestrated and branched endovascular devices for complex AAAs (see Chapter 8), there is every reason to follow the IFU, especially in the elective setting.

Special care should be taken in the management of the iliac limb components. An Italian single centre study showed a very low rate of late iliac limb occlusions when using a dedicated protocol for intra-operative iliac limb management: (1) pre-EVAR angioplasty of common and external iliac artery (EIA) stenoses; (2) precise contralateral iliac limb deployment at the same level as the flow divider (even if the endograft IFU allowed its deployment more proximally); (3) iliac limb kissing ballooning with high pressure non-compliant balloons; (4) iliac limb stenting for residual tortuosity or kinking and adjunctive external iliac stenting for residual stenosis or dissection after EVAR.[416] A systematic review, including four observational studies with 1 132 patients, reported no differences between the cross limb (Ballerina) and standard limb configuration in terms of limb occlusion, endoleaks, or mortality.[417]

Recommendation 56		New
For endovascular abdominal aortic aneurysm repair, device selection should be considered based on aorto-iliac anatomy and the availability of unbiased long term durability data.		
Class	Level	References
IIa	C	Consensus

Recommendation 57			New
Endovascular abdominal aortic aneurysm repair outside the manufacturer's Instruction for Use is not recommended in the elective setting.			
Class	Level	References	ToE
III	C	Barry *et al.* (2021),[414] Antoniou *et al.* (2020)[415]	

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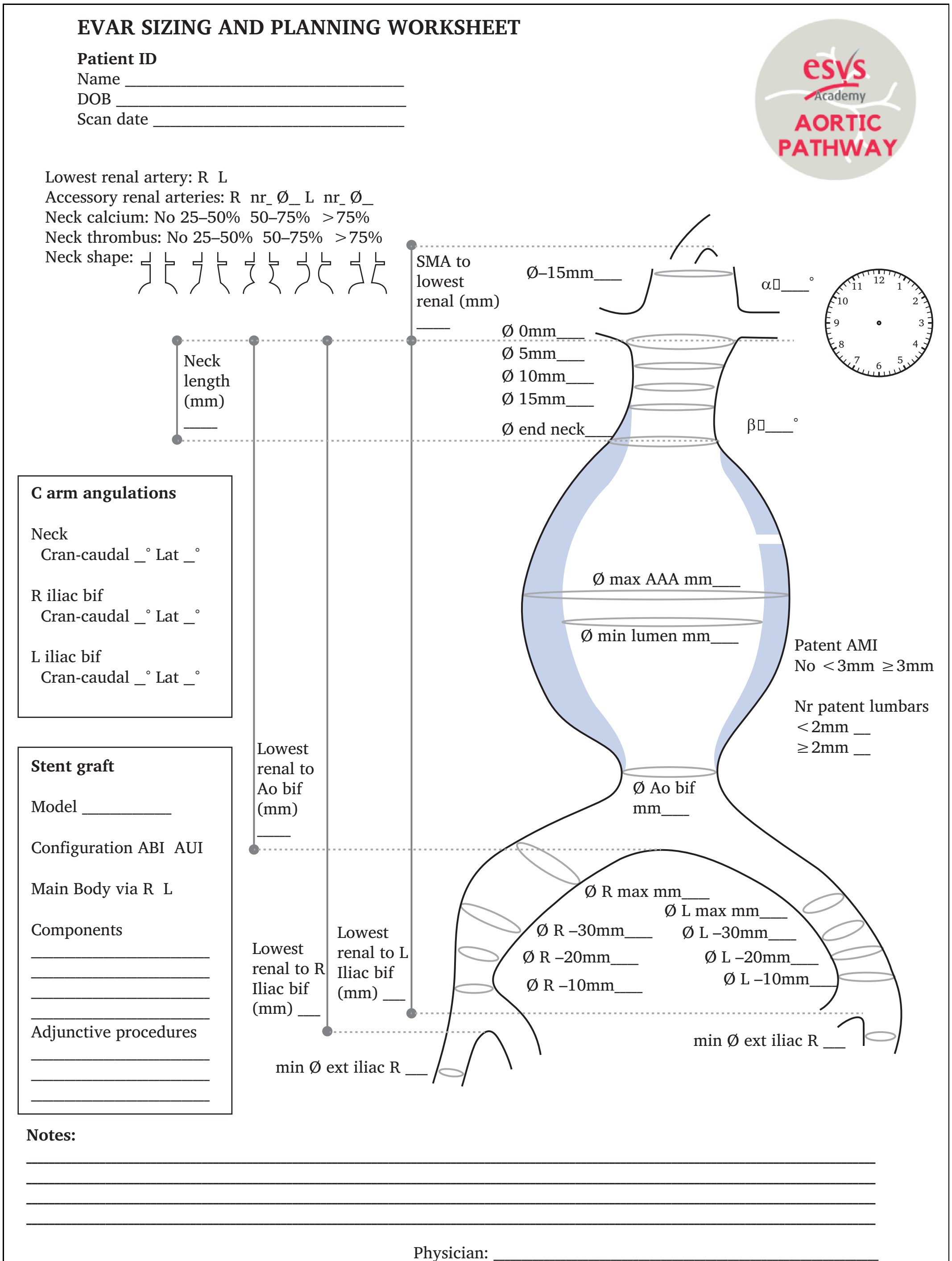

Figure 2. Anatomical metrics of relevance for endovascular abdominal aortic repair device planning modified from the European Society for Vascular Surgery Academy Sizing and Planning Workshop. EVAR = endovascular aortic repair; ABI = aorto-bi-iliac; AUI = aorto-uni-iliac; SMA = superior mesenteric artery; IMA = inferior mesenteric artery

5.3.2.1. Newer generation of stent grafts. In recent years, manufacturers have developed new stent grafts and delivery systems with lower profiles to allow endovascular AAA treatment in patients with small access vessels.

Although there are some series reporting favourable midterm outcomes for latest generation low profile stent grafts compared with standard profile stent grafts,[418] an Italian multicentre registry, including 619 patients, showed that the Zenith Flex Low Profile [LP] endograft (Cook Medical Inc, Bloomington, Ind) was associated with a significantly higher rate of late clinical failure and re-intervention compared with the Ovation stent graft (Endologix, Irvine, CA, USA) and the Incraft endograft system (Cordis Corporation, Bridgewater, NJ).[419] And, a recent

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Table 13. Range of anatomical requirements according to currently available stent grafts Instructions for Use.

Morphological applicability	Range
Minimum diameter of proximal neck	16–19 mm
Maximum diameter of proximal neck	28–32 mm
Minimum length of proximal neck	10–15 mm (varies depending on neck angle for some stent grafts)
Maximum angle β (infrarenal angle)	45–90° (varies depending on neck length for some stent grafts)
Maximum angle α (suprarenal angle)	45–60° (or not applicable for some stent grafts)
Maximum zone of calcification or thrombus of the proximal neck	50%
Minimum diameter of aortic bifurcation	12–21 mm
Minimum diameter of common iliac artery	7–11 mm
Maximum diameter of common iliac artery	18–25 mm
Minimum length of distal landing zone of common iliac artery	10–20 mm
Minimum ipsilateral access vessel diameter	5–8.5 mm (varies depending on proximal neck diameter for some stent grafts)
Minimum contralateral access vessel diameter	3.5–7 mm (varies depending on common iliac artery diameter for some stent grafts)

Swedish multicentre study, including 924 patients, found device type to be an independent risk factor for graft limb occlusion after EVAR. Specifically, the low profile device Zenith Alpha (Cook Medical Inc, Bloomington, Ind) demonstrated a strikingly high risk of limb occlusion compared with older devices; after a median 37 months of follow up, the limb occlusion rate was 12.4% in patients with Zenith Alpha, compared with 0.7% with Gore Excluder (W.L. Gore and Associates, Flagstaff, AZ, USA), and 3.3% with Endurant (Medtronic Cardiovascular, Santa Rosa, CA, USA). Similar findings were reported in a retrospective Norwegian study where the Zenith Alpha stent graft was a significant independent risk factor for limb graft occlusion compared with Endurant II (15% *vs*. 4%, OR 3.9).[420] In a nested case control analysis, the OR for graft limb occlusion was 5.3 (95% CI 2.0 – 14.3) for the Zenith Alpha device.[421] In a single centre Danish study, the cumulative incidence of graft limb occlusion after EVAR with the Zenith Alpha graft was 7% per limb up to three years post-operatively.[422] In a further analysis, this was not associated with risk factors suggested by the IFU,[422] and it is yet unclear whether the increased risk of graft limb occlusion has been addressed by the updated Zenith Alpha IFU.

Recently, alarming reports of a high frequency of late Type 3 endoleaks have been reported for the Endologix AFX Endovascular AAA System (Endologix, Irvine, CA, USA), with its novel bifurcated unibody (as opposed to regular modular stent graft systems). In a single centre time to event analysis, freedom from Type 3 endoleak was 48% at eight years for the early generation AFX device. Most of these were not detected until several years (> 4.5 years) after initial implantation.[423] In a recent linked registry claims study from the USA, the crude five year re-intervention rate was significantly higher for patients who received the early AFX device, 27% *vs*. 15 – 19% for other devices (HR 1.6, 95% CI 1.3 – 2.0).[13] Currently, it is uncertain whether the increased Type 3 endoleak risk has been addressed by the updated AFX2 device[424] and the U.S. FDA recently issued a recommendation for healthcare providers to consider using available alternative treatment options for patients with AAA rather than the *AFX2* device.[425]

The Valiant Navion thoracic stent graft system (Medtronic Inc, Santa Rosa, CA, USA) was designed with improved conformability and a lower profile, and early patient outcomes were generally positive. Recently, however, unexpected late structural failures of the stent graft have been observed with stent fractures and Type 3b endoleaks. In response to these adverse events, the manufacturer decided to issue a voluntary global recall of the device in 2021.[426,427]

These data emphasise that caution should be exercised when new and or modified stent grafts are being launched, and they should be subjected to careful and long term evaluation before these new devices can be considered safe. Thus, when upgrades of existing platforms are used in clinical practice, the need for long term follow up should be recognised, and evaluation in prospective registries, with complete follow up is recommended to ensure device performance and procedural durability through 10 years,[13,427–430] with analyses with sufficient power to detect non-inferiority of future graft performance.[431] This is also in line with the recent United States FDA recommendation of extended monitoring of device performance through 10 years post-EVAR.[432]

Recommendation 58			Changed
For newer generations of stent grafts for abdominal aortic aneurysm treatment based on existing platforms, such as low profile devices, long term follow up in prospective registries is recommended, to ensure device performance and procedural durability through 10 years.			
Class	**Level**	**References**	**ToE**
I	C	Consensus	

5.3.2.2. New techniques and concepts for abdominal aortic aneurysm repair. The *Nellix* endoprosthesis (Endologix, Inc, Irvine, CA, USA) was a new endovascular concept, called endovascular aneurysm sealing (EVAS) launched in the early 2010s, based on polymer filled polyurethane bags surrounding balloon expandable stents covered with PTFE to completely seal the aortic aneurysm sac. This approach was designed to prevent some of the complications of EVAR including endoleak and stent graft migration. After initial reports of encouraging early results, this new technology was rapidly disseminated with great enthusiasm.[433—435] However, reports gradually emerged of higher than expected rates of Type 1a endoleak, device migration, and aneurysm rupture,[12,436] and the ESVS 2019 AAA Guidelines recommended that new techniques and concepts (with special reference to EVAS) should only be used with caution within the framework of clinical trials, until adequately evaluated. Subsequent data revealed that the Nellix device presented an unacceptable failure rate, and the manufacturer eventually ended its production (Endologix, Nellix End of Life Communication. 10 May, 2022). The ESVS Guideline Committee recently published a Focused Update to encourage the identification of patients in whom a *Nellix* device has been implanted and enrol them in enhanced surveillance programmes and if device failure is detected, offer those patients early elective device explantation if feasible.[7] The rise and fall of the Nellix device illustrates the importance of rigorous evaluation of new technology, methods, and devices before they are widely adopted. The safety and efficacy for several new innovative technologies on the market remains unclear and further data are needed before these can be recommended for routine use in clinical practice.[427,428] Endosuture aneurysm repair (ESAR) is another example of a relatively new and yet unproven concept falling under this category. ESAR is intended to treat AAAs with a short neck outside IFU of standard EVAR devices by means of endostaples or endoanchors, such as the *Heli-FX EndoAnchor* (Medtronic Cardiovascular, Santa Rosa, CA, USA). SOCRATES (ShOrt neCK AAA RAndomised trial) is an ongoing RCT comparing ESAR with fenestrated EVAR (fEVAR) in short neck AAA (4 — 15 mm). ESAR is further discussed in Chapter 8.

Recommendation 59		**Unchanged**
New techniques and concepts for abdominal aortic aneurysm treatment are not recommended to be used routinely in clinical practice but should only be used within the framework of studies approved by research ethics committees, until adequately evaluated.		
Class	**Level**	**References**
III	C	Consensus

The new EU regulation 2017/745 on medical devices, or EU MDR,[437] is a major update to medical device regulations in Europe. The MDR replaces the previous EU Medical Device Directive and is designed to modernise the EU regulatory system to better address the current needs of the market and new technologies. Devices that received a CE mark under the Medical Device Directive are allowed to continue to market in the EU but will need to be recertified under MDR by a Notified Body before 2024. The MDR has an increased focus on device safety with greater emphasis on clinical data and an expanded focus on regulating the entire lifecycle of a medical device by establishing new requirements for post-market surveillance studies. Exactly how this will affect the EVAR device market remains unclear.

5.3.2.3. Access. Stent grafts are generally delivered through the femoral artery either through a surgical cut down or percutaneously. Surgical exposure may be obtained by means of a limited longitudinal or transverse incision (under general, regional or local anaesthesia) and has the advantage of direct control of the artery and free choice of the ideal puncture site. A Cochrane review of 237 patients (283 groins) reported, with low certainty of evidence, fewer surgical wound infections in transverse groin incisions compared with longitudinal incisions, but no differences in lymphocele or lymphorrhoea. No other outcomes were evaluated in those studies.[438]

The percutaneous approach is less invasive and relies on arterial closure devices which usually need to be inserted before the sheath is introduced.[439] Femoral calcification represents the only predictor of percutaneous access failure.[440]

A recent systematic review identified four RCTs with a total of 368 participants (530 access sites) comparing surgical cut down with total percutaneous access for elective EVAR, with all patients being suitable for both methods. No significant differences were detected between the two methods regarding access site complications or infection, post-operative bleeding or haematoma, access related arterial injury, femoral artery occlusion, pseudoaneurysm, hospital length of stay, or peri-operative mortality, while seroma and lymphorrhoea were significantly less frequent after percutaneous EVAR compared with cut down EVAR (0% *vs.* 3%, OR 0.18, 95% CI 0.04 — 0.83) and the procedure time was significantly shorter (—12 minutes). All trials were, however, judged to be at high risk of bias or have some concerns, and the level of the body of evidence was low or very low for all outcomes, and the authors concluded that the evidence was very uncertain about the effect of percutaneous EVAR on clinically important outcomes.[441] Thus, there are no data clearly favouring one method over another, but the choice between percutaneous access or cut down should be determined by patient factors and operator preference.

In a systematic review and meta-analysis including 1 422 subjects from RCTs, US guidance was associated with a 49% reduction in overall complications, including haematoma and accidental venepuncture, and a 42% improvement in the likelihood of first attempt success compared with palpation guided access.[442] In a more recent systematic review and meta-analysis including 1 553 patients from five RCTs, US guidance femoral access (*vs.* palpation with or without fluoroscopy guidance) was associated with a reduction in the rate of vascular access related complications (1.9% *vs.* 4.3%, $p < .010$). However, this was primarily driven by a reduction in local haematomas, while the observed numerical reduction in major bleeding (0.3% *vs.* 1.3%, $p = .080$) or minor bleeding (1.4% *vs.* 2.8%, $p = .070$) did not reach statistical significance.[442] Furthermore, US guidance was associated with less venepuncture (3.7% *vs.* 16.9%, $p < .001$), a higher rate of first pass success (80.3% *vs.* 50.5%, $p < .001$), lower number of attempts ($p < .001$), and less access time (mean 24 seconds, $p < .001$).[443] In another systematic review and meta-analysis comparing US and fluoroscopy guided transfemoral TAVR access, including 3 875 patients from eight observational studies, the US guided approach was associated with significantly reduced risk of access site vascular complications (OR 0.50) and access site bleeding complications (OR 0.59).[444] In a more recent RCT US guided femoral artery cannulation had a higher rate of success, faster cannulation, and fewer venepunctures compared with fluoroscopic guidance, while the rates of complications did not differ.[445] US guidance seems to have the greatest benefit in patients with a high common femoral artery bifurcation (above the femoral head)[443] and appears to be easier to master for trainees.[445]

Recommendation 60			Changed
For endovascular abdominal aortic aneurysms repair, the choice of percutaneous access or cut down should be considered based on patient factors and operator preferences.			
Class	Level	References	ToE
IIa	B	Antoniou and Antoniou (2021)[441]	

Recommendation 61			Changed
For endovascular abdominal aortic aneurysms repair by a percutaneous approach, ultrasound guidance is recommended.			
Class	Level	References	ToE
I	A	Sobolev *et al.* (2015),[442] Seto *et al.* (2010),[443] Kotronias *et al.* (2021),[444] Stone *et al.* (2020)[445]	

5.3.2.4. Accessory renal arteries. Accessory renal arteries are present in 9 – 16% of patients undergoing EVAR, with half likely to be covered.[446] Potential consequences are renal infarction with risk of deterioration of renal function (particularly with pre-existing renal insufficiency) and risk of persistent Type 2 endoleak (T2EL).[447]

A systematic review found four studies that did not observe any significant changes of post-operative renal function, whereas one study reported an early transient increase in creatinine after coverage of accessory renal arteries that resolved within 30 – 90 days. The frequency of renal infarction varied between 20% and 84%. No significant change in BP, mortality, and mean length of hospital stay was observed. Five studies did not observe endoleaks related to accessory renal artery coverage, whereas one reported the occurrence of T2EL in three of 18 patients (17%) who had accessory renal artery coverage.[446] A recent meta-analysis including 302 patients with covered accessory renal arteries with a mean diameter $<$ 4 mm after standard EVAR and complex EVAR confirmed these results, with increased risk of renal infarction but no clinical effect on renal function or mortality rate.[448]

Thus, current evidence supports the covering of accessory renal arteries located in the proximal fixation zone, ensuring a good seal using the entire aortic neck despite sacrificing small calibre accessory renal arteries. It is recommended to try to preserve larger ($\geq$ 4 mm in diameter) or assumed significant accessory renal arteries (supplies $>$ 1/3 of the renal parenchyma), especially in cases with pre-operative renal insufficiency. Custom made fEVAR[449] or chimney EVAR (chEVAR)[450] are possible options to preserve accessory renal arteries in patients not suitable for OSR (see section 8.3).

There is currently no evidence to support pre-emptive embolisation of accessory renal arteries to be covered during EVAR,[446] however, it may be considered in large accessory renal arteries ($>$ 3 mm) that originate from the aneurysm sac.[451]

Recommendation 62			Changed
For patients undergoing endovascular abdominal aortic aneurysm repair, preservation of large accessory renal arteries (≥ 4 mm) or those that supply a significant portion of the kidney (> 1/3) should be considered, however without compromising adequate sealing.			
Class	Level	References	ToE
IIa	C	Lareyre *et al.* (2019),[446] Spanos *et al.* (2014)[448]	

Recommendation 63			New
For patients undergoing endovascular abdominal aortic aneurysm repair, routine pre-emptive embolisation of accessory renal arteries is not indicated.			
Class	Level	References	ToE
III	C	Lareyre *et al.* (2019)[446]	

5.3.2.5. Pre-emptive embolisation of inferior mesenteric artery, lumbar arteries, and sac. T2EL represent the most frequent complication in the follow up of patients treated by EVAR. Factors associated with persistent or recurrent T2EL include coil embolisation of internal iliac arteries, distal graft extension to the external iliac artery, age $\geq$ 80 years, and anatomical factors such as number and diameter of patent side branches arising from the aneurysm (IMA $\geq$ 3 mm and lumbar arteries $\geq$ 2 mm), and sac thrombus.[452–455]

Pre-emptive embolisation of side branches and or the aneurysm sac has been proposed to decrease the risk of T2EL and consequently of persistent aneurysm growth, re-interventions, and aneurysm related death. However, these techniques increase the procedure time, cost, and risk of complications.

In a recent meta-analysis, including 1 812 patients from 12 studies (one RCT and 11 retrospective controlled cohort studies), the overall incidence of T2EL was significantly lower in the embolisation group *vs*. the control group (17.3% *vs*. 24.5%, OR 0.36) as well as the incidence of persistent T2EL (15.3% *vs*. 30.0%, OR 0.37). Five studies reported a significantly lower incidence of sac enlargement

(10.2% *vs*. 24.9%, OR 0.25). Nine studies reported lower T2EL related re-interventions in the embolisation group (1.3% *vs*. 10.4%, OR 0.14). The technical success of collateral artery embolisation was 92.1% (455/494) in the 12 studies: 1.2% (10/829) of patients suffered a mild complication of collateral artery embolisation, and 2/829 patients died because of the embolisation.[456]

In the only RCT, 106 patients at risk of T2EL (patent IMA ≥ 3 mm, lumbar arteries ≥ 2 mm) were randomised to receive EVAR with or without IMA embolisation. After a mean follow up of 22 months, the incidence of T2EL was significantly lower in the embolisation group (24.5% *vs.* 49.1%), with an absolute risk reduction of 24.5% and number needed to treat 4.1. The aneurysm sac shrank significantly more in the embolisation group (−5.7 ± 7.3 mm *vs.* −2.8 ± 6.6 mm), and the incidence of aneurysm sac growth related to T2EL was significantly lower in the embolisation group (3.8% *vs.* 17.0%). There were no complications related to IMA embolisation or re-interventions associated with T2EL.[457]

A systematic review and meta-analysis on pre-emptive non-selective aneurysm sac embolisation, including 900 patients from seven studies (one RCT and six observational studies), showed a significantly lower rate of T2EL in the embolisation group compared with the no embolisation group (OR 0.21) and a corresponding lower re-intervention rate (OR 0.15), with no differences in complication rates between groups.[458]

In a recent meta-analysis of four RCTs (three on embolisation of the AAA sac and one on embolisation of a patent IMA) including a total of 393 patients randomised, pre-emptive embolisation was associated with significantly lower odds of T2EL (OR 0.45) and sac expansion (OR 0.19), but there was no significant difference in aneurysm related mortality, overall mortality, aneurysm rupture, or T2EL related re-intervention. The risk of bias was high for all outcomes and the certainty of evidence was very low or low for all outcomes. The authors concluded that limited, low certainty data suggest pre-emptive embolisation confers no clinical benefits in EVAR.[459]

A recent Finnish study compared routine attempted IMA embolisation prior to EVAR (strategy in centre A) and leaving the IMA untouched (strategy in centre B). Of 395 patients treated at centre A, the IMA was patent in 268 (67.8%) and embolisation was performed in 164 (41.5%). Centre B treated 337 patients of which 279 (82.8%) had patent IMAs. After more than five years of follow up, there were no differences in re-intervention rates due to T2ELs (12.9% *vs*. 10.4%), sac enlargement (20.3% *vs*. 19.6%), rupture rates (2.5% *vs*. 1.0%) or conversion rates (2.1% *vs*. 1.5%). The authors concluded that routinely embolising the IMA does not yield any significant clinical benefit and should therefore be abandoned.[460]

While current evidence suggests a beneficial effect of pre-emptive embolisation of side branches on T2EL and re-intervention rate, only IMA embolisation has been associated with a reduced rate of aneurysm sac growth, and there is a lack of cost effectiveness data and as yet no evidence on the potential effect on the rupture rate.[461,462] Furthermore, additional adjunctive procedures and implantation of foreign material may expose the patient to the risk of potentially serious complications, such as infection.[463] Therefore, a higher LoE is required to support a broad change of practice. Until then, pre-emptive embolisation may be considered only in selective cases.

Recommendation 64			New
For patients undergoing endovascular repair of an abdominal aortic aneurysm, routine pre-emptive embolisation of the inferior mesenteric artery, lumbar arteries, and non-selective aneurysm sac embolisation is not indicated.			
Class	Level	References	ToE
III	B	Zhang *et al.* (2022),[456] Samura *et al.* (2020),[457] Li (2020),[458] Kontopodis *et al.* (2023)[459]	

5.3.3. Open surgical repair *vs.* endovascular aortic repair.

Several RCTs have compared open and endovascular AAA treatment in patients with suitable anatomy, including the United Kingdom Endovascular Aneurysm Repair 1 (EVAR 1) trial,[464–466] the Dutch Randomised Endovascular Aneurysm Management (DREAM) trial,[467,468] the Open vs. Endovascular Repair of Abdominal Aortic Aneurysm (OVER) trial,[469] and the Anevrysme de l'aorte abdominale, Chirurgie vs. Endoprothese (ACE) trials[470] (Table 14). They have shown a significant early survival benefit for EVAR of intact AAA. However, this benefit is lost during midterm follow up.

A meta-analysis of 2 783 individual patient data with 14 245 person years of follow up, reported data on mortality, aneurysm related mortality, and re-intervention considering the four RCTs of EVAR *vs*. OSR mentioned above.[471] In the EVAR group, total mortality was significantly lower between 0 and six months (46/1 393 *vs*. 73/1 390 deaths; pooled HR 0.61) due to a lower 30 day operative mortality, but the advantage was lost in the long term since total mortality for the two groups over the follow up period of the trials showed no significant differences. In terms of aneurysm related mortality, there was no difference between EVAR and OSR after 30 days and up to three years of follow up, but after three years the number of aneurysm related deaths was higher in the EVAR group (3 *vs*. 19 deaths). The re-intervention rate was higher in the EVAR group but not all trials reported incision related complications after OSR. When taking incisional hernias, bowel obstructions, and other laparotomy based complications into account, as was done in the Open *vs*. Endovascular Repair of Abdominal aortic Aneurysm (OVER) trial,[469] the difference in secondary interventions between groups appear much less significant than that observed in the EVAR1[464,472] or DREAM trials.[468]

The EVAR 2 trial is the only RCT evaluating frail patients not suitable for OSR, for whom EVAR was originally designed. A total of 404 patients, with an AAA ≥ 55 mm in diameter and physically ineligible for OSR were included between 1999 and 2004 in the UK.[268] There was no benefit of early EVAR (*vs*. no treatment) on AAA related or total mortality at four years of follow up, which was explained by a higher than expected peri-operative mortality (7.3%) after EVAR in this cohort of frail patients and a very high overall mortality.[464] After up to 10 years of follow up EVAR (compared with no repair) was associated with a significantly lower rate of aneurysm related

Table 14. Summary of randomised controlled trials comparing elective endovascular aortic repair (treated within the Instruction For Use of the device) and open surgical repair for abdominal aortic aneurysms.

Study	Country	Recruitment period	Patients – *n*	Main findings
EVAR-1[464–466]	UK	1999–2003	1 082	Lower peri-operative mortality after EVAR (1.7% *vs.* 4.7%) Early survival benefit lost after two years, with similar long term survival Higher aneurysm related mortality in the EVAR group after 8 years (7% *vs.* 1%), mainly attributable to secondary aneurysm sac rupture Higher re-intervention rate after EVAR
DREAM[467,468]	The Netherlands and Belgium	2000–2003	351	Lower peri-operative mortality after EVAR (1.2% *vs.* 4.6%) Early survival benefit was lost by the end of the first year, with similar long term survival (38.4% *vs.* 41.7% after 12–15 year follow up) Higher re-intervention rate after EVAR (86.4% *vs.* 65.1%)
OVER[469]	USA	2002–2008	881	Lower peri-operative mortality after EVAR (0.5% *vs.* 3%) Early survival benefit sustained up till three years but not thereafter No difference in re-intervention rate No difference in quality of life No difference in cost and cost effectiveness
ACE[470]	France	2003–2008	316	No difference in peri-operative mortality rate (1.3% *vs.* 0.6%) No difference in long term survival up till three years Higher re-intervention rate after EVAR (16% *vs.* 2.4%)

EVAR-1 = the United Kingdom Endovascular Aneurysm Repair 1 trial; DREAM = the Dutch Randomised Endovascular Aneurysm Management trial; OVER = the Open vs. Endovascular Repair of Abdominal Aortic Aneurysm trial; ACE = the Anevrysme de l'aorte abdominale trial Chirurgie vs. Endoprothese.

mortality but also with higher rates of complications and re-interventions and no difference in all cause mortality. During eight years of follow up, EVAR was considerably more expensive than no repair.[472] At long term 15 year follow up, focusing on the remaining surviving original EVAR 2 cohort representing a subgroup of fitter patients than the overall EVAR 2 cohort, yet deemed unfit for OSR (at that time), there was a significantly lower aneurysm related mortality in the EVAR group, but due to a high overall mortality no difference in overall life expectancy was noted. The authors concluded that EVAR does not increase overall life expectancy in patients ineligible for open repair but may reduce aneurysm related mortality.[270,465] A recent propensity score matched study, including 350 patients with poor cardiopulmonary exercise test metrics deemed unfit for OSR, however, suggested EVAR may offer a survival advantage in selected patients. The one, three, and five year mortality in the EVAR group was 7%, 40%, and 68%, respectively, compared with 25%, 68%, and 82% in the conservative management group, all $p < .001$. Furthermore, the EVAR strategy was cost effective, with an incremental cost effectiveness ratio of €10 000 per quality adjusted life year gained.[473]

A recent meta-analysis, including 21 490 patients with high risk AAA from 27 studies, found a significantly lower peri-operative mortality after EVAR compared with OSR (OR 0.64). The early survival benefit was, however, lost during follow up, and the authors concluded that an endovascular strategy may be preferable over open repair in an elderly and frail population with limited physiological reserve and life expectancy, in whom a good early result is more important than long term outcome. The lack of a widely accepted definition of high risk, however, makes it difficult to interpret the significance of different study results in today's clinical practice.[474]

In the OVER trial, the only RCT evaluating cost and cost effectiveness, no difference was seen between EVAR and OSR.[469] This was confirmed in a model study from The Netherlands.[475] A systematic review noted, however, that previous published cost effectiveness analyses of EVAR do not provide a clear answer about whether elective EVAR is a cost effective solution and calls for cost effectiveness analysis of EVAR that incorporates more recent technological advances and the improved experience that clinicians have with EVAR,[476] and a recent systematic review pointed out that as health systems vary among different countries, generalising health economic results should be done with caution.[477]

Owing to the rapid technological and medical developments, the existing RCTs comparing OSR and EVAR are partly outdated and thereby not entirely relevant for today's situation. Devices used in the RCTs were mainly first or second generation EVAR devices. Other factors of potential importance are improvements in pre-operative imaging and planning, the transition from general anaesthesia to percutaneous techniques under local anaesthesia, the rapid technical development of intra-operative imaging systems and the evolution of post-operative management. It is therefore necessary to include more recent observational and registry data in the overall evaluation. Thus, despite data from multiple RCTs and meta-analysis, representing the highest LoE, the existing LoE is rated as mediocre (Level B). Results of most recent meta-analysis comparing elective EVAR and open surgical repair (OSR) for AAA are reported in Table 15.

Recent large population based registry studies from Europe and the USA have shown a sustained increased utilisation of EVAR with a continued decrease in mortality and morbidity, despite older and sicker patients being treated.[10,11,15,19,37,484] The contemporary 30 day mortality after elective EVAR is around 1%, compared with a three to five times higher mortality after OSR.[11,484,485] The improved short term survival is sustained for at least five years.[10,11,486]

Table 15. Summary of meta-analysis comparing elective endovascular aortic repair and open surgical repair for abdominal aortic aneurysms.

Author	Study type included	Recruitment period	Patients – *n*	Main findings
Powell *et al.* (2017)[471]	4 RCTs	1999–2008	2783	Lower all cause mortality after EVAR within six months (3.3% *vs.* 5.3%, HR 0.61), thereafter no difference No difference in AAA related mortality between 30 days and three years, thereafter higher in the EVAR group Higher re-intervention rate after EVAR, but when taking laparotomy based complications into account, as was done in the OVER trial, the difference was less significant
Giannopoulos *et al.* (2020)[478]	5 RCT	1998–2008	2823	No difference in all cause mortality or AAA related mortality after 4–8 and > 8 years follow up Higher re-intervention rate after EVAR (29% *vs.* 15%)
Antoniou *et al.* (2020)[479]	7 RCT	1999–2011	2983	Lower all cause mortality within 30 days (OR 0.36) and six months (HR 0.62) after EVAR Lower AAA related mortality within six months after EVAR (HR 0.42), but higher after > 8 years follow up (HR 5.12) Higher re-intervention rate (HR 2.13), aneurysm rupture (OR 5.08) and death due to rupture (OR 3.57) after > 8 years after EVAR
Bulder *et al.* (2019)[480]	4 RCT, 20 REG, 29 CS	1993–2015	189 022	Lower 30 day all cause mortality after EVAR (1.2% *vs.* 3.2%), thereafter no difference
Li *et al.* (2019)[481]	3 RCT, 68 CS	1999–2018	299 784	Higher all cause mortality (OR 1.19), re-intervention (2.12), and secondary rupture rate (OR 2.47) after 5–9 years follow up after EVAR No difference in all cause mortality, but higher re-intervention rate (OR 2.47) and secondary rupture rate (OR 8.10) after EVAR after > 10 years follow up (up to 15 years)
Yokoyama *et al.* (2020)[482]	4 RCT, 7 PSS	1999–2016	106 243	Lower peri-operative all cause mortality after EVAR (RR 0.39), no difference between 0 and two years, higher between two and six years after EVAR (HR 1.15), and no difference between six and 10 years or ≥10 years
Alothman *et al.* (2020)[483]	4 RCT, 12 CS	2004–2017	61 379	Lower peri-operative all cause mortality after EVAR (1.2% *vs.* 4.5%, thereafter no difference No difference in aneurysm related mortality, higher rate of late aneurysm sac rupture after EVAR (1.8% *vs.* 0.4%) and of re-intervention (OR 1.94)

REG = registries; CS = cohort studies; PSS = propensity score matched studies; RCT = randomised controlled trial; EVAR = endovascular aneurysm repair; AAA = abdominal aortic aneurysm; HR = hazard ratio; OR = odds ratio; OVER = the Open vs. Endovascular Repair of Abdominal Aortic Aneurysm trial.

Also, a marked reduction in operating time, surgical complications, and ICU and hospital length of stay after EVAR have been observed in recent years[484,487] and when comparing stent grafts introduced after 2004 with those used prior to that time, the newer stent grafts have performed substantially better in terms of long term rates of re-intervention, conversion, and AAA growth.[488] In a descriptive comparison of the results between the EVAR 1 trial and a more recent observational non-randomised prospective registry ENGAGE, freedom from all cause mortality was 74.4% in the EVAR 1 trial and 74.6% in the ENGAGE registry through the four year time point, the aneurysm related mortality was 4.2% *vs*. 1.9%, death due to rupture was 1.6% *vs*. 0.5%, and re-intervention rate was 19.3% *vs*. 10.9%.[489]

The evidence from RCTs is predominantly applicable to AAA patients younger than 80 years, whereas today the greatest increases in AAA repair appear to be in those over 80 years.[10,11,485] This group has also seen the most pronounced improvement in outcome after AAA repair, which is probably to be related to the preferential use of EVAR for treatment of octogenarians. In a nationwide Swedish study the 30 day mortality rate after elective AAA repair among octogenarians was 2%, of which 80% were treated by EVAR.[11] In a report from the VQI database in the USA the 30 day and one year mortality rates after elective EVAR in octogenarians were 1.6% and 6.2% respectively. The corresponding mortality rate after OSR was 6.7% and 11.9% respectively.[490] Data from the ENGAGE registry suggest that octogenarians treated by EVAR have a higher incidence of complication with longer hospital stay and a longer than expected recovery time (> 12 months) than younger patients.[491] In a recent analysis of the American College of Surgeons NSQIP database including 12 267 EVAR procedures performed between 2011 and 2017, age was identified as a predictor of 30 day death. However, this difference disappeared after adjustment for comorbidities in a propensity score matched analysis, suggesting age alone should not exclude patients from EVAR.[492] Similar findings, that comorbidity rather than age is important, were seen in the Dutch Surgical Aneurysm Audit.[38] In a systematic review, elderly patients (80 – 90 years old) with low surgical risk had a significantly lower 30 day mortality rate after EVAR than OSR (2.1% *vs*. 8.8%; RR 0.25).[493] Against this background, it is reasonable to consider elective AAA repair of patients over 80 years with reasonable life expectancy and QoL, having been informed about the various treatment options (see Chapter 11 on SDM). This information, from modern cohort and registry studies indicating that EVAR can be offered to more patients today with improved results, is an important supplement to that from older RCTs when evaluating operating techniques today.

The choice of surgical technique should be discussed between the treating clinician and the patient and multiple factors should be considered when individualising a patient treatment plan. These include (1) anatomical suitability for EVAR, (2) physiological reserves and fitness for surgery, (3) life expectancy, (4) patient preferences, and (5) needs and expectations, including the importance of sexual function, and anticipated compliance with frequent lifelong surveillance and follow up. It is therefore not possible to provide detailed recommendations and is important to allow freedom for individualised decision making, respecting the patient's choice whenever possible[494,495] (see also Chapter 11).

Nearly all the evidence suggests a significant short term survival benefit for EVAR over OSR. Yet, there are indications that an increased rate of complications may occur after 8 — 10 years with earlier generation EVAR devices and uncertain durability of current devices, particular the low profile devices (see section 5.3.2.1). Thus, although EVAR should be considered the preferred treatment modality in most patients, it is reasonable to consider an OSR first strategy in younger, fit patients with a long life expectancy > 10 — 15 years. The normal (average) survival after elective AAA repair is about nine years.[486] Conversely, elective AAA repair is not recommended in patients with limited life expectancy, e.g., in patients with terminal cancer or severe cardiac failure. A pragmatic definition of limited life expectancy is less than two to three years.

It should be noted that this chapter refers to patients with an asymptomatic infrarenal AAA undergoing elective repair. Importantly, the present concepts should not be used to deduce recommendations for other situations.

Recommendation 65		Unchanged
For most patients with suitable anatomy and reasonable life expectancy, endovascular repair should be considered the preferred treatment modality for elective abdominal aortic aneurysm repair.		
Class	**Level**	**References ToE**
IIa	**B**	Lilja *et al.* (2017),[11] Beck *et al.* (2016),[15] Mani *et al.* (2015),[19] Budtz-Lilly *et al.* (2018),[44] Greenhalgh *et al.* (2004),[464] van Schaik *et al.* (2017),[468] Lederle *et al.* (2009),[469] Powell *et al.* (2017),[471] Greenhalgh *et al.* (2010),[472] Giannopoulos *et al.* (2020),[478] Antoniou *et al.* (2020),[479] Bulder *et al.* (2019),[480] Li *et al.* (2019),[481] Yokoyama *et al.* (2020),[482] Yin *et al.* (2019),[484] Verzini *et al.* (2014),[488] Hicks *et al.* (2016),[490] Reise *et al.* (2010),[494] Faggioli *et al.* (2011),[495] Trenner *et al.* (2018)[496]

Recommendation 66		Unchanged
For most patients with long life expectancy, open surgical repair should be considered as the preferred treatment modality for elective abdominal aortic aneurysm repair.		
Class	**Level**	**References ToE**
IIa	**B**	Greenhalgh *et al.* (2004),[464] van Schaik *et al.* (2017),[468] Lederle *et al.* (2009),[469] Becquemin *et al.* (2011),[470] Powell *et al.* (2017),[471] Giannopoulos *et al.* (2020),[478] Antoniou *et al.* (2020),[479] Bulder *et al.* (2019),[480] Li *et al.* (2019),[481] Yokoyama *et al.* (2020)[482]

Recommendation 67		Unchanged
For patients with limited life expectancy, elective abdominal aortic aneurysm repair is not recommended, either open or endovascular repair.		
Class	**Level**	**References ToE**
III	**B**	Greenhalgh *et al.* (2010)[268]

5.3.4. Laparoscopic aortic repair. Laparoscopic aortic surgery is suggested as a less invasive alternative to OSR when EVAR is not indicated.[497,498] Laparoscopic techniques for the treatment of AAA include a total laparoscopic approach, a laparoscopic assisted surgical approach (laparoscopic dissection with endo-aneurysmorrhaphy via mini-laparotomy), or a hand assisted laparoscopic approach, or a robot assisted laparoscopic approach. This technique is technically demanding and requires an extensive experience in laparoscopic surgery.[499] In a prospective comparative multicentre study, laparoscopic aortic surgery was associated with a significantly higher risk of death and adverse events compared with OSR, despite a highly experienced laparoscopic surgical team.[500]

Recommendation 68		Unchanged
Laparoscopic abdominal aortic aneurysm repair is not recommended.		
Class	**Level**	**References ToE**
III	**C**	Economopoulos *et al.* (2013),[499] Ricco *et al.* (2016)[500]

5.4. Peri-operative complications after elective abdominal aortic aneurysm repair

Elective EVAR and OSR of AAA are procedures with a high risk of major complications. In an international Delphi consensus study among vascular surgeons, MI, stroke, renal failure, bowel ischaemia, peripheral thromboembolism requiring minor or major amputation, infection and spinal cord ischaemia (SCI) were agreed upon as major complications after EVAR and OSR[501] In a joint analysis of VASCUNET and the International Consortium of Vascular Registries (ICVR) of 60 273 elective procedures between 2010 and 2016, the risk of bleeding, stroke, renal replacement therapy, respiratory failure, and bowel ischaemia after EVAR were below 1%, whereas the prevalence of a cardiac event was 3.0%. All the aforementioned complications were more prevalent after OSR (1 – 3%), with respiratory failure (5.7%) and cardiac event (8.9%) being the most common.[39] In a systematic review, the risk of major complications after elective EVAR and OSR was consistently and significantly higher for women than men.[263]

Delay in timely recognition and management of complications (failure to rescue) is the principal determinant of peri-operative mortality after OSR and EVAR.[502] Although bowel ischaemia is a rare complication after elective EVAR (0.3%) and OSR (2.0%), it carries a very high risk of death (43.6% and 43.4%, respectively). Prompt recognition of bowel ischaemia is of the utmost importance, yet often difficult. Pain, metabolic acidosis, and oliguria should raise awareness of bowel ischaemia. CTA may be helpful to confirm patency of the visceral arteries but is often late at recognising bowel ischaemia, and sigmoidoscopy may add to establishing the diagnosis, yet the clinical value of both modalities is uncertain since the positive predictive value is low with the low *a priori* probability of bowel ischaemia. Post-operative bleeding with the need to return to theatre occurred in 2.2%, but had a 28.3% risk of failure to rescue after OSR.[39] In a study of 9 719 elective procedures in the US, bowel ischaemia and return to the OR for bleeding were also the main drivers of failure to rescue.[503]

On an aggregate level, there seems to be a clear association between hospital volume and failure to rescue. In the VASCUNET/ICVR study the highest volume hospitals significantly less often had failure to rescue after OSR and EVAR than the lowest volume hospitals, OR 0.22 and 0.54, respectively.[39] The outcomes for OSR are supported by data from the SVS-VQI, with an OR for failure to rescue of 0.48 for centres performing $>$ 10 OSR per annum *vs*. those performing $\leq$ three procedures. Patients were also twice as likely to die within 30 days (3% $>$ 10 procedures *vs*. 6% $\leq$ six procedures).[503]

Despite the clear volume–outcome association for failure to rescue, little is known about the contribution of specific process and structural indicators to better outcomes. One obvious explanation may be the prompt recognition of a major complication and subsequent action to mitigate negative effects. Local resources and policy may influence the ICU admission selection process, but usually all patients undergoing OSR and high risk EVAR patients should be offered ICU surveillance for advanced monitoring and early detection and management of complications. Furthermore, in view of the high risk of cardiac complications, 24/7 access to coronary catheterisation is important in any hospital that performs AAA repair.[39,276]

Recommendation 69		New
All patients with an abdominal aortic aneurysm undergoing open surgical repair and high risk patients undergoing endovascular repair are recommended to have early post-operative monitoring in an intensive care or high dependency unit.		
Class	**Level**	**References**
IIa	C	Consensus

Early or enhanced recovery after surgery (ERAS) programmes have been designed to accelerate the post-operative recovery of surgical patients by reducing the surgical stress response.[504] ERAS involves an integrated, multidisciplinary common pathway including thorough pre-operative counselling to prepare the patient mentally and physically, the use of epidural anaesthesia and minimised surgical access, optimal pain control with the avoidance of side effects, early post-operative mobilisation and oral nutrition as well as the avoidance (or early removal) of drains and urethral catheters. Data from cohort studies suggest that ERAS may be feasible and potentially beneficial in open AAA surgery, however, the design of the ERAS AAA program and its place in clinical practice is yet to be defined.[505–508]

6. MANAGEMENT OF RUPTURED AND SYMPTOMATIC ABDOMINAL AORTIC ANEURYSM

Distinction between symptomatic and rAAA is crucial because results differ significantly between the two groups. A rAAA is defined as an acute haemorrhage from the AAA outside the true aortic wall with the presence of retroperitoneal and or intraperitoneal blood. A contained rAAA is when the haematoma is temporarily sealed by the retroperitoneum. Symptomatic AAAs are those presenting with abdominal and or back pain, a tender AAA on palpation, or embolic events, but without breach of the aortic wall.

6.1. Pre-operative evaluation

6.1.1. Clinical and radiological evaluation. The classical triad of hypotension, abdominal, and or back pain, and a pulsatile abdominal mass are present in about 50% of patients with a rAAA. Misdiagnosis may occur in 32 – 39% of patients, particularly in those presenting without haemodynamic shock.[509–511] A systematic review identified ureteric colic and MI as being the most common erroneous differential diagnoses.[511]

Emergency US may be useful in identifying the presence of an AAA, but its sensitivity to detect retroperitoneal haemorrhage is low.[512] As a result, US cannot be used to identify a leak; however, the presence of an AAA in an unstable patient is very suggestive of a rAAA. In the endovascular era, another drawback of US is that it lacks information about anatomical suitability for EVAR. Therefore, an immediate CTA of the thoraco-abdominal aorta and iliac vessels is the key imaging modality for all patients with suspected rAAA.[122,513]

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Most patients with a rAAA who reach the hospital alive are sufficiently stable to undergo CTA to confirm the diagnosis and to plan for OSR or EVAR.[509,514–518] Haemodynamic instability is defined as loss, or reduced level of consciousness or systolic BP < 80 mmHg.[519–521] Circulatory instability is however relative, and in most situations it is both preferable and feasible to conduct a CTA. In a review and meta-analysis, EVAR for patients with a haemodynamically unstable rAAA was associated with a significantly decreased in hospital mortality compared with OSR (37% *vs*. 62%).[522]

If, however, the patient is too unstable, he or she may be transported directly to the operating room for emergency OSR or intra-operative imaging for confirmation of the diagnosis and potentially determination of the suitability for EVAR. An intra-operative aortogram, with or without an aortic occlusion balloon (AOB), may be an emergency compromise solution for determination of initial EVAR eligibility and device selection, with subsequent measurements obtained either by DSA or IVUS.[523]

Recommendation 70		Changed
Patients with a suspected ruptured abdominal aortic aneurysm should undergo prompt imaging of the thoraco-abdominal aorta and of the access vessels with computed tomography angiography.		
Class	**Level**	**References** ToE
I	B	Smidfelt *et al.* (2017),[509] Lloyd *et al.* (2004),[514] Boyle *et al.* (2005),[515] Reimerink *et al.* (2013),[516] Starnes *et al.* (2010),[517] IMPROVE Trial Investigators (2017)[518]

6.1.2. Aortocaval fistula. An aortocaval fistula (ACF) occurs when an AAA ruptures into the inferior vena cava (IVC), and is seen in 2 – 6% of all rAAA.[524,525] Unlike standard rAAA, most ACFs present without signs of massive bleeding and retroperitoneal haematoma because the aortic rupture shunts directly into the IVC, but rather with symptoms from rapid arteriovenous shunting and secondary venous hypertension.[526] In a series of 50 consecutive cases of rAAA with ACF shock, congestive heart failure, pelvic, and lower extremity venous hypertension were present in 48%, 26%, and 75% respectively.[527] The diagnosis is confirmed pre-operatively by CTA, with early caval opacification in the arterial phase.[528]

During OSR for ACF, the fistula is closed surgically from the inside of the aneurysm sac. The aneurysm is thereafter repaired in the usual way. Although EVAR excludes the aneurysm from the circulation, it does not control the fistula itself, which is left open as a persistent communication between the aneurysm sac and the IVC. In the presence of a T2EL, this might pose a particular management problem and whether and how to treat the persistent fistulas is a matter of debate with different treatment strategies described. In case of persistent endoleak (> six months) associated with aneurysm sac enlargement, increased cardiac output and heart failure, and rarely, pulmonary embolisation, closure of the fistula is suggested, either by direct embolisation, plugging or stent grafting of the IVC.[526,529,530] if there is no endoleak and or favourable aneurysm sac remodelling, conservative management has been suggested.[526,530] In a systematic review, summarising data from 110 case reports with 196 patients, the 30 day survival was 87.5% after OSR and 97.6% after EVAR, and after a median of 14 month follow up 86% and 95%. After EVAR, 40% showed an endoleak, most often T2EL, and the re-intervention rate was 35.7% (compared with 2.5% after OSR).[529]

Recommendation 71		New
After endovascular repair of abdominal aortic aneurysm rupture into the inferior vena cava, subsequent endovascular closure of the aortocaval fistula may be considered in the presence of an endoleak associated with increased cardiac output, heart failure, or pulmonary embolisation.		
Class	**Level**	**References**
IIb	C	Consensus

6.2. Peri-operative management

6.2.1. Permissive hypotension and transfusion protocol. There is considerable evidence that vigorous fluid replacement, known as the normotensive resuscitation strategy, may exacerbate bleeding and prejudice outcome. On the other hand, a permissive hypotension resuscitation strategy (otherwise known as hypotensive haemostasis or delayed volume resuscitation) refers to a policy of delaying aggressive fluid resuscitation until proximal aortic control is achieved.[531,532] This may limit excessive haemorrhage by allowing clot formation and avoiding development of iatrogenic coagulopathy. Although there are several published animal and human studies on the beneficial role of permissive hypotension in trauma, no direct comparative study exists on permissive hypotension *vs*. normotensive resuscitation strategies in the management of haemorrhagic shock in patients with rAAA.[532,533] Nevertheless, nowadays permissive hypotension is considered a safe, well documented, and widespread practice in the management of patients with rAAA.[531,534–540] Preferentially, resuscitation efforts should be managed with the administration of blood and blood products with a suggested fresh frozen plasma to red blood cell ratio close to 1:1.[541–543] A step further is a policy of actively lowering BP using pharmacological agents. Some authors use the term hypotensive haemostasis to describe this active management and distinguish it from permissive hypotension, the latter being more of a passive process of not responding to hypotension, as long as the patient remains conscious and stable albeit hypotensive. A Dutch study evaluated the feasibility of a protocol of

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hypotensive haemostasis using intravenous nitrates.[539] The aim was to limit pre-hospital intravenous fluid administration to 500 mL and to maintain systolic BP between 50 and 100 mmHg. The desired systolic BP range was reached in 46% of cases, whereas in 54%, a systolic BP > 100 mmHg was recorded for > 60 minutes. To date, whether pharmacological lowering of BP is beneficial remains unclear.[539]

Equally, the ideal BP that is allowed for permissive hypotension is debatable. There is increasing data that BP targets in elderly patients should not be as low as in fit young trauma patients (e.g., soldiers) although most of the data for permissive hypotension was generated from this young group. In the Immediate Management of Patient with Ruptured Aneurysm: Open vs. Endovascular Repair (IMPROVE) trial, the lowest systolic BP was independently associated with the 30 day mortality rate and it was suggested that a minimum systolic BP of 70 mmHg may be too low a threshold for permissive hypotension in patients with rAAA.[544] Nevertheless, the recommendation to implement a policy of permissive hypotension provided the patient remains conscious, with a target systolic pressure 70 − 90 mmHg, remains, but the evidence has been reassessed and downgraded to Level C.

For coagulation resuscitation with blood products and coagulation factors, please consult established principles of massive transfusion and local guidelines. Other measures that contribute to keeping BP down are adequate pain management.

Recommendation 72		**Changed**	
For patients with a ruptured abdominal aortic aneurysm, a policy of permissive hypotension is recommended.*			
Class	Level	References	ToE
I	C	Dick *et al.* (2013),[531] Moreno *et al.* (2018),[533] Hechelhammer *et al.* (2005),[534] Mayer *et al.* (2009),[536] Ohki *et al.* (2000),[537] Roberts *et al.* (2006),[538] van der Vliet *et al.* (2007),[539] Veith *et al.* (2002),[540] Powell *et al.* (2014)[545]	

* E.g., by restricting fluid resuscitation, with consciousness and ability to speak as appropriate markers of adequate cerebral perfusion.

6.2.2. Anaesthesia. OSR requires general anaesthesia to approach the rAAA via a midline transperitoneal or, less often, a left retroperitoneal incision.[546] Close cooperation between the anaesthetic and surgical teams is needed, since vasodilation on induction will often lead to sudden hypotension. Therefore, the surgical team should be scrubbed up and gowned, the surgical field should be prepped and draped, and all should be ready to start the operation prior to the induction of anaesthesia. This is important to minimise delays and to control bleeding rapidly.

In contrast to OSR, EVAR can be performed under local anaesthesia, supplemented, if needed, by intravenous sedation.[547] Local anaesthesia has been advocated to prevent circulatory collapse caused by the induction of general anaesthesia and to promote peritoneal tamponade. Common reasons for conversion to general anaesthesia are loss of consciousness during the operation because of severe hypovolaemic shock, severe discomfort from rupture, endovascular instrumentation of the aorta and iliac arteries, need for iliac artery access, and creation of a femorofemoral bypass after deployment of an aorto-uni-iliac (AUI) stent graft.[519,548–551] Movement artefacts due to patient discomfort have been reported to be the reason for suboptimal stent graft deployment and inadvertent coverage of the renal arteries or more distal placement of the main body of the device. As a result, not all operators share the same enthusiasm for local anaesthesia.[550,552] Nevertheless, in recent years, with growing experience and given that the use of local anaesthesia for EVAR in rAAAs has been associated with improved survival, this type of anaesthetic approach has been adopted widely.[519,553–558] In a *post hoc* analysis of the IMPROVE trial, patients who received EVAR under local anaesthesia alone had a greatly reduced 30 day mortality rate compared with those who were treated under general anaesthesia.[544] In a VQI study, including 3 330 patients with rAAA, those treated by EVAR under local anaesthesia (*vs.* general anaesthesia) had significantly lower mortality rates at 30 days (15.5% *vs.* 23.3%, HR 0.7) and at one year (22.5% *vs.* 32.3%, HR 0.7).[557] In a recent meta-analysis, including 4 336 patients from 10 cohort studies, EVAR under local anaesthesia (*vs.* general anaesthesia) was associated with a significantly lower peri-operative mortality rate (17.3% *vs.* 26.4%, OR 0.49).[559] The survival benefit was greatest in haemodynamically stable patients.[557,559]

Recommendation 73		**Unchanged**	
For patients undergoing endovascular repair of a ruptured abdominal aortic aneurysm, local anaesthesia should be considered as the anaesthetic modality of choice, whenever tolerated by the patient.			
Class	Level	References	ToE
IIa	B	Karkos *et al.* (2008),[519] Powell *et al.* (2014),[545] Bellamkonda *et al.* (2021),[553] Bennett *et al.* (2019),[554] Chen *et al.* (2019),[555] Deng *et al.* (2021),[556] Faizer *et al.* (2019),[557] Mouton *et al.* (2019),[558] Lei *et al.* (2022)[559]	

6.2.3. Aortic occlusion balloon. Previous studies have demonstrated that approximately one third of patients with a rAAA undergoing EVAR are haemodynamically unstable and one in four experience complete circulatory collapse.[551,560,561] Proximal aortic control during OSR is achieved by either infrarenal aortic cross clamping or suprarenal or supracoeliac clamping followed by repositioning of the clamp to an infrarenal position as soon as feasible. Proximal aortic control can also be achieved by an endovascular AOB (resuscitative endovascular balloon occlusion of the aorta) during EVAR or as an alternative to conventional aortic cross clamping in haemodynamically unstable patients undergoing OSR.[562] This can be achieved by a transfemorally placed AOB supported by a long sheath in the supracoeliac aorta or through a transbrachial approach.[563–565]

Few reports on the effect of AOB related to open rAAA repair exist.[523,565,566] One study showed that, compared with conventional aortic clamping, AOB was associated with reduced intra-operative mortality, but not in hospital mortality.[523] Concerning those undergoing EVAR, a meta-analysis of 39 studies documented that a total of 200 of 1277 patients (14.1%) required AOB. Death was significantly lower in studies with a higher rate of AOB use, suggesting that the use of an AOB in patients with unstable rAAA undergoing EVAR may improve the results.[567]

Although AOB has been shown to improve haemodynamic parameters, the evidence base is weak with no clear reduction in haemorrhage related death. Formal, prospective study is warranted to clarify its role in the rAAA setting.[568,569] The GWC therefore decided to downgrade the recommendation on its use. Finally, when faced with a rAAA patient in circulatory collapse, some advocate placement of an AOB blind in the emergency room. However, whether such a manoeuvre is beneficial or safe remains to be proven, and until then, is not advised.

Recommendation 74			Changed
Haemodynamically unstable patients with a ruptured abdominal aortic aneurysm undergoing open or endovascular repair may be considered for aortic balloon occlusion under fluoroscopy guidance to obtain proximal control.			
Class	Level	References	ToE
IIb	C	Hechelhammer *et al.* (2005),[534] Mayer *et al.* (2009),[536] Ohki *et al.* (2000),[537] Veith *et al.* (2002),[540] Lachat *et al.* (2002),[547] Karkos *et al.* (2011)[551] Veith *et al.* (2009),[561] Bath *et al.* (2018),[565] Karkos *et al.* (2015)[567]	

6.2.4. Graft configuration. During OSR the diseased aortic segment is replaced by a prosthetic Dacron or ePTFE graft in a tube or bifurcated configuration in the same way as in elective repair (see Chapter 5).

Both AUI and bifurcated device configurations have been successfully used in EVAR for rAAAs.[516,560,570,571] The choice of a bifurcated over an AUI stent graft in the rAAA setting depends on the expertise and preference of the operator, stent graft availability, aneurysm anatomy and haemodynamic status of the patient.[551,560,571] A bifurcated option is more anatomically suited and avoids a femorofemoral bypass, but a drawback may be the time taken to cannulate the contralateral limb. The latter is a crucial factor in patients with rAAA, and any delay in excluding the aneurysm may have a negative impact on survival. The AUI approach is easier and quicker, has a higher eligibility rate, requires fewer stent grafts in stock, but also requires a femorofemoral graft. The latter has the disadvantages of an extra-anatomical bypass plus the fact that local anaesthesia may need to be converted to general anaesthesia. Furthermore, single centre reports have suggested that a bifurcated stent graft may be associated with a lower mortality rate than AUI devices [547,551,570,572] and the IMPROVE trial has suggested that graft infection rates are lower with bifurcated devices.[573] Thus, a bifurcated device, in preference over an AUI device, may be considered whenever anatomically suitable. It is also advised that the devices used for rAAAs should be the ones that the operator and the team routinely use in elective EVAR and with which the team has significant experience.

An important technical aspect of emergency EVAR is the degree of stent graft oversizing in the presence of existing hypovolaemia. The haemodynamic condition of the patient on presentation may influence this and, to avoid an intra-operative or late Type Ia or Ib endoleak, 30% oversizing is preferable when treating a rAAA assessed by CTA performed during permissive hypotension.[574]

Recommendation 75			Changed
Patients undergoing endovascular repair for a ruptured abdominal aortic aneurysm may be considered for a bifurcated device, in preference to an aorto-uni-iliac device, whenever anatomically suitable.			
Class	Level	References	ToE
IIb	C	Mayer *et al.* (2009),[536] Powell *et al.* (2014),[545] Karkos *et al.* (2011),[551] Karkos *et al.* (2014),[570] Carrafiello *et al.* (2012),[575] Rokosh *et al.* (2023)[576]	

Recommendation 76			New
Patients undergoing endovascular repair for a ruptured abdominal aortic aneurysm in whom imaging was performed during permissive hypotension, should be considered for stent graft oversizing of up to 30%.			
Class	Level	References	ToE
IIa	C	van der Riet *et al.* (2022)[574]	

6.2.5. Peri-operative anticoagulation. Whether to give intravenous heparin intra-operatively is a matter of debate.

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Although this is a universal policy during elective AAA repair, the intra-operative administration of intravenous heparin during open or endovascular rAAA repair remains controversial. The risk of exacerbating bleeding should be balanced against the benefits of the thromboembolic protection provided by heparin.[577,578] Regardless of whether systemic anticoagulation is used at the onset, serious consideration should be given to heparin administration and systemic anticoagulation should be considered during EVAR as soon as the aneurysm has been fully excluded (with the delivery system and sheaths still in place) or if proximal aortic control with an AOB or clamp has been accomplished. Intravascular thrombosis requiring thrombectomy or open conversion may be needed if anticoagulation is withheld throughout the entire procedure.

According to the American College of Chest Physicians, patients undergoing repair of a rAAA are categorised as high risk for DVT,[579] but are also at increased risk of major bleeding. Therefore, when considering DVT prophylaxis, the DVT risk should be weighed against the bleeding risk. A reasonable approach is to use mechanical prophylaxis with sequential compression devices until the risk of major bleeding has subsided. Once the high risk of major bleeding has subsided, pharmacological prophylaxis with either LMWH or unfractionated heparin can be started. In most patients, this can be safely initiated within 24 − 48 hours of surgery unless there are signs of ongoing bleeding or a clinically significant coagulopathy. This should be continued throughout the hospital stay and continued in selected patients after discharge based on individual risk factors and level of mobilisation.[579]

Recommendation 77		New
In ruptured abdominal aortic aneurysm repair, intra-operative administration of systemic anticoagulation with heparin should be considered once the rupture bleeding has been controlled.		
Class	**Level**	**References**
IIa	C	Consensus

Recommendation 78		New
Patients with a ruptured abdominal aortic aneurysm should be considered for post-operative deep vein thrombosis prophylaxis with low molecular weight or unfractionated heparin unless there are signs of ongoing bleeding or of a clinically significant coagulopathy.		
Class	**Level**	**References**
IIa	C	Consensus

6.2.6. Non-operative management and palliation. Patients deemed unlikely to survive surgery may be turned down and managed palliatively. Non-intervention rates vary significantly across countries with some surgeons or centres being very selective and others adopting an all comer policy.[580,581] The decision to withhold treatment in patients who have a low chance of survival is difficult. Clinical judgements usually have to be made quickly, and a decision to operate is often taken despite a low chance of success. To predict futility of open or endovascular repair for rAAA and select patients for palliation, different scoring systems and algorithms have been developed. Modern mortality risk stratification scores, such as the Vascular Study Group of New England score, the Dutch Aneurysm Score, and the Harborview Medical Centre score, all seem to accurately predict real world post-operative mortality after rAAA.[582−585] The latter has the advantage that it solely relies on pre-operative variables: age $>$ 76 years, pH $<$ 7.2, creatinine $>$ 2 mg/dL ($>$ 177 μmol/L), and any episode of hypotension (systolic BP $<$ 70 mm Hg). As a result, the Harborview Medical Centre score may be practical when discussing the treatment options with referring physicians, patients, and their family members to help guide transfer and treatment decision making. Nevertheless, clinical decision making on withholding treatment or opting for palliation based entirely on a scoring system is not recommended.[582−585]

Single centre or multicentre series, registry data and meta-analyses suggest that good or at least acceptable results can also be achieved in patients aged $>$ 80 years.[486,581,586−592] A meta-analysis of 36 studies published before 2010, showed an immediate post-operative mortality rate of 59% in patients $>$ 80 years old. Furthermore, intermediate survival data from six studies were available on 111 operation survivors with one, two, and three year pooled survival rates of 82%, 76%, and 69%, respectively.[586] Pooling data from eight modern series including 7 526 octogenarians published between 2013 and October 2018 reported a 30 day mortality rate of 43% and one year mortality rate of 47%, i.e., figures similar to the outcome at all ages.[587] Swedvasc data also showed that octogenarians surviving the initial 90 days had surprisingly good long term survival ($>$ 50% after five years), which is only slightly less than the general population.[593] In a single centre study from Switzerland, mortality of rAAA treated mainly with OSR was not independently related to advanced age but mainly driven by cardiac disease and manifest hypovolaemic shock, with an almost normal long term prognosis.[594] Taking QoL into account, it is encouraging that half of the octogenarians in the Dutch multicentre study were still alive one year after rAAA repair and $>$ 80% returned to their pre-rupture home situation.[587] These data justify a more confident approach to repair of rAAA in the elderly and patients should therefore not be denied treatment based on age alone.[486,581,586,587]

Finally, if cardiopulmonary resuscitation (CPR) is required before repair, mortality rates may approach 100%. So, should CPR be continued and repair offered, or should these patients be treated non-operatively? A multicentre

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study in 176 surgically treated patients with rAAA from The Netherlands concluded that a rAAA following pre-operative CPR is not necessarily a lethal combination.[595] Thirteen of these 176 patients (7.4%) needed CPR. Two CPR patients treated by EVAR survived, whereas survival in the 11 CPR patients who underwent OSR was 27% (three of 11). Therefore, patients with rAAA needing CPR should not necessarily be denied intervention. However, it is reasonable to adopt a restrictive and selective approach in this highly vulnerable patient group knowing the often dismal outcome.

Recommendation 79		Unchanged
Selection of patients with ruptured abdominal aortic aneurysm for palliation based entirely on scoring systems or solely on advanced age is not recommended.		
Class	**Level**	**References** **ToE**
III	B	Karkos *et al.* (2008),[519] De Rango *et al.* (2016),[581] Hemingway *et al.* (2021),[582] Garland *et al.* (2018),[583] Hansen *et al.* (2019),[584] Ciaramella *et al.* (2021),[585] Biancari *et al.* (2011),[586] Roosendaal *et al.* (2020),[587] Shahidi *et al.* (2009),[588] Raats *et al.* (2014),[589] Yamaguchi *et al.* (2020),[590] Tambyraja *et al.* (2005),[596] Acosta *et al.* (2006),[597] Conroy *et al.* (2011),[598] Kurc *et al.* (2012),[599] Robinson *et al.* (2013),[600] van Beek *et al.* (2015),[601] Thompson *et al.* (2016),[602] Vos *et al.* (2016),[603] Reite *et al.* (2017),[604] von Meijenfeldt *et al.* (2017),[605] Sweeting *et al.* (2018),[606] Roosendaal *et al.* (2021)[607]

6.3. Open surgical repair vs. endovascular aortic repair

Data suggest a decreasing trend in OSR mortality for rAAA.[608] The Swedvasc registry documented a decrease in mortality from 38% to 28% between 1994 and 2010 with almost entirely OSR.[609] A collected world experience from the rAAA investigators (with data registered from 13 centres committed to EVAR whenever possible) reported 36% mortality for 763 patients (8 – 53%) who were offered OSR.[561] Furthermore, in the three RCTs on patients with rAAA, the 30 day mortality was 25 – 40.6% after OSR.[516,544,562] In the Amsterdam Acute Aneurysm Trial (AJAX) and the Endovasculaire ou Chirurgie dans les Anévrysmes aorto-iliaques Rompus (ECAR) trials, patients randomised in the OSR arm were all suitable for EVAR, whereas in the IMPROVE trial some were not, as patients were randomised prior to CTA into an endovascular strategy or an immediate OSR.

The reported peri-operative mortality rates after EVAR for rAAAs range from 13% to 53%.[536,551,560,561,610,611] In general, reported figures from observational studies and administrative registries are much lower than those traditionally quoted for OSR with several studies reporting a mortality rate of 20% or less (Table 16).[46,485,557,580,612–629]

Four RCTs comparing OSR with EVAR for rAAA have been published to date[516,544,562,610] (Table 17). All four RCTs documented no statistical difference in perioperative mortality between the two therapeutic options. Individual patient meta-analysis of the three recent RCTs (IMPROVE, AJAX, ECAR) showed, again, no differences in the 30 day and the 90 day mortality rates between EVAR and OSR.[630] Similarly, when summarising the world experience on the topic, there was a conspicuous contradiction between the pooled results of the observational studies, the administrative registries and the RCTs.[611] The observational studies and administrative registries showed that EVAR improved short term survival, whereas the RCTs pooled together (ECAR, IMPROVE, AJAX) demonstrated no such advantage.[630] The disparate results are most probably explained by the differences in study quality and selection bias (in terms of patient confounders, aneurysm anatomy, haemodynamic instability, rejection rates, logistics, operator experience, etc.).[630] Specifically, observational studies and registries are more prone to selection bias. This is because patients must be stable enough for CTA to be considered for EVAR and, therefore, in these studies, there is likely to be a selection bias of more stable patients undergoing EVAR as opposed to OSR. Finally, one should keep in mind that the RCT results, especially in the IMPROVE trial, are given on an intention to treat basis, with some patients receiving a treatment different from the one intended.[544]

Some observational studies have shown little difference in the long term mortality between EVAR and OSR.[621,632–635] In contrast, a large VQI study (2003 – 2018) demonstrated clear survival benefits of EVAR over OSR.[628] Similarly, a recent Swedvasc study on 8 928 rAAA repairs showed that use of EVAR was associated with a significantly reduced long term mortality rate (HR 0.80).[636]

When considering the evidence from RCTs, the one year results from the IMPROVE trial suggested that an endovascular first strategy for rAAA does not offer an early survival benefit, but is associated with faster discharge, better QoL, and is cost effective.[637] When pooled together, the one year results of the three recent RCTs (IMPROVE, AJAX, ECAR) suggest that there is a consistent but non-significant trend for lower mortality after EVAR.[638] The three year results of the IMPROVE trial suggest that, compared with OSR, an endovascular strategy is associated with a survival advantage, a gain in quality adjusted life years, similar levels of re-intervention, and reduced costs, and that this strategy is cost effective. These findings support the increased use of an EVAR for rAAA.[518] This is also

Table 16. **Comparison of peri-operative mortality figures between endovascular and open repair in administrative databases of patients with ruptured abdominal aortic aneurysm (series published from 2010 onwards with > 1 000 patients).**

Author	Publication year	Country	Study period	Patients (EVAR/OSR) − *n*	Death − % EVAR	Death − % OSR
Holt *et al.* (Hospital Episode Statistics)[612]	2010	UK	2003−2008	4 414 (335/4 079)	32	47
Mani *et al.* (Vascunet)[613]	2011	International	2005−2009	7 040 (824/6 216)	20	33
Park *et al.* (Nationwide Inpatient Sample)[46]	2013	USA	2005−2009	16 557 (3 796/12 761)	27	41
Mohan and Hamblin (Nationwide Inpatient Sample)[614]	2014	USA	2001−2010	42 126 (8 140/33 986)	26	39
Edwards *et al.* (Medicare)[615]	2014	USA	2001−2008	10 998 (1 126/9 872) 1 099 propensity score matched patient pairs	34*	48*
Gupta *et al.* (National Surgical Quality Improvement Program)[616]	2014	USA	2005−2010	1 447 (499/948)	24	39
Speicher *et al.* (National Surgical Quality Improvement Program)[617]	2014	USA	2005−2011	1 997 (614/1 383)	26	38
Karthikesalingam *et al.* (Hospital Episode Statistics & Nationwide Inpatient Sample)[580]	2014	England USA	2005−2010 2005−2010	6 897 (569/6 328) 19 174 (4 003/15 171)	32 27	43 46
Ali *et al.* (Vascular Quality Initiative)[631]	2015	USA	2003−2013	1 165 (514/651)	25	33
Karthikesalingam *et al.* (Hospital Episode Statistics & Swedvasc)[43]	2016	England Sweden	2003−2012 2003−2012	12 467 (1 184/11 283) 2 829 (464/2 365)	28 21	41 31
Gunnarsson (Swedvasc)[619]	2016	Sweden	2008−2012	1 304 (343/961)	22	30
Robinson *et al.* (Vascular Quality Initiative)[621]	2016	USA	2003−2013	1 282 (590/692)	23	35
Aziz *et al.* (National Surgical Quality Improvement Program)[618]	2016	USA	2005−2010	2 179 (845/1 334)	17	32
Portelli Tremont *et al.* (Medicare)[620]	2016	USA	2005−2009	8 480 (1 939/6 541)	31	44
Stuntz *et al.* (Nationwide Inpatient Sample)[623]	2017	USA	2002−2014	62 869 (13 426/49 443)	25	33
Budtz-Lilly *et al.* (Vascunet)[44]	2018	International	2010−2013	9 320 (2 155/7 165)	18	32
Gupta *et al.* (Premier Healthcare Database)[624]	2018	USA	2009−2015	3 164 (1 614/1 550)	24	36
Faizer *et al.* (Vascular Quality Initiative)[557]	2019	USA	2003−2017	3 330 (1 736/1 594)	22	34
Azuma (Japanese Society for Vascular Surgery)[625]	2019	Japan	2012	1 698 (345/1 353)	12	19
Behrendt *et al.* (Health insurance claims, DAK-Gesundheit)[626]	2019	Germany	2008−2016	1 477 (517/960)	17	32
Melillo *et al.* (National Surgical Quality Improvement Program)[629]	2020	USA	2008−2016	3 806 (1 843/1 963)	19	29
Salata *et al.* (Administrative data, province of Ontario)[627]	2020	Canada	2003−2016	2 692 (261/2 431)	NA†	NA†
Wang *et al.* (Vascular Quality Initiative)[628]	2020	USA	2003−2018	4 929 (2 749/2 180)	21	34

EVAR = endovascular aneurysm repair; OSR = open surgical repair; NA = not available.
* After propensity score matching. Result not included in summary data.
† EVAR patients were at lower hazard for all cause mortality when compared with OSR (hazard ratio 0.49; 95% confidence interval 0.37−0.65; $p < .01$).

supported by a large Medicare study including > 10 000 patients with rAAA, of whom 1 126 underwent EVAR. After propensity score matching, the peri-operative mortality was significantly lower after EVAR (33.8% after EVAR *vs*. 47.7% after OSR), a difference that persisted for more than four years.[615] Similarly, a time to event meta-analysis of three RCTs and 22 observational studies including 31 383 patients, suggested that EVAR showed a sustained mortality benefit during follow up compared with OSR. The overall all cause mortality was significantly lower after EVAR than after OSR (HR 0.79). However, the post-discharge all cause mortality was not significantly different (HR 1.10). Meta-regression showed the mortality differences in favour of EVAR were more pronounced in more recent studies and recently treated patients.[639,640] Finally, aortic anatomy seems to influence the long term outcome, for both OSR and EVAR. When patients are grouped based on aortic anatomy and whether EVAR is performed inside or outside the IFU, hostile aneurysm anatomy is associated with increased long term mortality and complications after EVAR for rAAA.[640,641] An analysis of the VQI database concluded that outside IFU EVAR for rAAAs yields inferior in hospital survival compared with inside IFU EVAR, but still remains associated with reduced in hospital complications when compared with more complex open or endovascular repair strategies.[642]

The complication rate after rAAA repair varies significantly between series. Indicative rates of post-operative complications after OSR are pulmonary in 42%, cardiac in 18%, acute kidney injury in 17%, colonic ischaemia in 9%,

Table 17. Peri-operative mortality figures in the four randomised controlled trials comparing endovascular and open repair of ruptured abdominal aortic aneurysm.

RCT	Country	Recruitment period	Patients – *n*	30 day mortality rate – %	
				Randomised to EVAR	Randomised to OSR
Nottingham 2006[610]	UK	2002–2004	32	53	53
AJAX 2013[516]	The Netherlands	2004–2011	116	28	29
IMPROVE 2014[544]	UK	2009–2013	613	35	37
ECAR 2015[562]	France	2008–2013	107	18	24
Summary data			868	32.6	34.9

RCT = randomised control trial; EVAR = endovascular aneurysm repair; OSR = open surgical repair; AJAX = Amsterdam Acute Aneurysm Trial; IMPROVE = Immediate Management of Patient with Ruptured Aneurysm: Open *vs.* Endovascular Repair; ECAR = Endovasculaire ou Chirurgie dans les Anévrysmes aorto-iliaques Rompus.

and wound complications in 7%.[643] End organ ischaemia, such as post-operative colonic ischaemia and acute lower limb ischaemia are specifically discussed in section 6.5.

Emergency EVAR also carries the risk of several complications like those encountered after OSR. Whether EVAR is superior to OSR in terms of major morbidity remains to be seen;[644] however, an analysis of the VQI database suggested that EVAR is associated with lower in hospital morbidity than OSR. Specifically, the incidence of cardiac complications (EVAR 29% *vs*. OSR 38%), respiratory complications (28% *vs*. 46%), renal insufficiency (24% *vs*. 38%), lower extremity ischaemia (2.7% *vs*. 8.1%), and bowel ischaemia (3.9% *vs*. 10%) were significantly lower after EVAR than after OSR. Furthermore, median ICU length of stay (EVAR, two days *vs*. OSR, six days) and hospital length of stay (six *vs*. 13 days) were lower after EVAR.[544,631] These observations were confirmed by the IMPROVE trial and a recent meta-analysis of propensity score matched data.[635]

In the most recent publication from the IMPROVE trial, the re-intervention rates were similar after EVAR and OSR for rAAA and most common in the first 90 days.[573] The rate of midterm (between three months and three years) re-interventions after EVAR was high (9.5 per 100 person years) and most commonly performed for endoleak or other endograft related complications that occurred in 17% of patients. Endoleaks causing secondary rupture or requiring re-intervention consisted mainly of Type 1A and 1B endoleaks which, when detected require immediate treatment. T2EL were not the cause of any secondary rupture in the IMPROVE trial but were the commonest reason for re-intervention in the midterm.[573] This suggests that surveillance policies after rAAA repair need to be more strictly enforced and more intensive than those offered after elective repair,[573] which is particularly necessary for patients with rAAA undergoing EVAR outside IFU.

In conclusion, the benefit of EVAR for rAAA has been demonstrated in RCTs and large cohort studies, which is why the recommendation for EVAR as the first option in rAAA remains, whereas it is considered justified to upgrade the LoE to Level A.

Recommendation 80			Changed
For patients with a ruptured abdominal aortic aneurysm and suitable anatomy endovascular repair is recommended as the first line treatment option.			
Class	**Level**	**References**	**ToE**
I	A	IMPROVE Trial Investigators (2017),[518] Gupta *et al.* (2018),[624] Salata *et al.* (2020),[627] Wang *et al.* (2020),[628] D'Oria *et al.* (2023),[636] IMPROVE Trial Investigators (2015),[637] Sweeting *et al.* (2015),[638] Kontopodis *et al.* (2020)[640]	

6.4. Peri-operative complications after ruptured abdominal aortic aneurysm repair

6.4.1. Intra-abdominal hypertension and abdominal compartment syndrome. Intra-abdominal hypertension (IAH) is defined as a sustained or repeated pathological elevation of intra-abdominal pressure (IAP) $>$ 12 mmHg. Abdominal compartment syndrome (ACS) is defined as a sustained IAP $>$ 20 mm Hg (with or without an abdominal perfusion pressure $<$ 60 mmHg) that is associated with new organ dysfunction or failure. Abdominal perfusion pressure is defined as the mean arterial pressure minus the IAP.[645,646]

IAH and ACS may occur after both open and endovascular repair of rAAA. It is estimated that if measured consistently, an IAP $>$ 20 mmHg occurs in about half the patients after open rAAA repair, and 20% will develop ACS.[647] In a meta-analysis of 39 series that were published between 2000 and 2012, the pooled ACS rate after EVAR for rAAA was calculated at 8%, but this figure exceeded 20% with improved awareness and vigilant monitoring.[570] In a more recent meta-analysis of 46 studies, the pooled incidence of ACS after EVAR for rAAA was approximately 9%.[648] This may be explained by the fact that permissive hypotension and massive transfusion protocols have been widely adopted.

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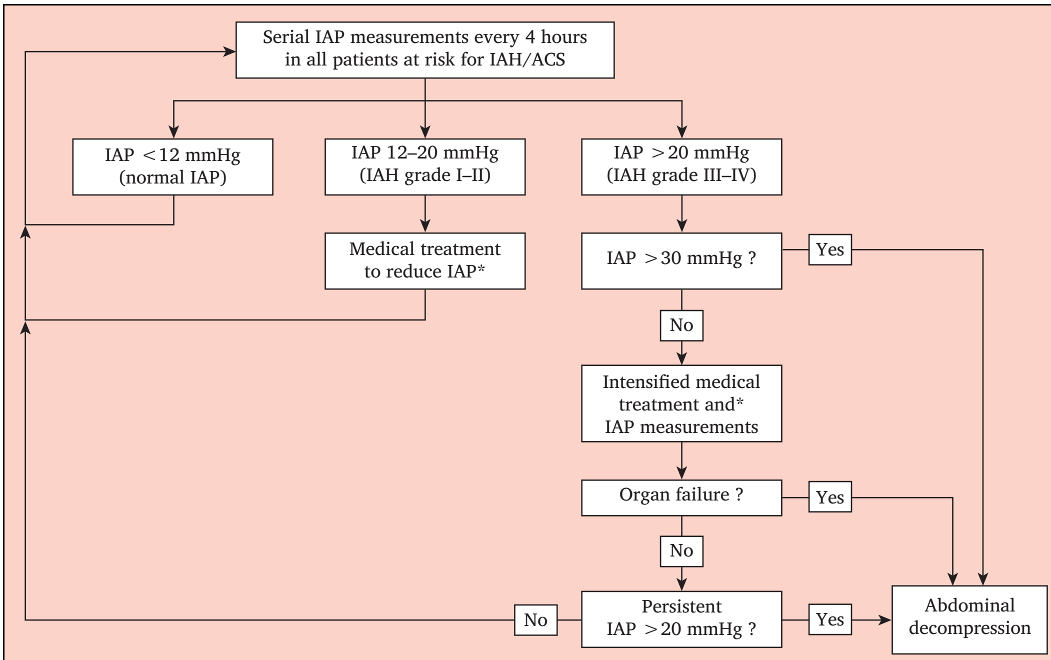

Figure 3. Algorithm for the management of abdominal compartment syndrome after open or endovascular repair of ruptured abdominal aortic aneurysms.[655] IAP = intra-abdominal pressure; IAH = intra-abdominal hypertension; ACS = abdominal compartment syndrome. *See Table 18.

Patients with longer operation time and extensive fluid resuscitation are at higher risk of ACS, whereas a policy of pre-operative permissive hypotension may be protective.[649,650] Risk factors for ACS in patients undergoing EVAR for rAAA, include (1) use of an AOB; (2) severe coagulopathy; (3) massive transfusion requirements; (4) pre-operative loss of consciousness; (5) low pre-operative BP < 70 mm Hg, and (6) the emergency conversion of modular bifurcated stent grafts to AUI devices.[651—653] Nationwide rAAA data from the Swedvasc registry suggest ACS rates of 6.8% after OSR and 6.9% after EVAR between 2008 and 2013 (with an additional 10.7% prophylactically left open after OSR); and 3.7% after OSR and 7.5% after EVAR between 2008 and 2015, which is probably a consequence of the increased use of EVAR in rAAA, which means that more unstable patients are being treated endovascularly.[651,653,654] Therefore, every rAAA patient should be monitored closely to detect ACS early and to initiate appropriate treatment.

A management algorithm for IAH and ACS is summarised in Figure 3.[655] When IAH or ACS is suspected, non-surgical management (Table 18) should be attempted to reduce IAP at first. If conservative measures prove unsuccessful and ACS has developed, decompression midline laparotomy is indicated.[570,645,646,649—651,653—660] The Swedvasc registry provides interesting data about the timing of decompressive laparotomy and the pathophysiological findings. Decompression was performed within 24 hours in 48.7%, after 24 — 48 hours in 26.1%, and after > 48 hours in 25.2%. The three main pathophysiological findings at laparotomy were bowel ischaemia (23.3%), post-operative bleeding (29.3%), and oedema (47.4%). The timing of decompression differed depending on the main underlying pathophysiological finding: post-operative bleeding median 11 hours, oedema 29 hours, and bowel ischaemia 52 hours. Decompression was performed earlier after EVAR compared with OSR (median three hours *vs*. 31 hours).[654]

The development of ACS after open or endovascular treatment of rAAAs is strongly associated with death.[656] In the Swedvasc registry, the 30 day, 90 day, and one year mortality rates after rAAA repair were 42.4%, 58.7%, and 60.7% in patients who developed ACS compared with 23.5%, 27.2%, and 31.8% in patients who did not develop ACS.[651] In the two meta-analyses on ACS post-EVAR for rAAA, data on the outcomes of ACS were available for 76 and 169 patients, of whom 35 (47%) and 94 (55.6%), respectively, died.[570,648]

Prolonged open abdomen treatment is associated with major morbidity, prolonged hospital stay, and need for re-interventions.[657,661,662] Delayed primary fascial closure should therefore be performed as soon as feasible to minimise the risk of large ventral hernias, intestinal fistulas, and graft infection. Different methods exist for temporary abdominal closure of the open abdomen, such as the vacuum pack system with or without mesh bridge, the vacuum assisted wound closure, and the vacuum assisted wound closure with mesh mediated fascial traction.[536,570,647,660—663] According to a systematic review, the vacuum assisted wound closure with mesh mediated traction may achieve a high fascial closure rate without ventral hernia even after long term open abdomen therapy.[662]

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Table 18. Summary of medical treatment options for intra-abdominal hypertension and abdominal compartment syndrome.

Improve abdominal wall compliance	Pain relief (epidural anaesthesia) Avoid morphine Neuromuscular blockade (may reduce intra-abdominal pressure by 50%)
Evacuate intra-luminal and or abdominal content	Nasogastric decompression Paracentesis (seldom feasible)
Correct positive fluid balance	Avoid over resuscitation and crystalloids Whole blood and colloids (20% albumin) Diuretics (furosemide) Renal replacement therapy if indicated
Organ support	Optimise ventilation (positive end expiratory pressure) Vasopressors (abdominal perfusion pressure > 60 mmHg)

Recommendation 81 — Unchanged

After open or endovascular treatment for a ruptured abdominal aortic aneurysm, post-operative monitoring of intra-abdominal pressure is recommended for early diagnosis and management of intra-abdominal hypertension or abdominal compartment syndrome.

Class	Level	References	ToE
I	B	Karkos *et al.* (2014),[570] SÁ *et al.* (2020),[648] Ersryd *et al.* (2016),[651] Ersryd *et al.* (2021)[653] Ersryd *et al.* (2019),[654] Adkar *et al.* (2017),[656] Björck *et al.* (2008),[657] Mayer *et al.* (2014)[658]	

Recommendation 82 — Unchanged

Patients with abdominal compartment syndrome after open or endovascular treatment of a ruptured abdominal aortic aneurysm should be treated with decompressive laparotomy.

Class	Level	References	ToE
I	B	Mayer *et al.* (2009),[647] Steenberge *et al.* (2021),[649] Ersryd *et al.* (2021),[653] Ersryd *et al.* (2019),[654] Adkar *et al.* (2017),[656] Seternes *et al.* (2017),[660] De Waele *et al.* (2016)[664]	

Recommendation 83 — Changed

In the management of open abdomen following decompression for abdominal compartment syndrome after open or endovascular treatment of ruptured abdominal aortic aneurysm, a vacuum assisted closure system with mesh mediated traction and early abdominal closure should be considered.

Class	Level	References	ToE
IIa	C	Mayer *et al.* (2009),[647] Seternes *et al.* (2017),[660] Acosta *et al.* (2016),[662] Petersson *et al.* (2007)[663]	

6.4.2. Colonic ischaemia. Post-operative colonic ischaemia is a serious complication of both open and endovascular repair of rAAAs. In a meta-analysis, including 52 670 patients from 101 studies, the pooled prevalence of clinically relevant bowel ischaemia after rAAA repair was 10%, and approximately 4% of patients die of its consequences. The risk of bowel ischaemia was higher after OSR than after EVAR (RR 1.8).[665] The reported rate of colonic ischaemia is higher in studies performing routine sigmoidoscopy in all patients after rAAA repair, ranging between 14% and 32%, but it also includes cases of mild or moderate ischaemia that are often treated conservatively.[666–668] Sigmoidoscopy is accurate for ruling out colonic ischaemia after rAAA repair, but is less specific in diagnosing the presence of clinically relevant transmural colonic ischaemia.[665] Therefore, routine sigmoidoscopy is not recommended after rAAA repair. Instead, a selective approach, with sigmoidoscopy in patients with a clinical suspicion or at high risk, is advocated.[665–672] Post-operatively, all patients with rAAA should be closely monitored for signs of colonic ischaemia. When the diagnosis is suspected, frequent clinical assessments, monitoring of IAP (which has been found to have a strong correlation with colonic ischaemia), liberal use of sigmoidoscopy, and early exploratory laparotomy are recommended to confirm the diagnosis and to improve the overall management (Fig. 4).[665,669,672,673]

Recommendation 84 — New

For patients undergoing open or endovascular treatment for ruptured abdominal aortic aneurysm in whom colonic ischaemia is suspected, flexible sigmoidoscopy should be considered, to confirm the diagnosis.

Class	Level	References	ToE
IIa	B	Champagne *et al.* (2007),[666] Tøttrup *et al.* (2013),[667] Megalopoulos *et al.* (2007),[668] Jalalzadeh *et al.* (2019),[669] Urbonavicius *et al.* (2020),[671] Djavani *et al.* (2009)[673]	

6.4.3. Acute lower limb ischaemia. Acute lower limb ischaemia following OSR or EVAR for rAAA represents a serious condition that may lead to amputation and death if not treated promptly. The incidence of this complication in the American College of Surgeons NSQIP database was 4.8% with no significant differences between EVAR and OSR. This percentage is significantly higher than the 1.6% rate

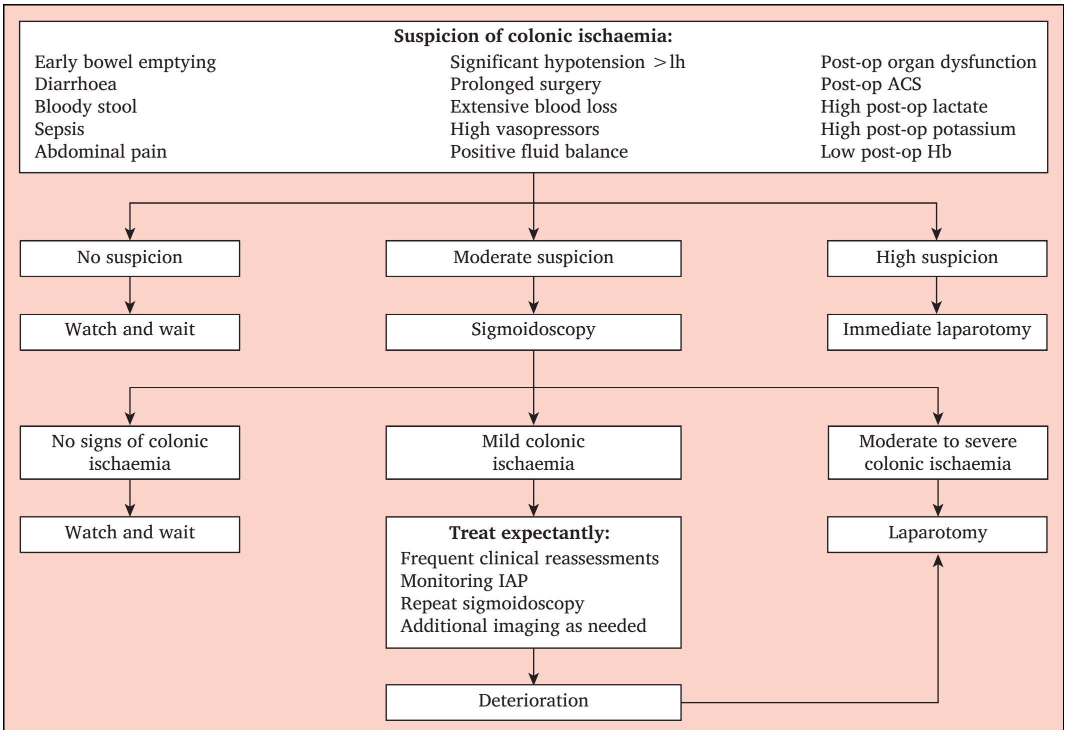

Figure 4. Flowchart of the diagnostic process of colonic ischaemia in patients surviving ruptured abdominal aortic aneurysm repair. Modified from Jalalzadeh *et al.*[669] rAAA = ruptured abdominal aortic aneurysm; OSR = open surgical repair; EVAR = endovascular aneurysm repair; Hb = haemoglobin; post-op = post-operative; ACS = abdominal compartment syndrome; IAP = intra-abdominal pressure.

documented for elective or symptomatic AAAs ($p <$ .001).[674] Haemodynamic instability, prolonged aortic cross clamp time and operation time, lack of heparin administration, and thromboembolic events may all play a role in its development. Such patients have significantly worse outcomes in terms of 30 day mortality (20.5 *vs.* 4.6%, $p <$ .001). If lower limb ischaemia is suspected on table, immediate revascularisation may be necessary depending on the aetiology.[573,601,633]

6.5. Symptomatic non-ruptured abdominal aortic aneurysm

Symptomatic non-ruptured aneurysm has a variable definition, varying from tenderness on palpation to evidence of peripheral emboli with no other obvious source, or unexplained back or abdominal pain. Such instances of aneurysms < 55 mm diameter require urgent investigations to substantiate the symptomatic diagnosis.

For symptomatic non-ruptured AAAs, the optimal timing of treatment is debated. These aneurysms are thought to have a higher rupture risk than asymptomatic aneurysms, while emergency repair under less favourable circumstances is associated with a higher risk of peri-operative complications.[675–680] Delay in operative repair might improve outcome by allowing a more complete risk assessment, patient optimisation, and avoidance of out of hours operations by less experienced surgical and anaesthetic teams.[677,681] Therefore, the management of these cases should involve a brief period of rapid assessment and optimisation followed by urgent repair under optimum conditions.[678,680,682] Careful monitoring with strict BP and pain management awaiting repair is important.

Recommendation 85		Changed
Patients with a symptomatic non-ruptured abdominal aortic aneurysm may be considered for a brief period of rapid assessment and optimisation followed by urgent repair under optimal conditions (ideally during working hours).		
Class	**Level**	**References** **ToE**
IIb	**B**	Haug *et al.* (2004),[675] Tambyraja *et al.* (2004),[676] Ten Bosch *et al.* (2016),[677] De Martino *et al.* (2010),[678] Abdulrasak *et al.* (2020),[679] Soden *et al.* (2016),[680] Cambria *et al.* (1994),[681] O'Donnell *et al.* (2019)[682]

7. LONG TERM OUTCOME AND FOLLOW UP AFTER ABDOMINAL AORTIC ANEURYSM REPAIR

This chapter focuses on long term outcomes and management after infrarenal AAA repair by both OSR and EVAR.

This includes secondary prevention, complications occurring after the peri-operative period, and implications for follow up. For juxtarenal AAA, see Chapter 8.

Patients who have undergone AAA repair are at increased risk of death compared with the general population. In a meta-analysis of survivors after elective AAA repair, including 107 814 patients in 36 studies, the five year survival rate was 69%, which is lower than individuals without AAA but higher than observed for other vascular diseases such as PAOD.[683]

The long term survival after AAA repair is affected by age, sex, comorbidities, and regional differences.[42,683,684] End stage renal disease and COPD requiring supplementary oxygen are particularly relevant predictors of late death in patients with AAA, increasing risk over three fold.[684] Baseline AAA diameter is also a consistent predictor of survival.[683,685–687] Large AAAs are associated not only with a higher mortality rate, but also more secondary interventions, post-repair ruptures and loss of follow up.[688] Female sex has been suggested to negatively affect survival after AAA repair, but evidence is conflicting.[689,690] In octogenarians, longevity after AAA repair is not significantly different from that of an age matched population without AAA.[691]

Unlike peri-operative mortality, which has gradually decreased over time, late death after AAA repair remains high with no major improvements over the last two decades.[690] The most common causes are cardiovascular (particularly IHD), lung cancer and pulmonary disease.[39,690,692]

In a case–control analysis of 19 505 patients with AAA operated on in the UK, the five year freedom from adverse cardiovascular events was 86% among patients with AAA and 93% for controls.[693] The annual risk of MI, stroke, and death was increased approximately twofold compared with a matched population in a Danish cohort of patients with AAA.[694] After EVAR, patients with a wide proximal neck diameter ($\geq$ 30 mm) were found to be at higher risk of death from a cardiovascular cause (HR 2.16), whereas $>$ 25% circumferential neck thrombus was protective (HR 0.32).[686] Cancer related death is more common among survivors after AAA repair, which is probably due to common risk factors for atherosclerosis and several types of cancer, such as smoking.[692,695]

Notably, survival after the first 90 days does not differ significantly between ruptured and intact AAA repair.[486,696] Although the risk of late aneurysm related death is difficult to assess due to the uncertainty in cause of death registration and lack of adequate long term cohorts, it has been reported to be less than 3%.[692,697]

7.1. Medical management after abdominal aortic aneurysm repair

Most patients requiring AAA repair suffer from advanced atherosclerotic disease and other smoke related comorbidities.[698,699] Despite the increased risk, no RCTs have been performed to assess whether medical management modifies the prognosis of these patients[190] but there is consensus that secondary prevention directed at risk factor management and medication for any underlying cardiovascular disease should be continued.

Best medical treatment includes antiplatelet therapy, statins and antihypertensive medication, although evidence on individual drugs may be conflicting.[700–703] In a recent meta-analysis including 69 790 patients from 11 cohort studies, statin use was associated with a 35% relative risk reduction in mortality rate for patients after AAA repair.[338] A subsequent meta-analysis on the same subject, including 134 290 patients, confirmed these findings reporting a lower short (OR 0.51) and long term (OR 0.67) mortality rate for statin users.[207] Guidelines directed at the medical management of each individual risk factor and atherosclerotic medication should be consulted for detailed recommendations.[336,704] In the management of patients following AAA repair, re-assessment of bleeding risk, dose adjustment and compliance with best medical treatment should be ensured at regular intervals.

Recommendation 86		Changed
Patients operated on for an abdominal aortic aneurysm should receive post-operative cardiovascular risk management including statin therapy, antiplatelet medication, and blood pressure control.		
Class	**Level**	**References** ToE
I	B	Xiong *et al* (2022),[207] Risum *et al.* (2021),[338] Khashram *et al.* (2017)[702] Zhang *et al.* (2015),[703] Lindstrom *et al.* (2021)[705]

7.2. Late complications after abdominal aortic aneurysm repair

Late complications may occur after both OSR and EVAR. While some complications are unique to one of the techniques (e.g., incisional hernias after OSR or endoleak after EVAR), others may occur irrespective of the technique used (e.g., graft infection or graft occlusion). A summary of frequent late complications after OSR is presented in Table 19, and after EVAR in Table 20. Patients treated by EVAR are more likely to experience aortic complications and secondary interventions than those treated by OSR.[479,481,483]

7.2.1. Graft occlusion. Graft occlusion is a relatively frequent complication after OSR and EVAR, accounting for roughly one third of all secondary interventions. After OSR with a bifurcated prosthesis, limb occlusion occurs in 1 – 5%[707,708] and after EVAR in 5.6%.[721] Graft occlusion presents as acute limb ischaemia in 32 – 44% of cases, as chronic limb ischaemia in 50 – 60% and some are asymptomatic and detected incidentally on imaging (7%).[721,722] Roughly half of all stent graft occlusions present after 30 days.[721]

The strongest risk factor for EVAR limb occlusion is extension to the EIA.[723–727] Other risk factors for limb

Table 19. Long term complications after open surgical repair of abdominal aortic aneurysm, and their incidence within five and 10−15 years.[403,404,465,468,706–710]

Complication	Estimated incidence within five years	Estimated incidence within 10−15 years
Para-anastomotic aneurysm	1−2%	4% at 10 years − 12% at 15 years
Graft occlusion	1%	5% at 15 years
Incisional hernia	5−12%	5−21%
Graft infection	0.5−5%	−
Secondary aorto-enteric fistula	< 1%	−

occlusion include iliac artery angulation, tortuosity, calcification or stenosis, stent graft oversizing ≥ 15%, small AAA or narrow aortic bifurcation, and stent graft material.[421,724,728,729] Some evidence points towards an increased risk of occlusion when low profile stent grafts are used.[419,421,730] The heterogeneity and retrospective nature of data precludes a specific recommendation on this subject, apart from the previously given general recommendation for enhanced monitoring and long term follow up of new generation devices.

Stent graft obstruction due to kinking or stenosis may be detected prior to occlusion, due to new or worsening symptoms, or on routine follow up imaging, often requiring intervention. In a recent meta-analysis, open surgery (usually thrombectomy or extra-anatomical bypass) was more frequently used (61%), followed by endovascular repair with or without thrombolysis (17%) with hybrid procedures performed in 8%. Conservative management was preferred in 13%. The mortality rate was 3.6% and amputation rate 3.1%. Recurrence rates remain high, at 8.0%.[721] There is no evidence in the literature regarding superiority of one treatment option over the other, and the treatment strategy should be patient tailored.[721,722]

Thrombus deposits inside stent grafts has been investigated as a potential source of occlusions or thromboembolic events. These deposits may be caused by systemic and local haemodynamic factors and stent graft characteristics. Sharp cross sectional decreases in graft size (taper), as observed in aorto-uni-iliac devices or devices with large bodies and small diameter limbs, seem especially prone to induce mural thrombus[731] (Fig. 5). A meta-analysis comprising five observational studies including 808 patients (mean follow up 10 − 68 months) reported mural thrombosis in 21%, the majority developing within the first year after implantation, but no evidence suggesting an increased risk of occlusion or thromboembolism in affected patients. Furthermore, no correlation between antithrombotic regimen and development (or prevention) of mural thrombus was found. As such, no specific therapy is

Table 20. Long term complications after endovascular repair of abdominal aortic aneurysm.[465,468,469,711–720]

Complication	Meaning	Estimated incidence within five years	Rupture risk*
Type 1 endoleak	Sealing zone failure	5%	High
Type 1a	From proximal seal		
Type 1b	From distal seal		
Type 1c	From iliac occluder†		
Type 2 endoleak	Retrograde flow from aortic side branches	20−40% of which 10% persistent at two years	Low if no AAA sac expansion Intermediate if AAA sac expansion
Type 2a	One vessel visible		
Type 2b	More than one vessel visible		
Type 3 endoleak	Midgraft failure	1−3%	High
Type 3a	Separation or poor apposition of modular components		
Type 3b	Graft disruption		
Type 4 endoleak	Graft porosity		Low
Undetermined endoleak	Visible endoleak with no clear origin		Intermediate
Post-EVAR growth without endoleak		1%	Intermediate
Graft infection		0.5−1%	High
Post-EVAR rupture		1−6%	−
Graft obstruction	Partial or total obstruction of blood flow, including kinking	0.5−1%	Low
Migration	Proximal (descending) or distal (ascending) migration	0−9%	High if associated with Type 1 endoleak

* Rupture risk based on rough estimates indirectly derived from literature and expert panel opinion (low: < 1% year, intermediate 1 − 5%/year, high > 5%/year).
† In treatment with aorto-uni-iliac devices.

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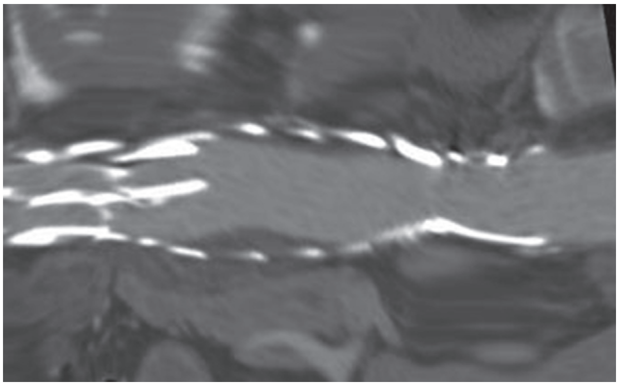

Figure 5. Example of benign mural thrombus formation inside the main body of a stent graft, due to a barrel shape configuration (computed tomography angiography with centre lumen line reconstruction).

indicated.[732] However, a recent small observational study suggested that escalation of antithrombotic therapy could stop progression or resolve thrombus.[733] It is important to note that studies investigating thrombus deposits inside stent grafts did not specifically investigate partial thrombosis of endograft limbs, which may occur because of kinking or obstruction. Although most evidence suggests that asymptomatic mural thrombosis with no significant haemodynamic effect may be managed with vigilance only, there is uncertainty regarding which patients may benefit from treatment by secondary intervention or escalation of antithrombotic medication. An individualised therapeutic strategy is therefore recommended for patients with thrombus that results in symptoms, shows significant evolution over time, or results in haemodynamically significant stenosis.

Recommendation 87 **Changed**

Patients operated on for an abdominal aortic aneurysm with new onset or worsening of lower limb ischaemia are recommended immediate evaluation of graft related problems, such as limb kinking or occlusion.

Class	Level	References	ToE
I	B	Hammond *et al.* (2018),[721] Coelho *et al.* (2019)[722]	

Recommendation 88 **New**

For patients treated by endovascular abdominal aortic aneurysm repair who present with asymptomatic non-obstructive mural thrombus formation limited to the main body of stent graft, intervention or escalation of antithrombotic therapy is not indicated.

Class	Level	References	ToE
III	C	Perini *et al.* (2018),[732] Bianchini *et al.* (2020)[734]	

Recommendation 89 **New**

Patients treated by endovascular abdominal aortic aneurysm repair who present with symptomatic, evolving, or haemodynamically significant thrombus formation inside the stent graft may be considered for individualised intervention or escalation of antithrombotic therapy.

Class	Level	References	ToE
IIb	C	Perini *et al.* (2018),[732] Russell *et al.* (2022)[733]	

7.2.2. Aortic and stent graft infection and graft enteric fistula. Prosthetic graft infection is a serious complication with a poor prognosis. It occurs between 0.3% and 6% after OSR[735] and 0.2 — 1% after EVAR.[736—738] The reported frequency of secondary graft enteric fistula (GEF) is 0.3 — 4.3%, with a two to four fold risk after OSR compared with EVAR.[707,739—743]

Risk factors for AGI include prosthetic material in the groin, emergency operations, intestinal injury, peri-operative infections, bacteraemia, need for extra-anatomical bypass in aorto-uni-iliac stent grafts, previous coil embolisation of the hypogastric artery, diabetes and immunosuppression.[463,737,744,745] Because of the high morbidity and mortality of AGI and GEF (20 — 75% combined morbidity and mortality in various series),[741,746,747] prevention is key, and early diagnosis and aggressive treatment are essential.[341]

Overall, management of AGI is highly complex, and patients should preferably be managed in high volume centres for multidisciplinary evaluation and treatment, as recommended in the ESVS Clinical Practice Guidelines on the Management of Vascular Graft and Endograft Infections Guidelines (Class I, Level C)[341] According to this document, diagnosis of AGI should follow the Management of Aortic Graft Infection Collaboration (MAGIC) criteria (Class I, Level C), and every effort should be made to obtain microbiological proof of the causative agent (Class I, Level C). CTA is the preferred diagnostic modality (Class I, Level B), adding 18-fluoro-deoxyglucose positron emission tomography (^{18}F-FDG/PET-CT) and or white blood cell scintigraphy (WBCS) if necessary to improve diagnostic accuracy (Class I, Level B). A patient tailored approach is recommended, based on the patient's condition, anatomy, and state of infection, including presence of GEF, determining the therapeutic strategy. Prophylaxis of graft infection should be considered for dental procedures involving gingival or peri-apical manipulation or perforation involving the mucosa, as well as in other high infection risk procedures like abscess drainage (Class IIa, Level C). For more details and guidance on diagnosis and AGI workup, as well as antibiotic prophylaxis for surgical or dental procedures in patients with an aortic prosthesis, please refer to the ESVS Clinical Practice Guidelines on the Management of Vascular Graft and Endograft Infections.[341] A section on management is added to this document due to the relevant new evidence that became available after its publication. Numerous bacteria and fungi may cause AGI, but Gram positive bacteria and enterococci are the most common. More virulent pathogens such as *Staphylococcus aureus* or *Pseudomonas aeruginosa* are associated with a worse prognosis and higher risk of re-infection, while pathogens that typically colonise the skin such as *Staphylococcus epidermidis* or *corynebacteria* are less virulent.[748,749] Polymicrobial growth and Candida involvement is especially common in patients with GEF; 37% and 31% respectively.[749,750]

When infection is present, complete graft removal and infected tissue debridement should be considered.[737,744,751] The preferred treatment of AGI is an *in situ* reconstruction with extensive debridement of infected tissues, using infection resistant materials such as autologous deep vein, cryopreserved allografts, or xenopericardial grafts.[740,745,751–760] Prosthetic graft replacement is associated with higher risk of re-infection than autogenous reconstructions, while prosthetic grafts impregnated with silver and or antibiotics fared better than standard prosthetic grafts. Biological reconstructions are not, however, free from re-infection.[740,751,755,756,761,762]

Aortic ligation with extra-anatomical reconstruction is a reasonable alternative, especially when the patient's risk profile is high, or the local tissue infection is extensive. A recent large international multicentre study, including 182 patients with AGI with GEF, found no survival benefit of *in situ vs*. extra-anatomic reconstruction, while the latter were less likely to experience aorta related haemorrhage within 30 days post-operatively (3% aortic stump dehiscence *vs*. 11% anastomotic rupture).[743] This was confirmed in a nationwide study from Sweden, including 126 patients where 50% had enteric involvement, showing similar early survival between extra-anatomical and *in situ* reconstruction (81.7% *vs.* 76.4% respectively), five year survival (48.2% *vs*. 49.9%) and recurrent infection (20.3% *vs*. 17.0%). The rate of aortic stump blowout after extra-anatomic reconstruction and anastomosis dehiscence after *in situ* reconstruction during follow up was the same, 9.8%.[749] However, in a cohort of 241 patients with AGI without enteric involvement, extra-anatomic reconstruction was associated with nearly a two and one half fold higher re-infection and mortality rate compared with *in situ* reconstruction. Furthermore, omental and or muscle flap coverage of the repair appear to be protective.[743]

Aortic GEF frequently requires emergency treatment.[763] Synchronous and staged procedures using *in situ* or extra-anatomical strategies and autologous, homologous, or prosthetic material have been used for vascular repair.[736,742,743,764–767] Enteric repair can be performed with duodenorrhaphy, with or without omental interposition and with or without enterostomy, or duodenal resection or reconstruction. A literature review including 331 aortic GEF cases suggests that the use of omental interposition and *in situ* vascular reconstruction may be advantageous, and that duodenal diversion is preferable to basic closure of the fistula.[747] A review and pooled data analysis of 823 GAF cases suggests that a staged endovascular (bridge) to open surgery, for bleeding control, is associated with better early survival.[763] Intestinal complications are a major risk factor increasing the risk of death by at least three fold. Assessment and surgical management of the enteric defect by a specialist in intestinal surgery and a liberal use of second look are suggested.[742,747,763,766]

Long term systemic antibiotic treatment is recommended in all patients treated for AGI, with a minimum treatment duration of six weeks.[764] The exact duration of antibiotic treatment, which may be lifelong, needs to be managed individually, and should be done in close collaboration with infectious disease specialists. In a multicentre study based on the Vascular Low Frequency Disease Consortium (VLFDC), including 182 patients with an aortic GEF, duration of antibiotic use (HR 0.92) and rifampicin use at the time of discharge (HR 0.20) independently decreased mortality. Re-infection developed in only 7% of those receiving lifelong culture directed antibiotics.[743] This was confirmed in a Swedish nationwide study, including 126 patients with AGI, where prolonged antimicrobial therapy (more than three months) was significantly associated with a reduced long term mortality rate (HR 0.3).[749] Testing for fungal agents and adjuvant anti-fungal treatment, preferably with echinocandins, should be considered in all patients with aortic GEF.

In patients, unsuitable for radical surgical therapy, a semi-conservative approach with partial graft removal or a conservative palliative medical management strategy may be considered.[737,740,744,768] In a recent Swedish nationwide study, including 169 patients with a surgically treated AGI, 43 had been treated with partial graft or stent graft removal. There was a trend towards worse unadjusted overall survival of the semi-conservative group compared with the radically treated group, particularly in the presence of a GEF. This was largely explained by higher age and the presence of more comorbidities, in the semi-conservative group. When adjusting for these confounders, there was no significant difference in long term survival between a semi-conservative and a radical surgical approach.[769] However, partial resection of infected grafts leads to significantly higher rates of re-infection, up to 39 – 45%, especially in patients with abdominal infection not isolated to a single graft limb, with Candida infection or with GEF.[769,770] Hence, partial resection of infected aortic grafts may be an alternative in comorbid patients with an isolated (localised) infection not comprising Candida or without a GEF. Nevertheless, the observed high recurrence rate warrants the need for close surveillance and prolonged or lifelong antimicrobial therapy in patients treated for AGI with partial graft removal. Leaving the bare metal top stent *in situ* can simplify explantation of an infected EVAR device. However, there is no evidence as to whether this is advisable, and it should be decided on a case by case basis.

Conservative management of AGI with antimicrobial therapy, alone or in combination with percutaneous drainage, sac irrigation or omentoplasty, with stent graft preservation should be considered as a last resource for high surgical risk patients, given the generally poor results, especially if GEF is present.[744,771,772] A recent retrospective single centre study from Sweden, however, reported encouraging outcomes of patients treated conservatively with AGI without fistula deemed unfit for surgical treatment, where the microbiological aetiology was identified, allowing for targeted antibiotic therapy. The Kaplan—Meier estimated survival was 98% at 30 days, 88% at one year, and 79% at three years, with 48% of the patients being able to discontinue antibiotic treatment after a median of 16 months.[773]

There is no specific evidence on how to follow up patients after management of infected aortic grafts. The ESVS Clinical Practice Guidelines on the Management of Vascular Graft and Endograft Infections recommend lifelong follow up after *in situ* reconstruction with cryopreserved allografts for abdominal aortic vascular graft or endograft infection, to detect allograft degeneration (Class I, level C).[341] A recent publication on outcomes after infected stent graft explantation described a follow up protocol as clinical examination with blood tests at one, three, and six months, and annually thereafter, PET-CT at six months (repeated if considered necessary) and CTA scans annually. However, other expert groups have reported different strategies or made no reference to surveillance protocols, and most recommend individualised strategies. Due to the paucity of evidence and heterogeneity of protocols, no general recommendation can be made.

Recommendation 90 — Changed

Patients with aortic graft or stent graft infection should be considered for radical treatment with complete graft or stent graft explantation as first line treatment.

Class	Level	References	ToE
IIa	B	Argyriou *et al.* (2017),[737] Li *et al.* (2018),[744] Khalid *et al.* (2023)[751] Janko *et al.* (2021)[770]	

Recommendation 91 — New

For patients undergoing complete explantation of an infected aortic graft or stent graft, *in situ* reconstruction using biological graft material should be considered the preferred repair modality.

Class	Level	References	ToE
IIa	C	Smeds *et al.* (2016),[740] Lyons *et al.* (2013),[745] Janko *et al.* (2022),[752] Colacchio *et al.* (2023),[753] Wang *et al.* (2022),[754] Langenskiold *et al.* (2021),[755] Weiss *et al.* (2021),[756] Xodo *et al.* (2022),[757] Alonso *et al.* (2021),[758] Almási-Sperling *et al.* (2020),[759] Heinola *et al.* (2016),[774] Schaefers *et al.* (2019)[775]	

Recommendation 92 — New

For patients undergoing complete explantation of an infected aortic graft or stent graft, extra-anatomic reconstruction may be considered an alternative repair modality in frail patients, in cases with extensive infections, or with graft enteric fistula.

Class	Level	References	ToE
IIb	C	Janko *et al.* (2021),[743] Gavali *et al.* (2021)[749]	

Recommendation 93 — Unchanged

For selected high risk patients with aortic graft or stent graft infection, conservative and or palliative options should be considered.

Class	Level	References	ToE
IIa	C	Argyriou *et al.* (2017),[737] Smeds *et al.* (2016),[740] Li *et al.* (2018)[744] Caradu *et al.* (2022)[776]	

Recommendation 94 — New

For selected high risk patients with an isolated (localised) aortic graft or stent graft infection not involving *Candida* and without enteric involvement, partial graft removal, rather than radical explantation, may be considered.

Class	Level	References	ToE
IIb	C	Janko *et al.* (2021),[743] Simmons *et al.* (2017)[768] Gavali *et al.* (2023)[769]	

Recommendation 95 — New

For patients with aorta or graft enteric fistula, adjuvant antifungal therapy should be considered, until fungal infection has been properly investigated.

Class	Level	References	ToE
IIa	C	Gavali *et al.* (2021),[749] Puges *et al.* (2021),[750] Janko *et al.* (2021)[770]	

Recommendation 96 — New

For patients treated for aortic graft or stent graft infection deemed at high risk of re-infection or when complete graft removal is not achieved, long term culture specific antibiotic therapy should be considered.

Class	Level	References	ToE
IIa	C	Gavali *et al.* (2021),[749] Charlton-Ouw *et al.* (2014),[764] Janko *et al.* (2021)[770]	

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Recommendation 97		Unchanged
For patients with an aortic prosthesis presenting with gastrointestinal bleeding, prompt assessment to identify a possible graft enteric fistula is recommended.		
Class	Level	References
I	C	Consensus

Recommendation 98		Unchanged
For patients with graft enteric fistula and bleeding, staged endovascular stent grafting as a bridge to open surgery may be considered.		
Class	Level	References ToE
IIb	C	Mauriac *et al.* (2021)[742] Janko *et al.* (2021),[743] Kakkos *et al.* (2016)[763]

Recommendation 99		New
For patients undergoing open repair of graft enteric fistula, assessment and management of the enteric defect by a gastrointestinal surgeon should be considered.		
Class	Level	References ToE
IIa	C	Mauriac *et al.* (2021),[742] Janko *et al.* (2021),[743] Rodrigues *et al.* (2014),[747] Kakkos *et al.* (2016),[763] Chopra *et al.* (2017)[766]

7.2.3. Sexual dysfunction.

Patients with AAA have a high baseline prevalence of sexual dysfunction. Up to 75% of patients report problems such as erectile dysfunction and retrograde ejaculation, often because of advanced age and comorbidities.[777]

In a prospective single centre study from Germany, 27% of the patients reported erectile dysfunction before OSR increasing to 53% one year after surgery. The corresponding frequencies after EVAR were 43% and 59% respectively.[778] In a systematic review, incidence of *de novo* erectile dysfunction ranged from 20% to 83% after OSR and 11% to 14% after EVAR. Despite these apparent differences, comparative studies had inconsistent findings.[779] While it can be expected that the rate of retrograde ejaculation is higher after OSR, the paucity of data exploring this subject does not allow clear conclusions.[779–781] After EVAR the reported incidence of new sexual dysfunction ranges up to 17% in patients with intra-operative unilateral IIA occlusion and up to 24% in bilateral occlusion.[779,782,783]

Long term prospective data analysing operative strategies, risk factors, and therapeutic options are currently not available. It is, however, important to inform patients about this complication and be aware of the pre-operative prevalence of sexual dysfunction in all male patients undergoing OSR and EVAR. Given the complex physiological and psychological nature of sexual dysfunction, affected patients should be evaluated by specialists in this field.

Recommendation 100		New
For patients treated for abdominal aortic aneurysm who are distressed by post-operative new onset sexual dysfunction, referral to specialised teams should be considered.		
Class	Level	References ToE
IIa	C	Regnier *et al.* (2018)[779]

7.2.4. Para-anastomotic aneurysm formation.

Para-anastomotic aneurysm formation may occur after OSR, either as a true aneurysm developing adjacent to the anastomosis or a false aneurysm caused by disruption of the anastomosis. Graft infection may be the underlying cause of secondary aneurysm formation, particularly within the first years after repair, and needs to be excluded.[737] The ESVS Clinical Practice Guidelines on the Management of Vascular Graft and Endograft Infections recommend that the MAGIC criteria are used for excluding associated graft infection. The use of ^{18}F-FGD-PET combined with CTA is also recommended as an additional imaging modality to improve diagnostic accuracy.[341] Historical series report an incidence up to 10% after 10 years in both aortic and femoral anastomoses. A contemporary study suggests lower incidences at five and 10 years for aortic para-anastomotic aneurysms (2.2% and 3.6%, respectively).[709]

Indications for therapy depend on aetiology (see section 7.2.2), para-anastomotic aneurysm size and clinical symptoms. There are no data to support size thresholds for repair of para-anastomotic aneurysms. While true aortic or iliac aneurysms proximal or distal to the anastomosis can be treated at a diameter threshold equivalent to that for elective therapy, a lower threshold diameter may be justified for false or saccular aneurysms. Both endovascular and open repair may be used to treat aortic and iliac para-anastomotic aneurysms. Depending on the extent of disease and landing zones, stent grafts with or without fenestrations or branches have been used with good outcomes and should be considered preferentially.[784,785] Open surgery is mostly used in femoral para-anastomotic aneurysms.[707,786–788]

Recommendation 101		New
For patients with para-anastomotic aneurysm formation after previous abdominal aortic aneurysm repair, infection as the underlying cause should be considered.		
Class	Level	References
IIa	C	Consensus

Recommendation 102		New
For patients with non-infectious para-anastomotic aneurysm formation after previous abdominal aortic aneurysm repair, endovascular repair should be considered preferentially.		
Class	Level	References ToE
IIa	C	Gallitto *et al.* (2020),[784] Spanos *et al.*[785]

7.2.5. Incisional hernia. Incisional hernia is a common and frequently under reported complication of OSR. A recent meta-analysis reported an average annual rate of hernia development varying between 10% for midline incisions to 3% for retroperitoneal incisions.[384] While prophylactic mesh reinforcement of midline incisions has been shown to reduce the risk of hernia development[408,409] (see section 5.3.1.4), there are no specific data on patients with AAA for management once the complication has developed. General guidelines for management of incisional hernias are advised.

7.2.6. Endoleaks. An endoleak signifies the presence of flow in the aneurysm sac outside the stent graft after EVAR.[789] It is identified in up to one third of cases,[717] although the prevalence depends on multiple factors including the type and frequency of imaging performed during follow up.

Endoleaks are classified into primary (present at the time of repair) or secondary (occurring after prior negative post-operative imaging), as well as on the cause of perigraft flow (Table 20).

Type 1 or 3 endoleaks are the most concerning since they expose the vessel wall to arterial blood pressure and pulsatile flow. The associated risk of secondary rupture is therefore high. T2ELs are more benign but may also be cumbersome if associated with continued AAA growth.[719] Type 4 endoleaks, related to graft porosity, have virtually disappeared in modern stent graft designs. Management of endoleaks is naturally conditioned by mechanism of development, ranging from basic vigilance to endovascular interventions or open conversion.

7.2.6.1. Type 1 endoleak. Persistent direct flow in the aneurysm sac due to inadequate proximal (Type 1a) or distal (Type 1b) seal of the stent graft is associated with a high risk of aneurysm rupture. Direct flow may also occur because of lack of seal in an iliac occluder (Type 1c) following AUI repair with femorofemoral crossover graft. In a meta-analysis including 190 ruptures after EVAR, Type I endoleak was reported in over 60% of cases[790] and other studies reported even higher proportions, up to 80%.[791,792] Attention should also be given to the evolution of the sealing zones over time, both proximally and distally. Progressive dilatation may exceed the nominal stent graft diameter and compromise seal.[793,794] Migration of the proximal main body is now less frequent due to the general use of grafts with active fixation (hooks or barbs)[696,795] but retrograde migration of iliac limbs may occur, predisposing to Type 1b endoleak.[712,796] Aneurysms with large flow lumen may be at especially high risk due to graft displacement over time.[797] When sealing zones are compromised, even without visible endoleaks, pre-emptive treatment may be considered.

Different endovascular options are available to resolve Type 1 endoleaks or improve sealing zones, depending on the mechanisms of failure. These include proximal or distal extensions, which most frequently require fenestrated or branched devices to preserve visceral branch vessels or internal iliac arteries. If has migration occurred and there is sufficient sealing zone for an extension cuff, this can be performed with low complexity. Most frequently, however, sufficient sealing can only be achieved by incorporating the renovisceral or internal iliac arteries. In elective settings, these procedures can be performed with very low risk.[798–807] The use of parallel stents (chimneys) has also been used successfully, and may be particularly useful in emergency settings.[808] Occasionally, apposition of the stent graft fabric with endovascular staples against the aortic wall is possible, provided the stent graft is adequately sized, has not migrated, and there is an appropriate sealing zone.[801,809,810] The literature however suggests a high risk of recurrence for Type 1 endoleaks treated with endostaples.[811] The use of embolisation agents for Type 1 endoleak is associated with high technical success, but the effective elimination of endoleak and protection from continued sac expansion and rupture have not been demonstrated.[812] Basic balloon dilation or insertion of a bare metal balloon expandable stent may be effective in selected primary Type 1 endoleaks, but largely depends on the absence of neck dilatation, and its durability remains unclear.[813,814] Two recent systematic reviews failed to identify the ideal endovascular management strategy, mostly due to significant heterogeneity and risk of bias in the literature.[815,816] Open conversion can also be performed with acceptable results in patients fit for OSR, and may be considered as an alternative to complex endovascular procedures if performed electively in experienced centres.[798,817,818] In a recent meta-analysis, the pooled 30 day mortality rate for elective open conversions was only 2.8%, but increased to 28% for urgent conversions.[819]

Recommendation 103			Unchanged
Patients with Type 1 endoleak after endovascular abdominal aortic aneurysm repair are recommended for prompt re-intervention to achieve a seal, primarily by endovascular means.			
Class	**Level**	**References**	**ToE**
I	B	Antoniou *et al.* (2015),[790] Schlösser *et al.* (2009),[792] Doumenc *et al.* (2021),[798] Budtz-Lilly *et al.* (2023),[806] Rajendran and May (2017)[820]	

Recommendation 104			New
Patients with compromised sealing zones* without visible endoleak after endovascular abdominal aortic aneurysm repair may be considered for intervention to improve the seal, primarily by endovascular means.			
Class	**Level**	**References**	**ToE**
IIb	C	Budtz-Lilly *et al.* (2023),[806] Bastos Gonçalves *et al.* (2013),[821] Bastos Gonçalves *et al.* (2014),[822] Baderkhan *et al.* (2018),[823] Geraedts *et al.* (2021)[824]	

* Inadequate seal (< 10 mm) or progressive neck dilatation.

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Recommendation 105		New
For patients with a compromised proximal seal* after endovascular abdominal aortic aneurysm repair, proximal extension with fenestrated and branched devices should be considered in preference to other endovascular techniques.		
Class	**Level**	**References** ToE
IIa	C	Doumenc *et al.* (2021),[798] Martin *et al.* (2014),[800] Wang *et al.* (2018),[802] Dias *et al.* (2018),[803] Falkensammer *et al.* (2017),[804] Budtz-Lilly *et al.* (2023),[806] Perini *et al.* (2019),[815] Juszczak *et al.* (2021)[807] Juszczak *et al.* (2020)[825]

* Inadequate seal (< 10 mm) or progressive neck dilatation.

Recommendation 106		New
For selected patients with a compromised proximal seal* after endovascular abdominal aortic aneurysm repair, elective open conversion may be considered as an alternative to complex endovascular interventions, provided the surgical risk is acceptable.		
Class	**Level**	**References** ToE
IIb	C	Doumenc *et al.* (2021),[798] Dias *et al.* (2018),[803] Scali *et al.* (2014),[817] Arnaoutakis *et al.* (2019),[818] Goudeketting *et al.* (2019)[819]

* Inadequate seal (< 10 mm) or progressive neck dilatation.

7.2.6.2. Type 2 endoleak. T2ELs originating from collateral vessels, are the most common type of endoleak and may be detected early after EVAR or later during follow up. In a follow up study including 2 367 patients with EVAR, 18% had early T2ELs which resolved spontaneously, 5% had persistent T2ELs, and 11% developed new onset T2EL during follow up.[452] Approximately half of the patients with persistent or late endoleaks developed sac growth, with a 50% secondary intervention rate at two years. In a recent meta-analysis, including 2 643 patients with a T2EL from 33 observational studies, 54% were diagnosed before 30 days of follow up and 8% after 12 months. Early diagnosed T2EL had a significantly higher odds of resolving as compared with those detected late (OR 2.41). Sac expansion associated with T2EL was documented in 29% and rupture in 1.1%.[826]

Risk factors for persistent or secondary T2ELs are summarised in Table 21. Conversely, prior embolisation of aortic branches or non-selective sac embolisation during implantation reduce the risk of T2ELs (see also Chapter 5).[482]

In the presence of aneurysm sac growth because of a suspected T2EL, additional imaging (e.g., contrast enhanced ultrasound [CEUS], dynamic CTA or magnetic resonance angiography [MRA], or selective angiography) should be performed to rule out other causes of growth, namely inadequate sealing with associated Type 1 or Type 3 endoleak. It is estimated that at 20% of patients with endoleaks initially classified as Type 2 have in fact Type 1 or 3 endoleaks.[719,844,845] Different imaging modalities used for EVAR follow up and their benefits and downsides in detecting and classifying endoleaks are presented below.

Table 21. Risk factors associated with persistent or late developing Type 2 endoleaks after endovascular abdominal aortic aneurysm repair.

Risk factors consistently reported in literature
Absence of circumferential thrombus in the aneurysm sac or large flow lumen[455,827–831]
Number of patent aortic side branches arising from AAA[827,831,832]
Inferior mesenteric artery patency[454,455,828,830,831,833]
Number of patent lumbar arteries > 3[453–455,829,831–834]
Diameter of inferior mesenteric artery ≥ 3 mm[454,455,834]
Diameter of lumbar arteries ≥ 2 mm[453–455]
Anticoagulant therapy[835–839]
Risk factors inconsistently reported or uncertain
Coil embolisation of hypogastric arteries[840]
Increasing age[840,841]
Female sex[831,841]
Absence of chronic obstructive pulmonary disease[840,841]
Chronic renal disease[841]
Hypertension[841]
Graft type[840]
Absence of post-implant syndrome[842,843]
No smoking history[831]
No peripheral arterial disease[831]

Although most T2EL are benign, rupture has been described.[792] In a systematic review, < 1% of the T2ELs resulted in a rupture. This low rupture rate is however based on retrospective studies where intervention has often been performed for persistent T2EL with aneurysm sac growth, and thus the true natural history is unknown. Although most ruptures due to T2EL seem to occur in the presence of sac expansion, rupture has also been reported without sac expansion.[719] Notably, rapid expansion suggests an occult Type 1 or 3 endoleak, which adds to the complexity of diagnosis.[844] More evidence pointing towards alternative, or at least additional, causes of sac growth comes from a meta-analysis on the treatment success of T2ELs, reporting that despite a high technical success rate, embolisation frequently fails to arrest subsequent aneurysm growth[846,847] and robust evidence for the benefit of T2EL treatment is lacking.[846,848]

A recent publication using the VQI data linked to Medicare claims, including 1 372 patients with T2EL (25% of the total cohort), reported a 74% spontaneous resolution rate, and a median 1 – 1.5 mm decrease in aneurysm diameter (compared with a median 4 mm for those without endoleak). Notably, no difference in mortality or re-intervention rates were observed up to three years. Conversely, a recent publication from Japan, including 4 957 patients with T2EL from a total of 17 099 EVAR treated patients, showed T2EL to be associated with a higher risk of AAA related death (1%

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vs. 0.2%), AAA rupture (0.8% — fatal in 0.4%, *vs.* 0.1%), sac enlargement $\geq$ 5 mm (27.4% *vs.* 2.7%), and re-intervention for sac enlargement (14.9% *vs.* 0.7%). However, follow up for patients with T2EL was longer (4.6 *vs.* 3.9 years), and the overall mortality rate was not different. Also, the occurrence of delayed Type I or III endoleak was more frequent in patients with a prior T2EL (1.9% *vs.* 0.07%), which could help explain the worse outcomes.

Based on the above, there is no strong evidence for when intervention is indicated for T2EL, but it is reasonable to proceed to invasive treatment when the aneurysm has expanded $>$ 10 mm compared with baseline or the lowest diameter during follow up using the same imaging modality and measurement method.[719,846,849]

There is also uncertainty about the optimal treatment to resolve T2ELs. Various endovascular and open techniques have been described. Endovascular treatment consists of transarterial, translumbar, transperitoneal, transcaval, or trans-sealing (between iliac graft and iliac arterial wall) embolisation of the aneurysm sac and feeding vessels. Although endovascular treatment is associated with high technical success, endoleak recurrence is common[845,847] and a clear definition for successful intervention is lacking, affecting the interpretation of evidence. According to systematic reviews of low quality data, translumbar and transcaval embolisation may have a higher technical success and lower rate of complications than trans-arterial embolisation[719,850—852] and translumbar fusion guided embolisation with needle trajectory planning are superior to standard techniques.[853] Different embolic agents have been used, with the most frequent being coils of different types, alone or in combination with liquid embolic agents (ethylene vinyl alcohol). While the latter seems more effective, the actual value in arresting growth and preventing rupture remains unclear.[854] A recent publication about the safety and efficacy of transarterial liquid embolisation, noted that up to one quarter of patients suffered peri-operative complications and the endoleak was eliminated in less than half.[855] A recent publication suggests that treatment success can be significantly improved by using intra-operative CEUS combined with cone beam CT for guidance during translumbar embolisation.

Surgical treatment options include open ligation of side branches feeding the endoleak, suturing of the ostia of the leaking branch after opening the aneurysm sac, or stent graft explantation. This is obviously more invasive and usually reserved for cases where a prior endovascular intervention has failed to arrest aneurysm growth. Nevertheless, open conversion offers a definitive solution to persistent sac expansion and may be considered in elective situations for patients fit for open repair.[851] Outcomes comparable to those of primary open juxtarenal aneurysm repair can be achieved for elective open conversion, provided there is local expertise.[817,819] When proximal and distal seal are preserved and a T2EL is the plausible cause of sac expansion, endo-aneurysmorraphy with graft preservation may be performed with satisfactory results.[856] Partial graft removal is an interesting alternative that allows easy access to bleeding lumbar arteries while avoiding suprarenal clamping and extensive dissection.[857] Laparoscopic ligation of the IMA may also be considered, but there is limited evidence on its benefit.[858]

Recommendation 107 — **Changed**

Secondary intervention for a Type 2 endoleak after endovascular abdominal aortic aneurysm repair should only be considered in the presence of significant aneurysm sac growth (≥ 10 mm compared with baseline or with the smallest diameter during follow up using the same imaging modality and measurement method), primarily by endovascular means, provided alternative causes including Type 1 or 3 endoleaks have been excluded.

Class	Level	References	ToE
IIa	C	Sidloff *et al.* (2013),[719] Madigan *et al.* (2019),[844] Wu *et al.* (2021),[845] Mulay *et al.* (2021),[846] Ultee *et al.* (2018),[847] Dijkstra *et al.* (2020),[849] Mansukhani *et al.* (2023)[859]	

Recommendation 108 — **New**

Patients with persistent aneurysm growth after endovascular attempt(s) to treat Type 2 endoleaks should be considered for elective open conversion with or without graft preservation.

Class	Level	References	ToE
IIa	C	Dias *et al.* (2018),[803] Goudeketting *et al.* (2019),[819] Madigan *et al.* 92019),[844] Wu *et al.* (2021),[845] Ultee *et al.* (2018)[847]	

7.2.6.3. Type 3 endoleak. An endoleak resulting from stent graft component separation or fabric tear is classified as Type 3. These endoleaks may occur due to maldeployment of stent grafts with inadequate overlap, stent graft migration (Type 3a), or material fatigue (Type 3b). Occasionally, device failure or imminent component disconnection is noted before endoleak develops.

It is estimated that stent graft failure occurs in 1 — 3% of patients after EVAR, and the incidence increases with off label use.[860,861] When sac expansion is observed during follow up, disruption of the graft may be suspected, but it is notably difficult to obtain a definitive diagnosis based on CTA. In a recent systematic review, prior diagnosis was only achieved in 20% of confirmed cases.[861] Contrast enhanced DUS was not found to improve the detection rate compared with CTA alone (28% *vs.* 29%) in a meta-analysis[862] and multimodal imaging including conventional DSA with proximal and distal balloon occlusion may be necessary to ensure optimal treatment.[863]

Like Type 1 endoleaks, Type 3 endoleaks expose the aneurysm to direct aortic pressure with subsequent risk of rupture.[792] Therefore, prompt intervention is warranted. Management of Type 3a endoleaks is usually

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straightforward, with the use of an extension limb to bridge the separated components. Occasionally, conversion to AUI may be necessary.[718] Type 3b endoleaks may be more challenging, depending on the location of the defect. Often, the exact location is impossible to determine. These endoleaks may be repaired by re-lining the defect, but a tailored approach is necessary. Conversion to open repair with or without graft preservation remains an acceptable option for suitable patients, especially after failed endovascular attempts.[423]

Exceptionally high frequencies of late Type 3 endoleaks have been reported for the AFX Endovascular AAA System (Endologix, Irvine, CA, USA) (see Chapter 5). Long term imaging surveillance is critical, and a low threshold for complete relining should be considered with any sign of sac enlargement, even if endoleak is not clearly demonstrated in patients with AFX grafts.[423]

Recommendation 109		Changed
For patients with Type 3 endoleak after endovascular abdominal aortic aneurysm repair, prompt re-intervention is recommended, primarily by endovascular means.		
Class	Level	References ToE
I	C	Maleux *et al.* (2017),[718] Schlösser *et al.* (2009),[792] Kwon *et al.* (2020),[860] Lowe *et al.* (2020),[861] Gennai *et al.* (2023)[864]

7.2.6.4. Type 4 endoleak. Leakage of blood through the stent graft due to material porosity in the early post-operative period is defined as a Type 4 endoleak. There is, however, only a single report of rupture resulting from this form of endoleak in two systematic reviews.[790,792] Due to improvements in graft materials, Type 4 endoleaks are rarely seen and may be considered transitory and benign.

7.2.6.5. Persistent aneurysm sac growth without visible endoleak. Occasionally, persistent aneurysm sac growth is noted without any visible endoleak. This has also been termed endotension or Type 5 endoleak. Several possible mechanisms have been suggested, including increased graft permeability, resulting in direct transmission of pressure through the graft to the aortic wall.[865] Historically, the first generation Gore Excluder stent grafts had a high rate of sac expansion due to endotension caused by graft permeability but this changed in 2004 with the introduction of a low porosity fabric and is no longer an issue.[866,867] Although endotension may result in AAA rupture, this is exceedingly rare.[790,792] In a series of 100 patients requiring stent graft explantation, endotension was the reason in only six cases.[868]

Most cases probably result from an endoleak which cannot be visualised with standard imaging modalities,[869,870] so efforts should be made to rule out other sources of endoleak, including multimodality imaging (see section 7.4.3). It may also be the result of failing sealing zones without overt endoleak. A multicentre retrospective study including 255 open conversions reported on the presence of occult endoleaks in 32 (12.5%) of patients at the time of conversion, the majority (78%) being Type 1 or 3. When endotension was the original diagnosis (25/255 cases), Type 1 or 2 endoleaks were identified in 15% and unidentified infection in 20%.[871]

As with T2EL, treatment is usually considered when there has been significant sac growth (> 10 mm). Aortic stent graft relining should be considered as the first line treatment if concern exists for graft integrity in older generation stent grafts, there is adequate room to extend the proximal and or distal seal zones, or patient risk profiles unsuitable for general anaesthesia or OSR. For patients with a favourable risk profile for OSR in the setting of failed stent graft relining or insufficient proximal and or distal seal zones, open stent graft explantation may be favoured.[872]

Recommendation 110		Unchanged
Patients with significant aneurysm sac growth (≥ 10 mm compared with baseline or with the smallest diameter during follow up using the same imaging modality and measurement method) after endovascular abdominal aortic aneurysm repair, without visible endoleak on standard imaging, should be considered for further diagnostic evaluation with alternative imaging modalities to exclude the presence of an occult endoleak or infection.		
Class	Level	References ToE
IIa	C	Schlösser *et al.* (2009),[792] Bussmann *et al.* (2017),[869] Perini *et al.* (2022)[871]

Recommendation 111		Unchanged
Patients with significant aneurysm sac growth (≥ 10 mm compared with baseline or with the smallest diameter during follow up using the same imaging modality and measurement method) after endovascular abdominal aortic aneurysm repair, without visible endoleak after multimodality imaging, should be considered for stent graft relining or conversion to open surgical repair.		
Class	Level	References ToE
IIa	C	Schlösser *et al.* (2009),[792] Bussmann *et al.* (2017),[869] Perini *et al.* (2022)[871]

7.2.7. Stent graft migration. Stent graft migration is usually defined as movement of the stent graft > 10 mm compared with fixed anatomical landmarks verified on flow centreline CT reconstructions, or any migration resulting in symptoms or secondary intervention. While proximal stent graft migration was a common event with the early generation stent grafts, the development of active supra- or infrarenal fixation in modern stent grafts has reduced its incidence significantly.[795,873,874] Migration may result in Type 1 endoleak, stent graft separation, kinking, or graft occlusion. Risk factors for proximal migration include short proximal fixation, angulated neck, large aneurysm size, large

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diameter proximal neck, stent graft type, and excessive dilatation of the proximal neck.[793,873,875–882] The role of oversizing is controversial, but there are indications that stent graft oversizing of > 30% may contribute to the risk of migration.[883,884] Disease progression with neck dilatation may be a cause of late migration, and is related to initial neck diameter.[793,875,880,881,885] In a recent meta-analysis, the pooled incidence of post-EVAR aortic neck dilatation was 22.9% over a follow up period ranging from one to 14 years. Aortic neck dilatation was significantly associated with the risk of Type 1 endoleak and stent graft migration (OR 2.95 and 5.95 respectively).[885] Rarely, disconnection of the suprarenal bare stent may cause migration and Type 1 endoleak.[886,887]

Cephalad migration may also occur at the distal landing zone of the stent graft, due to changes in aneurysm morphology, shrinkage of the aneurysm sac or graft movements in aneurysms with large flow lumina.[712,797] An iliac fixation length of > 20 mm has been suggested to reduce the risk of proximal stent graft migration.[712,888] EVAR with flared iliac limbs is associated with a higher risk of distal endoleaks.[712,888–890]

When proximal or distal migration are associated with Type 1 endoleak or compromised seal, the principles of management are similar to those mentioned in the corresponding chapter above. Selected cases of stent graft migration conserving long, non-compromised seal zones may be managed conservatively, however.

7.3. Follow up after open surgical repair for abdominal aortic aneurysm

Scheduled imaging after OSR is aimed at detecting possible asymptomatic complications like anastomotic pseudoaneurysm or progression of disease. A long term follow up study (mean 87 months) revealed aneurysms in non-contiguous arterial segments in 45% of patients, most requiring no treatment due to small size, and 19% had multiple late synchronous aneurysms.[708] An incidence of femoral or popliteal aneurysms of up to 14%[891] and of thoracic aortic aneurysms of 13%[892] has been reported after OSR for AAA.

No high level evidence is available regarding the potential benefit of post-operative imaging surveillance after OSR of AAA. Nevertheless, the risk of late para-anastomotic aneurysm and recurrent aortic aneurysm and peripheral aneurysm formation makes it reasonable to consider imaging surveillance of all patients after OSR of AAA, who are fit for treatment if a new aneurysm is detected.

The five year period interval is not supported by any hard evidence. It is based, however, on the expected time to develop late complications like anastomotic aneurysms and on the natural history for development of metachronous aneurysms.[709]

MRA or CTA scanning is the method of choice to detect para-anastomotic aneurysms and new true aortic aneurysms early[788] and DUS is the method of choice for peripheral aneurysms.

Table 22. Pros and cons of different follow up imaging modalities after endovascular abdominal aortic aneurysm repair.

	CTA	CT	DUS	CEUS	MRA
Availability	+++	+++	+++	+	+
Cost	++	++	+	+++	+++
Capacity to detect complications	+++	+	++	+++	+++
Assessment of aortic branches	+++	+	++	++	+++
Operator dependency	−	−	+++	+++	++
Nephrotoxicity	+++	−	−	−	+
Radiation exposure	+++	+++	−	−	−

+ / − indicate different degrees of importance for the respective characteristics.
EVAR = endovascular aneurysm repair; CTA = computed tomography angiography; CT = computed tomography; DUS = duplex ultrasound; CEUS = contrast enhanced ultrasound; MRA = magnetic resonance angiography.

Recommendation 112		Unchanged
Patients who have undergone open surgical repair for abdominal aortic aneurysm may be considered for imaging follow up of the entire aorta and peripheral arteries every five years.		
Class	**Level**	**References** ToE
IIb	C	Serizawa *et al.* (2021),[709] Diwan *et al.* (2000),[891] Chaer *et al.* (2012)[892]

7.4. Follow up after endovascular aortic repair

7.4.1. Imaging modalities for endovascular aortic repair follow up. The aim of post-operative imaging is to predict or detect complications. Various imaging modalities can be used during EVAR follow up. A list of imaging modalities and their pros and cons is presented in Table 22. Generally, CTA and or DUS form the basis for EVAR follow up imaging.[893]

CTA permits the assessment and detection of most EVAR complications. Typically, this involves dual (arterial and delayed) or triple phase (adding a native stage), thin slice (1 mm) scans.[894] An alternative using split bolus contrast injection has been proposed, reducing radiation exposure by 42% but increasing contrast administration from 100 cc to 130 mL.[895] CTA may also detect other incidental findings.[896] Non-contrast CT is limited as a standalone modality but may be complemented by DUS.

It is important to consider the cumulative risk of cancer resulting from repeated radiation exposure, especially in young patients with long life expectancy. The EVAR 1 trial suggested that a higher incidence of malignancy in the EVAR group resulted from such radiation increment, and another study also suggested a similar effect.[466,897] The ESVS recently issued recommendations on radiation safety, which also apply to surveillance after EVAR.[363] For further information, please consult the aforementioned document.

MRA can be used as an alternative to CTA with comparable results. In a systematic review comparing MRA and CT, MRA was more sensitive in detecting T2Els.[898] Using blood pool contrast agents in combination with T1 weighted MRA the delay between injection and imaging can be extended, improving visualisation of Type 2 (and Type 4) endoleaks.[899] MRA may therefore have a specific role in imaging patients with post-EVAR sac growth where CTA is negative or inconclusive. Stainless steel and cobalt—chromium—nickel stents are ferromagnetic and may result in significant artefacts.[900] Importantly, the heating effects and pulsatile drag forces that the magnetic field exerts on both stainless steel and nitinol stent grafts is generally considered harmless if 1.5 Tesla fields are used.[901,902]

DUS is an accepted alternative to CTA for EVAR follow up and is highly sensitive in detecting complications such as endoleaks.[903,904] DUS offers the possibility of repeated and reliable measurement of maximum aneurysm diameter at low cost and without exposing the patient to ionising radiation or nephrotoxic contrast. Diameter measurements with DUS cannot be directly compared with CT measurements,[905] and thus to assess sac dynamics post-EVAR, repeat examinations with the same imaging modality are required. The accuracy of DUS may be increased with the use of echogenic contrast.[906] Combination of 3D volume measurement and CEUS may further increase the role of DUS in EVAR follow up imaging.[907] US contrast agents (perfluorocarbon or sulphur hexafluoride) have contraindications that include unstable angina, a recent episode of acute coronary syndrome and severe pulmonary hypertension. Disadvantages of DUS include operator and equipment dependency, patient related factors (e.g., obesity, hernias, presence of calcification), and inability to assess sealing zone length, stent graft overlap, and device migration.

3D-CEUS has been shown to be more sensitive than CTA in identifying endoleaks and more accurate at defining the source and type of endoleak.[908] Digital tomosynthesis consists of an arbitrary number of section images from a single pass of the Xray tube. Combined with CEUS, it has been shown to be effective for the diagnosis of EVAR related complications. Digital tomosynthesis shows a good accuracy and negative predictive value (98% and 99% respectively), correctly identifying all graft fractures and migrations, despite underestimation of endoleaks, that are easily recognised by CEUS.[909]

In a meta-analysis of 21 studies comparing DUS with CTA, the sensitivity of DUS detecting endoleaks was 0.77 and specificity 0.97. Addition of US contrast increases the sensitivity of DUS to 0.98 but reduces specificity to 0.88.[893] A systematic review showed that both MRA and DUS may be more sensitive than CTA for detection of T2ELs. For the detection of Type 1 or 3 endoleaks, however, DUS and MRI offer no advantage.[910] A more recent meta-analysis, including 26 studies and 2 217 patients, investigated the diagnostic accuracy of CEUS for detection of endoleak. This study found that sensitivity and specificity of CEUS for all endoleaks were 0.94 and 0.93, respectively. For Type 1 or 3 endoleaks it was 0.97 and 1.00.[911] In a recent study, investigators tested the agreement between CTA and DUS for detecting clinically significant complications, and found a kappa of 0.91, meaning very good agreement. However, the sensitivity of DUS was only 89% compared with CTA, and some important complications related to loss of seal were missed by DUS.[912] Another recent publication, the ESSEA trial (Echo doppler vs. Scanner injecté pour le Suivi des Endoprotheses Aortiques Abdominals), investigated the accuracy of DUS in detecting major AAA related morphological abnormalities (Type 1 or 3 endoleaks, $\geq$ 70% limb stenosis, T2ELs with $\geq$ 2 mm sac expansion, or any sac expansion $\geq$ 5 mm) compared with CTA in a sample of 539 patients with EVAR. The negative predictive value and positive predictive value of DUS, compared with CTA, were 92% and 39%, respectively. The positive likelihood ratio was 4.87. DUS sensitivity reached 73% in patients requiring secondary interventions. The authors concluded that DUS had an overall low sensitivity to detect AAA related morphological abnormalities after EVAR, but this was improved in patients being considered for intervention.[913] In a retrospective study comparing the diagnostic accuracy of DUS and CEUS (CTA as the gold standard), the sensitivity and specificity were 46% *vs.* 93%, and 85% *vs.* 95%, respectively. CEUS and CTA were diagnostically equivalent, as opposed to DUS and CTA. All endoleaks detected by CTA that resulted in secondary interventions were detected by CEUS, but not all by DUS.[914]

Dynamic (time resolved) CTA has also been used increasingly with success in cases where the origin of endoleaks is obscure or their classification is unclear. By comparing the contrast phase inside the endograft and in the endoleak, it is possible to distinguish with a high level of certainty between a direct endoleak (Type 1 or 3) and a Type 2.[915] Despite being a promising technique, further data are needed and the described protocols vary greatly and require optimisation.

7.4.2. Endovascular aortic repair follow up regimens. Owing to the risk of graft related complications and late rupture after EVAR, regular imaging follow up has been regarded as mandatory. Despite recommendations from companies and guidelines from scientific and regulatory bodies, follow up protocols vary significantly between centres.[916,917] Repeated prophylactic imaging incurs significant cost and resource consumption, with implications for health economic evaluations.[918]

Three meta-analyses failed to demonstrate any survival advantage for patients with complete image follow up (*vs.* incomplete or no imaging), despite a higher rate of secondary interventions for patients with complete follow up.[919–921] The most recent meta-analysis, including 22 762

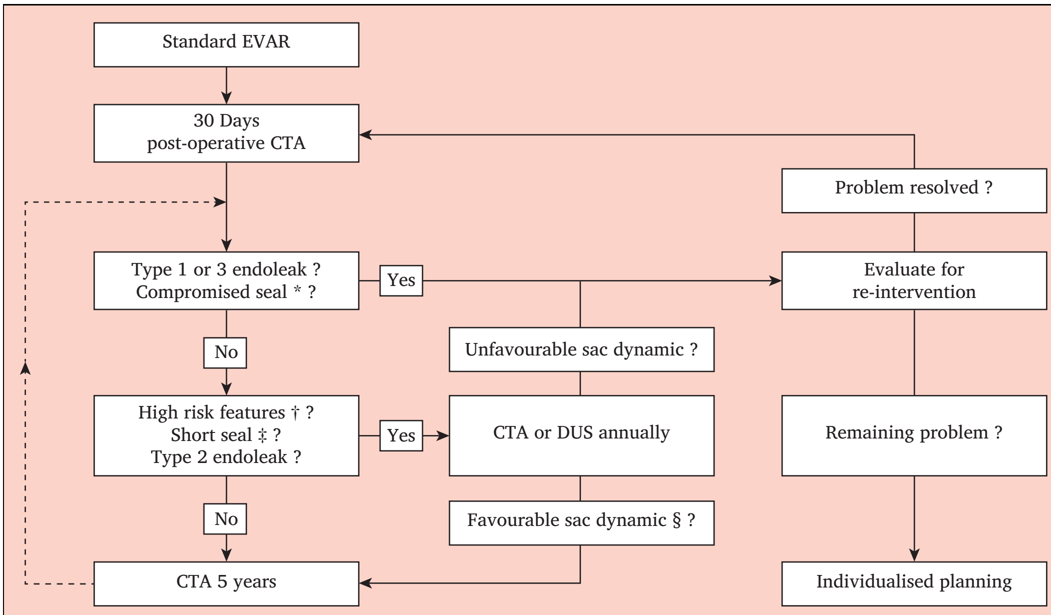

Figure 6. Recommended follow up algorithm after standard endovascular aneurysm repair (EVAR), according to Recommendations 112, 113, and 114. Not applicable to new EVAR device systems, non-standard technology, or complex EVAR. * Degradation of seal zones with impending endoleak, † Proximal neck diameter > 30 mm, proximal neck angulation > 60°, iliac diameter > 20 mm, investigational/new device, ‡ Proximal and distal seal < 10 mm, § Shrinkage > 5 mm from baseline when measured with same imaging modality. DUS = duplex ultrasound; CTA = computed tomography angiography.

patients from 13 cohort studies, could not demonstrate any difference in all cause mortality, aneurysm related mortality, or secondary intervention between patients who had incomplete or complete follow up after EVAR. Surprisingly, the odds of aneurysm rupture were lower in non-compliant patients (OR 0.63), which the authors termed the EVAR surveillance paradox.[921] However, heterogeneous surveillance protocols, observational and usually retrospective study design and lack of robust information on causes of death, coupled with concerns raised by the very long term outcomes of EVAR trials do not justify recommendations against image surveillance after EVAR.[922]

Early (within 30 days) post-operative imaging follow up after EVAR aims to assess the success of the intervention, i.e., aneurysm exclusion without access complications. CTA is preferred for this purpose and its findings have been shown to have the strongest prognostic importance (see below). DUS examination can be used in alternative to verify the absence of endoleaks and assess limb patency and flow, but since it lacks assessment of stent graft overlap, seal length, and kink, it may need to be completed with non-contrast CT.

Intra-operative angiography combined with cone beam CT for completion assessment could possibly replace the early (30 day) post-operative CTA.[923] A recent French single centre study found that use of a combination of intra-operative contrast enhanced cone beam CT and post-operative CEUS (*vs.* completion angiography followed by CTA) was significantly associated with a reduced rate of late stent graft related complications but did not appear to significantly protect against stent graft related re-interventions or all cause death.[924] Further investigations are required before its use in clinical practice can be determined.

Risk stratification and reduction of unnecessary imaging is an appealing way to improve the efficacy of post-operative follow up strategies. This can be performed based on anatomical risk and on early post-operative imaging, which has been shown to predict complications in a reliable way.[716,821–824,925,926]

Anatomical risk has been consistently found to predict future complications (Brown BJS 2010). Patients undergoing EVAR outside the manufacturer's IFU, an indirect measure of anatomical risk, have an increased risk of late failure, presumably because of lack of adequate seal.[413,415] Also, specific characteristics like wide (≥ 30 mm) or severely angulated aortic necks or ectatic iliac arteries, even within IFU, may suffer more rapid degeneration and therefore may need special attention.[712,927–929] However, pre-operative information alone may not be discriminative enough to guide decisions on risk stratification.[922,930]

Several studies have focused on the prognostic value of the first post-operative examination, mostly using CTA. Two important advantages exist: the actual seal achieved can be assessed; and T2ELs can be signalled. The concept is that only patients with a sub-optimal seal zone and or presence of endoleaks require routine imaging, at least during the first years after EVAR, while the remaining low risk patients may undergo imaging only if symptoms develop.[821,823,931]

An alternative (possibly complimentary) strategy consists of evaluating the early (up to two years) evolution of the aneurysm sac. If shrinkage (> 5 mm) is observed, routine imaging can be waived as this is a proxy of successful exclusion and a very low complication rate can be expected.[415,927,932]

Based on the above, a suggested follow up algorithm after EVAR (Fig. 6) would include early post-operative imaging for risk stratification into three groups:

- *The low risk group* (no endoleak, anatomy within IFU, without high risk features [proximal neck diameter < 30 mm and angulation $< 60°$, and iliac diameter < 20 mm], adequate overlap, and seal of ≥ 10 mm proximal and distal stent graft apposition to arterial wall) could be considered for limited follow up, with delayed imaging until five years after repair. At five years, CTA of the entire aorta and iliac arteries (or DUS + CT) is preferable, to assess for sustained EVAR success as well as progression of disease. It is estimated that this group may constitute about two thirds of all patients with EVAR.[823]
- *The high risk group* (presence of T2EL, insufficient overlap or seal < 10 mm, anatomy outside IFU, large proximal neck [> 30 mm], ectatic iliac fixation zones [> 20 mm], or extreme angulation [$> 60°$]) could be considered yearly examinations with either CTA or DUS. At each time point, re-evaluation of risk is necessary. Patients with sac shrinkage ≥ 10 mm can be regarded as low risk of failure, cross over to the low risk group and repeat imaging only five years after the operation.
- *EVAR failure group* (direct endoleak; Type 1 or 3 endoleak), obvious degradation of seal zones with impending endoleak, or aneurysm sac growth > 10 mm) should be considered for secondary intervention.

The clinical success of EVAR beyond five years after repair is less studied, as most current reports focus on five year results.[413,821,822,933] Worrying long term data suggesting a very long term increase in aortic events after EVAR,[466] possibly due to disease progression, indicates the need for long term imaging follow up of all patients with EVAR, suggested every five years, regardless of initial risk stratification.

This EVAR follow up scheme is indicated for standard EVAR devices with proven durability. Complex EVAR procedures, such as fenestrated and branched EVAR, patients treated with chimney grafts, or new EVAR device systems based on non-standard technology, require individualised follow up based on device, repair, and perceived risk of late failure.

Imaging after EVAR is only beneficial if an intention to treat complications electively exists. It is not uncommon to face the ethical dilemma of continuing or withholding surveillance in very elderly, frail or dependent individuals. There is no clear evidence to guide such decisions. However, good clinical judgement suggests no surveillance should continue to be offered to patients who are not considered candidates for elective secondary interventions. In the case of discharge from surveillance, patients may still be offered treatment with reasonable results in case of acute symptoms, whenever justified.[819]

Adherence to follow up is a critical aspect that should be stressed at each patient visit. This may be especially challenging for patients stratified as low risk. However, lifelong follow up after any form of AAA repair is mandatory for maintained treatment success.

Recommendation 113		Unchanged
Patients who have undergone endovascular abdominal aortic aneurysm repair are recommended early post-operative imaging (within 30 days) using computed tomography angiography, to assess the presence of endoleak, component overlap and sealing zone length.		
Class	**Level**	**References ToE**
I	B	Karthikesalingam *et al.* 2010),[716] Bastos Gonçalves *et al.* (2013),[821] Bastos Gonçalves *et al.* (2014),[822] Baderkhan *et al.* (2018),[823] Geraedts *et al.* (2021)[824]

Recommendation 114		Changed
Patients who have undergone endovascular abdominal aortic aneurysm repair and have been stratified as low risk of complications* based on early post-operative computed tomography angiography should be considered for low frequency imaging follow up during the first five years.		
Class	**Level**	**References ToE**
IIa	C	Bastos Gonçalves *et al.* (2013),[821] Bastos Gonçalves *et al.* (2014),[822] Baderkhan *et al.* (2018),[823] Geraedts *et al.* (2021),[824] Patel *et al.* (2010),[926] Antoniou *et al.* (2020)[927]

* No endoleak, anatomy within IFU, adequate overlap and seal of ≥ 10 mm proximal and distal stent graft apposition to arterial wall.

Recommendation 115		New
Patients who have undergone endovascular abdominal aortic aneurysm repair are recommended for long term imaging follow up (regardless of initial risk stratification), to detect late complications and identify late device failure and disease progression.		
Class	**Level**	**References ToE**
I	B	Patel *et al.* (2016),[466] Geraedts *et al.* (2022),[917] de Mik *et al.* (2019),[919] Grima *et al.* (2018),[920] Wanken *et al.* (2020)[922]

7.4.3. Diagnostic step up for occult undetermined endoleaks. When faced with an endoleak of unclear origin, when a Type 1 or 3 endoleak need to be ruled out concomitant with aT2EL, or when expansion is present but there is no visible endoleak, uncertainty exists regarding the following diagnostic steps. The first step is usually to perform either a CTA or DUS, depending on the primary follow up modality. If, despite the information from both CTA and DUS, doubt remains, CEUS may be considered as a second step, possibly incorporating 3D-CEUS to increase sensitivity. If CEUS is unavailable, contraindicated or inconclusive, dynamic CTA or MRA with a blood pool agent may be used as an alternative. In a study on open conversion, 20% of patients with growth and no endoleak presented unexpectedly with infection.[871] As such, when no other cause of growth is identified, ^{18}F-FDG/PET-CT or WBCS may be considered to rule out occult infection or the presence of an angiosarcoma. Direct aneurysm sac puncture with standard culturing combined with 16S rRNA/18S rRNA may further help to determine an occult microbiological aetiology.[934]

Diagnostic angiography, preferably with temporary proximal and or distal balloon occlusion, is usually reserved for cases where uncertainty persists despite multiple non-invasive imaging. When performed, materials should be readily available to allow for a definitive treatment whenever possible. Depending on local availability and logistics, adaptations of this protocol may be appropriate. Ultimately, stent graft relining or conversion to OSR should be considered as bailout (see section 7.2.6.5. Recommendation 110). Figure 7 displays a suggested algorithm for diagnostic step up for occult undetermined endoleaks.

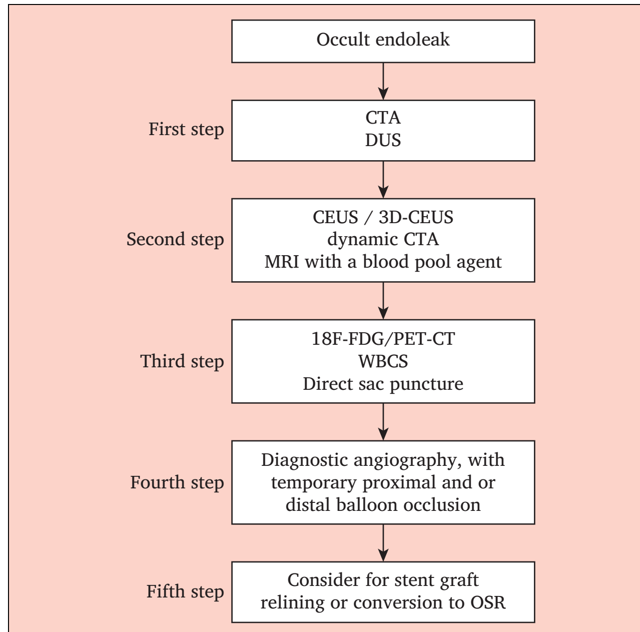

Figure 7. Suggested diagnostic step up for occult undetermined endoleaks after endovascular abdominal aortic aneurysm repair. CTA = computed tomography angiography; DUS = duplex ultrasound; CEUS = contrast enhanced ultrasound; MRI = magnetic resonance imaging; ^{18}F-FDG/PET-CT = positron emission tomography with 2-deoxy-2-fluorine-18-fluoro-D-glucose; WBCS = white blood cell scintigraphy; OSR = open surgical repair.

8. MANAGEMENT OF COMPLEX ABDOMINAL AORTIC ANEURYSMS

8.1. Definition and indications for repair of complex abdominal aortic aneurysms

Abdominal aortic aneurysms involving the renovisceral segment (without the involvement of the thoracic aorta) are collectively termed complex AAAs and include the following subgroups (Fig. 8).[935]

- Short neck infrarenal AAA: with an infrarenal aortic neck 4 − 10 mm in length.
- Juxtarenal AAA: with an infrarenal aortic neck < 4 mm in length, without direct involvement of the renal arteries.
- Pararenal AAA: with the involvement of at least one of the renal arteries but not the SMA.
- Paravisceral AAA: with the involvement of the renal arteries and the SMA, but not the coeliac artery.
- Suprarenal AAA: pararenal and paravisceral AAAs are frequently grouped together as suprarenal AAA.
- Type IV TAAA: with the involvement of the renal arteries, the SMA, and the coeliac artery. Thus, TAAA IV involves the entire abdominal aorta from the level of the diaphragm to the aortic bifurcation.

This chapter will include all the above mentioned complex AAAs. For management of small complex AAAs, see also Chapter 4. Special considerations regarding saccular and para-anastomotic pseudoaneurysms as well as dissections are addressed in Chapter 10, and for advice on type I − III and type V TAAA the ESVS guidelines on the Management of descending thoracic aorta disease should be consulted.[936]

Importantly, there is no clear threshold diameter for when an AAA neck or the renovisceral segment can be considered aneurysmal, and thus classified as a complex AAA. There is a gradual transition from a normal neck diameter (< 25 mm), to an ectatic neck (25 − 29 mm), and further to an aneurysmal neck (> 30 mm). Today's standard EVAR devices are available in sizes that can accommodate both ectatic and slightly aneurysmal necks, up to 32 mm (see section 5.3.2). Also in OSR, a large neck can usually be incorporated into a surgical graft. This creates a grey area as to whether an AAA should be managed as an infrarenal AAA, with standard repair, or as a complex AAA, with potentially more advanced repair methods. In practice, the management of these borderline AAAs is determined by factors such as fitness, age, and patient's preference. In a comorbid or old patient a more basic procedure with less immediate risks and shorter durability may be chosen, while in younger patients a more durable solution by means of complex repair is often justified.

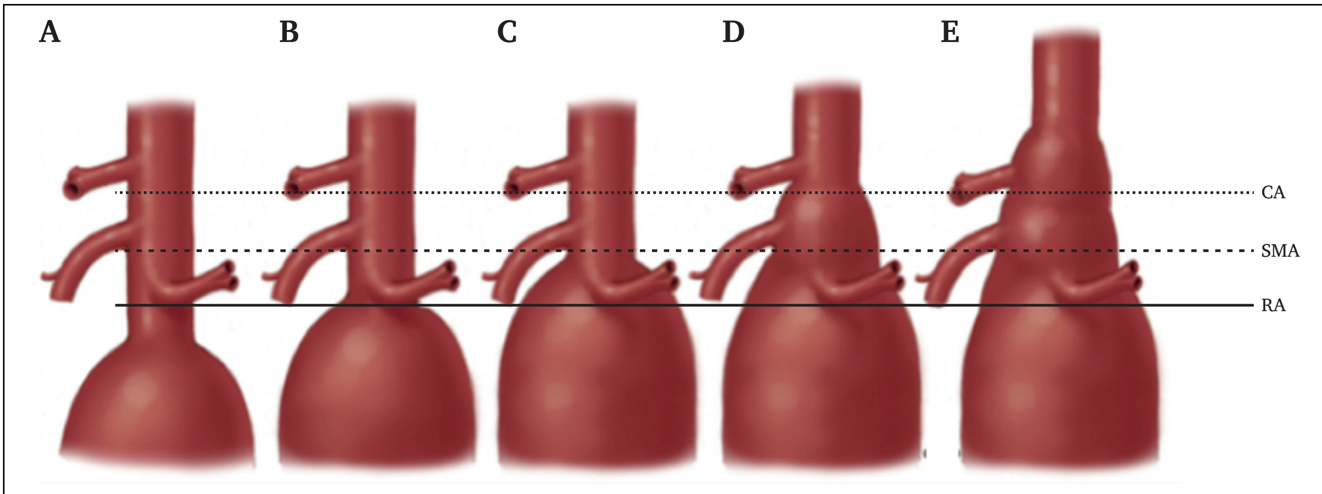

Figure 8. Anatomical classification of complex abdominal aortic aneurysms (AAAs) based on the proximal extensions of the aneurysm and their relationship with the renal arteries (RAs), the superior mesenteric artery (SMA), and the coeliac artery (CA). (A) Short neck infrarenal AAA. (B) Juxtarenal AAA. (C) Pararenal AAA. (D) Paravisceral AAA. (E) Type IV thoraco-abdominal aortic aneurysm. Pararenal and paravisceral AAAs are frequently grouped together as suprarenal AAA. Permission to reproduce granted from Elsevier *J Vasc Surg.*[935]

Complex AAAs are estimated to constitute about 15 – 20% of all AAAs. There are no data available on rupture risk and natural history specifically for complex AAAs. Because of the lack of evidence for this specific subgroup and the fact that complex AAA repair carries a higher risk, an individualised approach regarding indication for repair is appropriate. This is reflected in the weak recommendation (Class IIb) of a minimum threshold of 55 mm for when elective repair of complex AAA may be considered in men and 50 mm in women, whereas in practice a larger threshold diameter may be more appropriate in patients with increased comorbidities or more complex anatomy. In most published case series, patients were treated by open or endovascular repair when the mean or median diameter of the aneurysm was 60 mm. It is worthwhile to reiterate the (negative) Class III recommendation not to repair AAA below 55 mm (Recommendation 21, section 4.4), which understandably also applies to complex AAAs.

Patients with small complex AAAs can be kept under surveillance with US using the protocol for infrarenal AAA. For accurate pre-operative planning, CTA with one mm slices is recommended, allowing for 3D reconstructions for accurate measurement of the target vessels (see section 5.1).[937]

Recommendation 116			**Changed**
Patients with complex abdominal aortic aneurysms may be considered for elective repair at a diameter of ≥ 55 mm in men and ≥ 50 mm in women, taking into account fitness for repair, aneurysm anatomy, and patient preferences.			
Class	Level	References	ToE
IIb	C	Lancaster *et al.* (2022),[76] Ulug *et al.* (2020),[243] Cambria *et al.* (1995),[938] Hansen *et al.* (2010),[939] Piffaretti *et al.* (2019)[940]	

8.2. Elective repair of complex abdominal aortic aneurysms

8.2.1. Open surgical repair. While short neck AAAs present with an inadequate proximal sealing zone for standard EVAR, it is often possible to cross clamp the aorta below the renal arteries. Thus, the OSR is comparable with a standard OSR of an infrarenal AAA (see Chapter 5).

In juxtarenal AAAs, aortic cross clamping above one or both the renal arteries, with selective clamping of the renal arteries below the aortic clamp, may be required. Selective renal perfusion can be performed through an occlusion or perfusion catheter inserted from inside the aorta. The proximal anastomosis is usually performed just below the inferior border of the lower renal artery.

In suprarenal AAAs, suprarenal or supravisceral aortic cross clamping is required. The renal or visceral vessels are selectively perfused and reattached directly to the aortic graft or through selective bypasses.

OSR for a Type IV TAAA may be performed through a left thoracophrenolaparotomy in the VII-VIII intercostal space with partial circumferential phrenic incision, or through a subcostal laparotomy with medial visceral rotation. The renal and visceral vessels are selectively perfused and reattached as a single island of aortic wall in a bevelled aortic anastomosis, or as a single island of aortic wall including the visceral and renal vessels (Carrel patch) to an oval opening in the graft, or through selective vessel reattachments (branched grafts).[941,942]

The level of aortic clamping affects the outcome after OSR. In a study from the USA NSQIP, including 615 OSR for complex AAAs, clamp location above one or above both renal arteries was associated with no difference in mortality (3.5% *vs.* 2.1%) or renal dysfunction (6.9% *vs.* 4.9%). In contrast, supracoeliac clamping compared with clamping above one or both renal arteries was associated with a

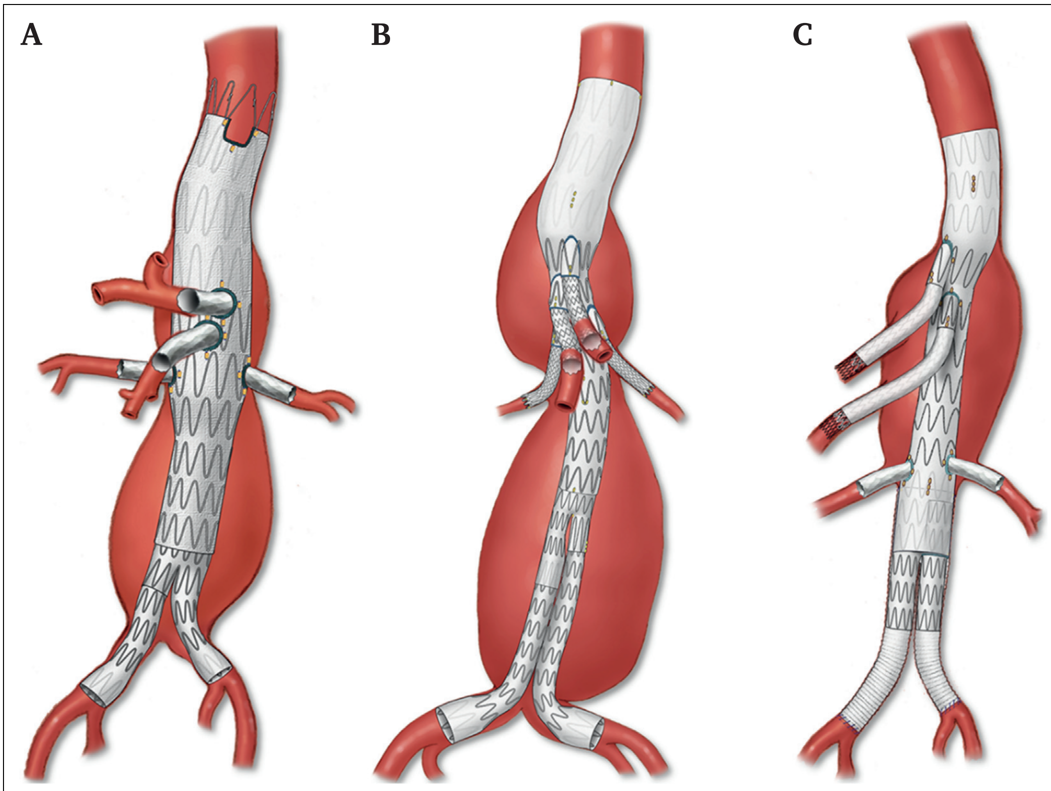

Figure 9. (A) Fenestrated endovascular aortic repair (fEVAR), (B) branched EVAR (bEVAR), and (C) f/bEVAR configurations. Permission to reproduce granted from Elsevier *J Vasc Surg*.[935]

higher mortality rate (8.0% *vs*. 2.8%) and increased rate of renal dysfunction (12% *vs*. 6.0%) and unplanned re-operations (24% *vs*. 10%). Thus, supracoeliac clamping should be avoided if clamping above one or both renal arteries is technically possible.[943]

There are no data to prefer one surgical approach or reconstruction technique over another, but this must be determined by individual patient's factors, such as extent of disease, and local preferences.

Several systematic reviews have provided a benchmark for complex AAA OSR.[944–946] In a systematic review of 21 case series comprising 1 575 patients, 30 day or in hospital mortality after open juxtarenal AAA repair was 4.1%. The mean AAA diameter at surgery was 61 mm and the mean age was 71 years. Fourteen per cent of the patients had post-operative renal dysfunction whereas permanent dialysis was necessary in 3% of patients.[946] Interpretation of the data is hampered because of the wide range of definitions for renal dysfunction applied in the various studies included in the review. In a series of patients included in the Vascular Study Group of New England registry, peri-operative mortality was 3.6% in 443 patients after elective OSR for a juxtarenal AAA or pararenal AAA, with 20% renal complications and 1% need for permanent dialysis.[947]

8.2.2. Fenestrated and branched endovascular aortic repair. Endovascular repair with fenestrated and or branched endografts (f/bEVAR) has become the treatment of choice of complex AAAs in most high volume centres.[935,948] bEVAR off the shelf devices may be an option for treatment of symptomatic or very large complex AAAs, when custom made solutions are not available.[949,950] PMEGs and *in situ* laser fenestration should be reserved for urgent patients, for whom the waiting time for manufacturing a custom made device (CMD) is too long or when a suitable off the shelf device is not available.[950]

Device specific contraindications for f/bEVAR include infection, connective tissue disorders, shaggy aortas, extreme aortic angulations, very diseased or stenotic visceral vessels or early divisions of visceral vessels not allowing delivery of a bridging stent.

The technique involves deployment of a main aortic stent graft body with fenestrations and or branches.[935] Fenestrations are preferable in cases where the aortic wall will be close to the endograft, e.g., in short neck AAAs and juxta- and pararenal AAAs. Branches are preferable when the aortic wall will be further from the endograft which typically occurs in some type IV TAAA. Scallops are sometimes included to increase the total seal of the repair without increasing its complexity (Fig. 9 — f/EVAR, bEVAR, f/bEVAR).

A recent meta-analysis, including 1 804 complex AAA endovascular repairs from 14 studies, reported a pooled technical success of 96.0%, frequency of Type 1 and 3 endoleak 7.6% and 2.5%, respectively, temporary and permanent kidney injury 13.19% and 0.71%, and SCI 2.0%. The overall aneurysm related mortality was 0.6% and the pooled estimate for re-intervention rate was 15.7%.[951] Another meta-analysis comparing fEVAR and OSR of

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juxtarenal AAA, including 2 974 patients from 27 studies, found no significant difference in post-operative mortality (3.3% *vs*. 4.2%), while major post-operative complications were less common (23.1% *vs*. 43.5%) and re-interventions more frequent (11.1% *vs*. 2.0%) after fEVAR. Target vessel occlusion was reported in 2 – 4% after fEVAR.[952]

Comparing standard fEVAR (stenting of renal arteries with or without a scallop for SMA) and complex fEVAR (stenting of renal arteries as well as SMA and or coeliac trunk) could not demonstrate any significant major difference in technical success rate, mortality or durability.[952–954] There are, however, also reports that more complex fEVAR increase complication rates (4% *vs*. 18%) compared with standard fEVAR.[955] Nevertheless, a liberal use of complex fEVAR is justified whenever needed to obtain an adequate proximal sealing zone for a durable repair. A minimum of 20 mm seal in a healthy and parallel walled aorta has been suggested,[953] while an excessively long sealing zone results in more extensive coverage of the aorta with the increased risk of SCI. Branched devices either with inner or outer branches involve more extended aortic coverage compared with fenestrated devices and therefore, should be reserved for type IV TAAAs.[956,957]

Recommendation 117		New
For patients undergoing endovascular repair of complex abdominal aortic aneurysms, consideration should be given to limiting the aortic coverage to reduce the risk of spinal cord ischaemia, however without compromising the proximal sealing zone.		
Class	**Level**	**References** ToE
IIa	C	Mastracci *et al.* (2015),[956] Bertoglio *et al.* (2018),[957] Spath *et al.* (2023)[958]

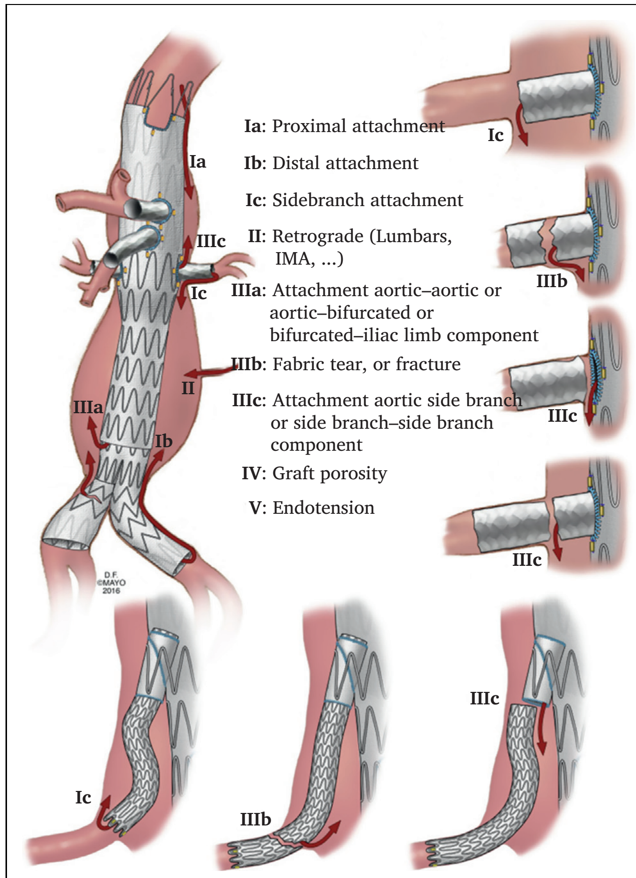

Figure 10. Endoleaks associated with failed bridging stents in the target vessels of fenestrated and branched endovascular aortic repair. IMA = inferior mesenteric artery. Permission to reproduce granted from Elsevier *J Vasc Surg.*[935]

Available data suggest that f/bEVAR for complex AAA has acceptable durability and complication rates[959–961] but re-interventions are needed between 24% and 39%.[960,961] The five year results of the USA Zenith Trial, including 67 patients treated with fEVAR for juxtarenal AAA, reported a 30 day mortality rate of 1.5% with a five year freedom from aneurysm related mortality of 97% and freedom from secondary intervention of 64%. There were no aneurysm ruptures or conversions to open surgery.[959] In a recent multicentre study from the USA Fenestrated and Branched Aortic Research Consortium, including 681 patients who had undergone f/bEVAR for complex AAA, secondary interventions were frequently indicated (18% at one year and 41% at five years of follow up), mostly done percutaneously (84%), and consisting of minor (70%) and low magnitude (according to the physiological effects) procedures (81%).[962] These data highlight the importance of close, lifelong surveillance and suggest that secondary intervention should be anticipated and if adequately addressed will not negatively affect survival.[962]

Covered bridging stents connect the f/bEVAR with the target renal and or visceral arteries. Endoleaks specifically associated with failed bridging stents include Type 1c endoleak (leakage through the distal attachment of the bridging stent in the target vessel), Type 3c endoleak (leakage through the proximal attachment of the bridging stent in the f/bEVAR main body, or between two bridging stent components), and Type 3d (leakage through a graft tear, perforation, or fracture in the bridging stent graft) (Fig. 10).[959] Target vessel instability is defined by a composite of any stent stenosis, separation, or Type 1c or Type 3c endoleak requiring re-intervention and stent occlusion, aneurysm rupture, or death due to target artery complication(s).[963]

Currently, dedicated bridging stents are lacking. A systematic review found renal target vessel more prone to complications than visceral arteries in f/bEVAR (6% *vs*. 2%) with a similar re-intervention rate between standard balloon expandable covered stents and self expandable covered stents.[964] For fenestrations, balloon expandable covered stents have been widely used due to their high radial forces.[965,966] Current data from retrospective studies suggest that target vessel instability and re-intervention rates are favourable for self expandable covered stents as bridging stent grafts in branches.[852] A range of different balloon expandable and self expandable covered stents are available on the market, with different properties and configurations. Due to the lack of comparative performance data it is not possible to recommend one covered stent over another, but selection should be decided on a case by case basis depending on anatomy and local experience. Following reports of failed bridging stent brands the availability of documented long term performance data in the f/bEVAR setting is another aspect to consider.[967] The technical success of f/bEVAR relies on accurate intra-operative imaging. Traditionally, DSA has been used to ensure correct stent graft deployment, assess the patency of side branches, and detect the presence of endoleaks.

Image fusion of CTA images with fluoroscopy can be achieved with automatic registration of the pre-operative CTA with an intra-operative non-contrast cone beam CT or with a two dimensional—3D technique after acquiring two fluoroscopic images at least 30° apart. Fusion imaging has been demonstrated to provide additional real time 3D guidance with reduced radiation, procedure time, and iodinated contrast doses during complex endovascular repairs.[365,968—973] In a recent meta-analysis, use of image fusion was associated with a significant reduced contrast volume (—79 mL), fluoroscopy time (—14 minutes), and procedure time (—52 minutes) in complex EVAR.[974] Image fusion should therefore be considered during complex EVAR procedures, which is also in agreement with the ESVS 2023 Radiation protection guidelines.[363]

The use of completion on table cone beam CT has been advocated to assure the quality of complex endovascular procedures.[975,976] The C arm, which includes both the Xray source and detectors, rotates around the patient during the acquisition of images, thus creating a 3D set of images similar to CT. The use of cone beam CT combined with a completion angiogram has been shown to be highly accurate in detecting complications intra-operatively post-EVAR.[923] In a single centre prospective study, including 154 patients undergoing complex EVAR, cone beam CT detected positive findings in 43 patients (28%); stent compression or kink in 17%, Type I or Type III endoleak in 10% and arterial dissection or thrombus in 5%. Of these, 27 patients (18%) had positive findings that prompted an intra-operative (17%) or delayed intervention (1%). DSA alone would not have detected positive findings in 34 of 43 patients (79%), including 21 patients (49%) who needed secondary interventions.[976] The technique however potentially exposes the patient to a higher effective dose of radiation than a single view completion DSA,[977] unless it can replace DSA and the subsequent post-operative CTA.[978] Furthermore, the feasibility of cone beam CT has not been evaluated for all the different available imaging systems. Thus, cone beam CT may be valuable adjunct to standard DSA for completion control after f/bEVAR, however, whether it can work as a standalone quality control technique in this setting remains too early to say.

Contemporary data suggest that use of intravascular ultrasound (IVUS) reduces fluoroscopy time, radiation, and contrast dose without compromising the technical success of the endovascular repair in the short term.[973,979,980] The use of IVUS to quality assure f/bEVAR deserves further investigation with reference to both efficacy and cost effectiveness.

Recommendation 118		New
During endovascular aortic repair of complex abdominal aortic aneurysms, the use of intra-operative image fusion should be considered, to reduce radiation exposure, contrast volume, and operating time.		
Class	**Level**	**References** **ToE**
IIa	**B**	de Ruiter *et al.* (2016),[972] Doelare *et al.* (2021)[974]

Recommendation 119		New
During endovascular repair of complex abdominal aortic aneurysms, the use of on table cone beam computed tomography imaging for completion control may be considered.		
Class	**Level**	**References** **ToE**
IIb	**C**	Tenorio *et al.* (2020)[976]

8.2.3. Open surgical repair *vs.* fenestrated and branched endovascular aortic repair. There are no direct comparisons between the outcomes of OSR and f/bEVAR, and available data are limited by selection and publication bias. Furthermore, the lack of independent long term follow up data makes it difficult to evaluate the durability of both techniques.

In a meta-analysis of fEVAR *vs.* OSR for complex AAA, data on more than 7 000 patients from 11 studies published between 2014 and 2019 were used in a propensity score matched analysis. The odds of peri-operative death after f/bEVAR were lower, although not significantly, than after OSR (OR 0.56, 95% CI 0.28 — 1.12), whereas the hazard of overall death during follow up was higher following f/bEVAR, but again, without reaching statistical significance (HR 1.25, 95% CI 0.93 — 1.67). The hazard of re-intervention was significantly higher after endovascular therapy (HR 2.11, 95% CI 1.39 — 3.18). The certainty for the body of evidence for peri-operative and overall mortality rates during follow up was judged to be very low and moderate, respectively, and for re-intervention it was judged to be high.[981]

In a recent network meta-analysis, including 7 854 patients from 23 observational studies who underwent repair for short neck AAA and juxtarenal AAA, the peri-operative mortality was significantly lower after fEVAR (RR 0.62) compared with OSR. This difference was not seen at midterm follow up (30 months). Compared with OSR, fEVAR was associated with a lower peri-operative MI rate (RR 0.37) but a higher midterm re-intervention rate (HR 1.65). All studies had a moderate or high risk of bias and confidence in the network findings (GRADE) was generally low, highlighting the need for better quality data.[948]

Another network meta-analysis evaluating OSR *vs.* f/bEVAR *vs.* chEVAR in juxta- and pararenal AAA, included a total of 4 369 patients from 16 observational studies, of which 10 was deemed as having serious or critical risk of bias, and six as having moderate risk of bias. The GRADE quality of evidence ranged from moderate to very low quality. A non-significant trend of a higher midterm (range 6 — 60 months) mortality rate was seen after f/bEVAR than

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OSR. A similar non-significant trend towards higher rates of aortic related re-intervention and side branch occlusion or stenosis was seen for both f/bEVAR and chEVAR compared with OSR. When comparing endovascular techniques, no significant preferences for either FEVAR or chEVAR were found.[982]

Most recently, the results from the UK COMPASS, a large cohort study using England wide registry data, included 999 patients undergoing elective repair for juxtarenal AAA (defined as neck length < 10 mm), were presented.[983] Subgroup analysis was stratified by neck length (0 − 4 mm $n = 568$ and 5 − 9 mm $n = 275$) and British Aneurysm Risk score (standard risk *vs.* high risk).[983] Patients treated with standard EVAR +/− adjuncts are not discussed here. Not surprisingly, peri-operative mortality was highest for high risk patients, 10.9% after OSR with neck length < 5 mm (*vs.* 1.7% after fEVAR) and 11.1% after OSR with neck length 5 − 9 mm (*vs.* no death after fEVAR). For standard risk patients, OSR mortality was 7.4% for those with neck length < 5 mm and 1.9% for neck length 5 − 9 mm *vs*. 2.3% and 0% after fEVAR. In a logistic regression model, peri-operative mortality was significantly lower after fEVAR than OSR (OR 0.25, $p < .001$). After a median of three years follow up overall mortality was significantly higher after fEVAR (*vs.* OSR) in standard risk patients with neck length 5 − 9 mm (21.2% *vs.* 7.5%), while the numerically worse overall long term survival in high risk patients or in patients with neck length < 5 mm treated by fEVAR (*vs.* OSR) did not reach statistical significance. There was no difference in late aneurysm related deaths between the techniques, regardless of risk score or neck length (HR 1.0, $p = .94$). The re-intervention rate at three years was significantly higher after fEVAR (27.7%) than OSR (17.8%). Of note, this is preliminary (unpublished) data presented at the Annual Scientific Meeting of the Vascular Society of Great Britain & Ireland in November 2022, and updated with final results in May 2023 after individual patient data auditing; definite conclusions cannot be drawn until the study has been peer reviewed.[984] Nevertheless, in the absence of RCTs this large contemporary nationwide study is a landmark study of value to discuss in this context. Whether the observed poor long term survival after fEVAR is due to the propensity score matching not being able to fully compensate for biases in clinical practice such as offering OSR to healthier patients who also have better longevity, and endovascular strategies to less healthy patients who do not have similar life expectancy, needs clarification.

A recent study evaluating change in health related QoL found a significant but transient decline in physical component scores after f/bEVAR for pararenal AAA, similar to patients treated with standard EVAR for AAA. Patients treated for TAAA (50% type IV TAAA) had lower QoL scores at baseline and did not show the same recovery after the initial post-operative decline.[985] There are no data on QoL after OSR for complex AAA.

In a cost effectiveness analysis published to date on data from the WINDOWS registry, costs were €38 212 for f/bEVAR compared with €16 497 for OSR. After two years of follow up from the same study there were no differences in mortality rate between the endovascular and OSR groups (11.2% *vs*. 11.4%). The total hospital costs were €41 786 for f/bEVAR *vs*. €21 142 for OSR.[986] In a cost effectiveness analysis commissioned by the National Health Service in the UK no evidence for the superiority of OSR or endovascular repair for juxtarenal AAA or TAAA could be established. As it was difficult to estimate costs because of the rapidly evolving endovascular technology, a cost effectiveness analysis was not deemed possible. They proposed a RCT to estimate the effect of f/bEVAR compared with OSR or conservative management.[987] It is, however, increasingly difficult to extrapolate conclusions about cost analyses across multiple healthcare systems in different countries, and a variety of national health system specific socio-economic cost and value considerations need to be taken into account.

In conclusion, due to the lack of high quality evidence and the complexity and variety of complex AAAs, decision making is complex and should be tailored to each individual patient and local health economies. Stratification of cases by anatomy and surgical risk may be useful in patients with a complex AAA. OSR with an anastomosis below the renal arteries with a short renal clamping time may be a preferable and a more durable option in fit patients with a short aortic neck. With more complex anatomy or high surgical risk because of comorbidities, an endovascular solution may be preferable.

Given the rarity and complexity of complex AAA treatment centralisation to specialised high volume centres that can offer both open and endovascular repair seems justified (see Chapter 2).

Recommendation 120			Changed
For patients with a complex abdominal aortic aneurysm and standard surgical risk, open or endovascular repair should be considered based on patient fitness, anatomy, and patient preference.			
Class	Level	References	ToE
IIa	C	Patel *et al.* (2022),[948] Antoniou *et al.* 92021),[981] Patel *et al.* (2021),[983] Doonan *et al.* (2019)[988]	

Recommendation 121			Changed
For patients with a complex abdominal aortic aneurysm and high surgical risk, endovascular repair with fenestrated and branched technologies should be considered as first line therapy.			
Class	Level	References	ToE
IIa	C	Patel *et al.* (2022),[948] Jones *et al.* (2019),[952] Antoniou *et al.* (2021),[981] Patel *et al.* (2021),[983] Doonan *et al.* (2019),[988] Caradu *et al.* (2018),[989]	

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8.2.4. Parallel grafts. Parallel grafts refer to an alternative technique to extend the (infrarenal) aortic neck by means of placing stent grafts in a chEVAR or snorkel or periscope configuration parallel to the main aortic graft. This technique has the advantage that it does not use CMDs that may take time to be manufactured, whereas a disadvantage might be the formation of gutters and potential subsequent Type 1a endoleaks, and graft occlusion.[945,990,991] The interpretation of the published research is hampered by the high risk of bias in patient selection and case mix, definition and ascertainment of patency, completeness of follow up, and scarce long term outcome data.

In a report from the PERICLES (performance of the chimney technique for the treatment of complex aortic pathologies registry) registry, in which 95% of the 517 patients had a juxtarenal AAA, the reported 30 day mortality rate for elective cases was 3.7% and 2.9% had a persistent endoleak. Chimney graft patency in patients who had imaging after a mean of 17 months follow up was 94% and was estimated to be 89% and 87% after two and three years, respectively.[992] In a later follow up analysis of 244 patients, the primary patency for chimneys was 94%, 93%, 92%, and 90% after 2.5, three, four, and five years of follow up, respectively.[993] Other studies have shown less favourable outcomes, with troubling rates of Type 1a gutter related endoleaks and target vessel occlusion.[990,991] In a systematic literature review of juxtarenal AAA repair the incidence of post-operative Type 1a endoleak was 7.6% after chEVAR compared with 3.7% after fEVAR.[994]

The best results with parallel grafts have been obtained in properly selected patients with a proximal landing zone of $\geq$ 15 mm, proper stent graft oversizing of 30%, and when the use of chimneys was restricted to a maximum of two. The HR of chimney graft occlusion has been described to increase by 1.8 for each additional chimney graft.[995,996] A nitinol polyester EVAR device with balloon expandable covered chimney stents is reported to be the preferred combination for chEVAR.[993,997]

Because of its uncertain effectiveness the parallel graft technique is not recommended in the elective setting, but should be reserved for urgent or bailout settings, and should not include more than two chimneys.

Recommendation 122			Changed
Endovascular repair for a complex abdominal aortic aneurysm using parallel graft techniques should only be considered as an option in the emergency setting, or as a bailout, and ideally be restricted to ≤ 2 chimneys.			
Class	**Level**	**References**	**ToE**
IIa	C	Donas *et al.* (2015),[992] Taneva *et al.* (2021),[993] Scali *et al.* (2018)[996]	

8.2.5. Novel and adjunctive endovascular techniques. Following the collapse of the EVAS concept and subsequent withdrawal of the device,[7] its use in complex AAA repair is no longer relevant. Other, novel therapeutic tools that could potentially expand the endovascular options in complex AAA repair include endostaples (also called endoanchors or endosutures) and *in situ* laser fenestration.

The Heli-FX EndoAnchors (Medtronic Vascular, Minneapolis, Minnesota, USA) are intended to provide fixation and sealing between the endovascular aortic graft and the native artery, and have been used in conjunction with standard EVAR devices for treating short neck AAAs. Among 100 patients with a hostile neck (length < 10 mm, diameter > 28 mm, angulation > 60°, conical configuration, or significant mural thrombus of calcium) freedom from Type 1a endoleak was 95% in primary treated patients and 77% in secondary treated patients after mean 13 months follow up.[998] In another cohort of 70 patients with an infrarenal neck length 4 − 10 mm, treated with the Endurant II or IIs endograft and Heli-FX EndoAnchors, four had a transient Type 1a endoleak and none experienced main body stent migration, aneurysm sac growth, or aneurysm rupture or requiring conversion to OSR through 12 months follow up.[999] In a meta-analysis, including 968 patients from eight studies with and without hostile neck, 6% developed a persistent Type 1a endoleak, 0.3% required an additional proximal aortic cuff due to migration of the main graft, and expansion of the aneurysm sac was found in 1.93% after mean six months follow up.[1000]

The literature on endostaplers is mainly limited to company sponsored reports and long term data on their effectiveness (and safety) is missing. Until further data on durability are available elective use of standard EVAR with endostaples to treat short neck AAAs should be limited to clinical trials (e.g., SOCRATES) approved by research ethics committees after obtaining patient's informed consent.[428]

Laser generated or needle assisted *in situ* fenestration of standard stent grafts is an off label technique mainly aimed at emergency treatment. The technology remains in its infancy, with only limited clinical data from technical and case reports.[1001−1003] Retrospective single centre studies report acceptable target vessel ischaemia time, bridging stent graft patency[1002] and a favourable outcome[1003] in the acute setting. A recent single centre study, including 44 patients treated for aortic pathologies involving the visceral segment, with 108 *in situ* laser fenestrations, reported a low 30 day mortality rate of 4.5% and favourable midterm outcome; with a Kaplan−Meier estimated two year survival of 73%, aortic related re-intervention free survival of 70%, and stent related re-intervention free survival of 91%.[1004] Long term data remain scarce and the technique is currently not recommended in the elective setting outside of investigational studies.[1005,1006]

Stent grafts deviating from the traditional concept for adequate sealing (> 15 mm) in the proximal neck with a self expandable stent graft such as the Ovation Alto Stent graft (Endologix Inc. Irvine, CA, USA) which uses a polymer based seal, claims in its IFU eligibility for proximal landing zones of > 7 mm, thus in practice juxtarenal AAA.[1007] The long term effectiveness and safety of the Ovation Alto device in short necks (< 15 mm) has, however, not yet been proven and therefore, the device is not recommended for use outside ethically approved clinical trials with patients' informed consent.[1008]

Recommendation 123			Unchanged
For patients with a complex abdominal aortic aneurysm, use of new techniques and concepts is not recommended in routine clinical practice and should be limited to studies approved by research ethics committees, until adequately evaluated.			
Class	Level	References	ToE
III	C	Qamhawi *et al.* (2020),[811] Prendes *et al.* (2022),[1006] Krievins *et al.* (2018),[1009] Barleben *et al.* (2020),[1010] Mathlouthi *et al.* (2022)[1011]	

8.2.6. Hybrid repair. The combination of visceral and renal artery rerouting (bypassing) associated with the endovascular exclusion of an aortic aneurysm with a standard stent graft is another treatment option known as hybrid repair. Data regarding hybrid repair of complex AAAs are scarce in the recent literature, however, some considerations could be extrapolated from experience in TAAA repairs with this approach.[1012] This technique initially challenged standard OSR as a less invasive treatment option. However, the presumed less invasive nature of hybrid TAAA repair does not seem to result in lower complication rates at early and midterm follow up.[1013—1015] In general, the hybrid approach is hampered by both the early disadvantages of open surgery, and the late ones of the endovascular approach, with the avoidance of aortic cross clamping as the sole advantage.[1013]

With the established role of conventional OSR, and the development of endovascular approaches such as fenestrated and branched stent grafts, the actual role of the hybrid repair in the treatment of complex AAAs is limited. However, the method of surgical bypass from the iliac artery to one or more visceral arteries can be used as a bailout for failure of endovascular bridging stents during or after f/bEVAR.

Recommendation 124			New
Hybrid repair, by means of visceral and renal artery rerouting (bypassing) combined with endovascular exclusion of the aneurysm, is not recommended as the first line treatment for complex abdominal aortic aneurysm.			
Class	Level	References	ToE
III	C	Moulakakis *et al.* (2012),[1013] Tshomba *et al.* (2012),[1014] Rosset *et al.* (2014)[1015]	

8.3. Preservation of renal function during complex abdominal aortic aneurysm repair

Since the treatment of complex AAA may involve the renal arteries and, as patients often have renal dysfunction, measures for preservation of renal function are of great importance.[1016] Several adjunctive methods have been reported, such as reducing suprarenal clamp time and renal perfusion during open surgery, and different pharmacological strategies. While data regarding specific protocols for complex AAAs are scarce, relevant information can also be extrapolated from the literature on renal protection during TAAA open surgery.[1017]

A suprarenal clamp time $>$ 25 minutes is reported to be associated with ischaemic damage to the kidney. Thus, an expeditious proximal anastomosis is advocated to restore direct renal perfusion as soon as possible during OSR for complex AAA, which is surely the most effective method of reducing acute kidney injury.[1018]

In patients undergoing OSR of a complex AAA, selective renal perfusion during extended suprarenal clamp time ($>$ 25 minutes) may prevent cellular necrosis and ischaemia reperfusion injury, and can be obtained with Pruitt occlusion perfusion catheters of adequate size (5 — 9 Fr). Several strategies for selective renal artery perfusion have been suggested. In a RCT from the TAAA field, patients who had renal perfusion with 4°C Ringer's lactate developed renal dysfunction significantly less often than those who had continuous perfusion with blood (21% *vs.* 63%).[1019] In another RCT 21% of patients having open TAAA repair who had renal perfusion with 4°C Ringer's lactate had renal dysfunction as opposed to 31% of those with perfusion with 4°C cold blood.[1020] A recent meta-analysis showed significantly reduced post-operative acute kidney injury with the use of (any) intra-operative cold renal perfusion during open complex aortic aneurysm repair (OR 0.46).[1021] Renal perfusion with warm blood requires a complex setting with extracorporeal blood circuits and offers only limited renal protection.[1022] In a RCT, including 90 patients undergoing elective open TAAA repair, comparing renal perfusion with 4°C crystalloid solution enriched with histidine-tryptophan-ketoglutarate (Custodiol, Dr Franz-Kohler Chaemie GmbH, Bensheim, Germany) with standard 4°C lactated Ringer's solution, the incidence of post-operative acute kidney injury was significantly lower in the Custodiol group (48.9% *vs.* 75.6%).[1023] Single centre reports have confirmed the benefits of renal hypothermia during the ischaemic period of both elective and ruptured juxtarenal AAA OSR.[1024,1025]

There are only limited data from underpowered studies on pharmacological protection of renal function. One RCT comparing mannitol *vs*. saline infusion before aortic cross clamping in 28 patients with an infrarenal AAA did not find a clinically relevant effect of mannitol on preservation of renal function.[1026] In another RCT comprising 60 patients having open infrarenal AAA repair, no difference was found in renal failure in patients allocated to fenoldopam *vs*. dopamine and sodium nitroprusside.[1027]

In conclusion, there is no compelling evidence for pharmacological protection of renal function during OSR of complex AAAs, whereas cold renal perfusion may be beneficial. As for infrarenal AAA repair, small accessory (polar) arteries (< 4 mm) supplying only a small part of the kidney can be ligated, while larger arteries are treated in the same fashion as renal arteries with selective perfusion and reattachment. And, if the left renal vein is divided for exposure, reconstructing the vein may be considered, especially if collaterals were divided and for cases of concomitant renal ischaemia (see Recommendation 51).[392,393,1018,1028,1029]

Recommendation 125			Changed
For patients undergoing open repair of a complex abdominal aortic aneurysm with a suprarenal clamp time > 25 minutes, cold renal perfusion should be considered.			
Class	Level	References	ToE
IIa	C	Jongkind *et al.* (2010),[944] Dubois *et al.* (2013),[1018] Köksoy *et al.* (2002),[1019] Lemaire *et al.* (2009),[1020] Yeung *et al.* (2008),[1024] Yeung *et al.* (2010)[1025]	

In patients undergoing complex endovascular AAA repair, strategies to reduce the risk of contrast induced nephropathy (CIN) should be implemented. In addition to dose reduction of iodine contrast media, withdrawal of nephrotoxic drugs (such as certain antibiotics, renin angiotensin aldosterone system inhibitors, and non-steroidal anti-inflammatory drugs) and ensuring adequate hydration may also lower the risk of CIN.[1030] Intravenous hydration with 0.9% saline is the prophylactic intervention best supported by evidence, to decrease the risk of CIN.[1031,1032] Several other prophylactic regimens to lower the risk of CIN have been proposed, for example acetylcysteine and hydration with sodium bicarbonate instead of saline, but none has been convincingly proven to be effective.[1033,1034] A large RCT found no benefit of intravenous sodium bicarbonate over intravenous sodium chloride or of oral acetylcysteine over placebo to prevent of contrast associated acute kidney injury.[1035]

In the treatment of complex AAA, preservation of large accessory renal arteries (≥ 4 mm) is feasible with low complication rates and good patency. It prevents early renal dysfunction and provides higher freedom for midterm renal dysfunction,[1036] although so far there is no demonstrated effect on death in early post-operative and follow up period.[448] Incorporation of < 4.0 mm renal arteries during f/bEVAR is associated with lower technical success, higher risk of arterial disruption and kidney loss, and lower patency rates at one year,[1037] and should be avoided.

Recommendation 126		New
For patients undergoing endovascular repair of a complex abdominal aortic aneurysm a strategy to preserve renal function by dose reduction of iodine contrast media, withdrawal of nephrotoxic drugs and ensuring adequate hydration should be considered.		
Class	Level	References
IIa	C	Consensus

Recommendation 127			New
For endovascular repair of a complex abdominal aortic aneurysm, preservation of large accessory renal arteries (≥ 4 mm) should be considered.			
Class	Level	References	ToE
IIa	C	Spanos *et al.* (2021),[448] Torrealba *et al.* (2022)[1036]	

8.4. Spinal cord ischaemia prevention in complex abdominal aortic aneurysm repair

Impairments in spinal cord perfusion are more frequently observed following open or endovascular repair of type I, II, and III TAAA, and specific considerations and recommendations in this field are reported in the recently published ESVS Clinical Practice Guidelines on thoracic and thoraco-abdominal aorta.

The occurrence of SCI after OSR of juxta- and pararenal AAAs is anecdotal, and rare after open repair of type IV TAAA, but should be considered as a potential complication.[941]

Endovascular repair of complex AAAs usually requires a supravisceral proximal sealing zone, and thereby a higher number of intercostal arteries are sacrificed compared with OSR. In a systematic review and meta-analysis, including 5 121 patients from 14 studies undergoing juxtarenal AAA repair, endovascular (*vs.* open) repair was associated with a significantly lower 30 day mortality (OR 0.50), acute renal failure (OR 0.50), bowel ischaemia (OR 0.50), and length of stay (−6 days) but with increased risk of SCI (OR 3.13).[988] However, a more recent multicentre study reported the absence of SCI after endovascular juxta- and pararenal AAA repair[1038] while another systematic review and meta-analysis found a low incidence of SCI (3%) after type IV TAAA endovascular repair.[1039]

Strategies for prevention, early detection, and treatment of SCI to be implemented include (1) staging the procedure, (2) maintaining a high BP (MAP > 80 mmHg) and oxygenation (haemoglobin level > 10 mg/dL), (3) preservation of collaterals, (4) cerebrospinal fluid drainage (CSFD) and (5) neuromonitoring.[1040,1041]

Prophylactic CSFD has been shown in RCTs to prevent SCI in open TAAA repair.[1042] However, there is a lack of evidence for its role in EVAR of complex AAA. The potential benefits of CSFD must be weighed against the risks. In a recent single centre study, including 448 complex AAA endovascular repairs of which 147 had prophylactic spinal fluid drainage, 12% developed drain related complication, whereof 2% were disabling.[1043]

In summary, SCI is infrequent after complex AAA repair. Therefore, routine use of prophylactic cerebrospinal fluid drainage during complex AAA repair is not indicated. It may, however, be considered in high risk (for SCI) patients, such as during endovascular repair of type IV TAAA with extensive aortic coverage or in patients with previous aortic surgery or with occluded hypogastric arteries. The most vulnerable period to develop SCI is immediately post-operatively. Rapid extubation to check the neurological state of patient is desirable. A policy of rescue drainage with urgent post-operative drain placement at the onset of symptoms (*vs.* prophylactic drainage) appears equally effective[1039,1044,1045] and is usually preferred today.

Recommendation 128		New
For patients undergoing open or endovascular repair of a complex abdominal aortic aneurysm, a policy of reactive (rescue) cerebrospinal fluid drainage may be considered preferable over routine prophylactic cerebrospinal fluid drainage.		
Class	Level	References
IIb	C	Consensus

8.5. Ruptured complex abdominal aortic aneurysm

Mortality after OSR for emergency complex AAA is high. In a recent multicentre study, including 374 patients who underwent an emergency complex open AAA repair, the overall 30 day mortality rate was 32%, and approaching 50% for type IV TAAAs.[1046]

A major limitation with fenestrated and branched CMDs is the time consuming manufacturing process.[75] Alternative on label endovascular options in the acute setting include off the shelf devices, such as PMEG, *in situ* fenestration and parallel graft techniques. Current evidence on emergency endovascular treatment of complex AAAs is mainly derived from small retrospective single centre studies, reporting high technical success and good midterm survival and durability.[950,1047–1053]

Comparative data between open and endovascular repair of ruptured complex AAAs are scarce. In a report from the American College of Surgeons NSQIP, including 338 patients with complex AAAs treated by OSR and 105 treated endovascularly, the 30 day mortality rate was 32.5% *vs.* 23.8% respectively. After propensity score weighting, the open cohort had 1.75 times the odds of death compared with the EVAR cohort (OR 1.8, $p = .06$). OSR was also associated with greater odds of pulmonary complications, colonic ischaemia, and longer ICU stays in survivors.[1054]

Considering the desperate situation and complexity of a ruptured complex AAA with lacking evidence, an individualised approach is advised in choosing the surgical treatment modality, taking the patient's fitness, anatomy, and patient preferences into account. Although the use of endostaples or *in situ* laser fenestration are not preferable in elective situations, the crisis situation of a ruptured complex AAA justifies a more liberal use of unproven technologies.

Recommendation 129			Changed
For patients with a ruptured complex abdominal aortic aneurysm (or who are deemed urgent for any other reason), open surgical repair or endovascular repair (with off the shelf branched stent graft, physician modified endograft, *in situ* fenestrations, or parallel grafts) should be considered based on patient status, anatomy, and patient preferences.			
Class	Level	References	ToE
IIa	C	Mayer *et al.* (2012),[535] Gouveia *et al.* (2022),[950] Taneva *et al.* (2021),[993] Konstantinou *et al.* (2020),[1049] Kolbel *et al.* (2021),[1053] Latz *et al.* (2020)[1054]	

8.6. Follow up after complex abdominal aortic aneurysm repair

There is no solid evidence on best practice for surveillance after complex AAA repair. However, as endovascular repair of complex AAAs is an evolving technique, robust surveillance is imperative. The major components of a post-f/bEVAR imaging examination include measurement of aortic aneurysm sac size, assessment for endoleak, and evaluation of target vessel patency and integrity. CTA is the primary imaging modality for follow up after f/bEVAR, and all patients should be included in a thorough follow up programme including at least a 30 day and one year post-operative CTA, and thereafter on an individualised basis.

Reports suggest that DUS and CEUS can be reliable alternatives to CTA for fEVAR surveillance.[1055–1057] Thus, in selected patients, DUS may replace CTA during continued follow up. DUS protocols for follow up after f/bEVAR can be based on those that have been established for standard EVAR, along with assessment of fenestrations and branches, as well as patency of the renal and mesenteric arteries.[1057]

Data on post-operative antithrombotic regimens after endovascular complex AAA repair are scarce. Although all patients with AAA should receive antiplatelet therapy, several large studies on complex endovascular repair did not specify their post-operative antithrombotic regimen,[953,992,1058,1059] whereas others used aspirin[1060] or dual antiplatelet therapy.[1061] A recent Delphi expert consensus report suggested prescription of dual antiplatelet therapy for up to six months following f/bEVAR to improve bridging stent patency.[1062] Dual antiplatelet treatment is, however, associated with an increased risk of bleeding, and the risk benefit ratio in the post-f/bEVAR setting needs to be investigated further before firmer recommendations can be formulated.

For target vessel occlusion after complex AAA repair, immediate catheter based revascularisation should be considered. If indicated, surgical revascularisation with bypass is a secondary option. There are no reliable data for the upper limit of warm ischaemia time for a kidney to be salvageable. Usually, however, a kidney is considered to be permanently damaged after 6 – 12 hours of warm ischaemia. In case of

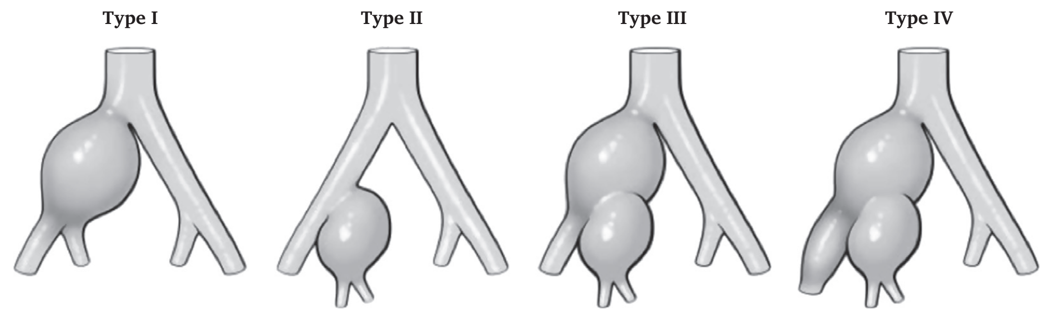

Figure 11. Isolated iliac artery aneurysm classification by Reber *et al.*[1069] Permission to reproduce granted from *Springer Nature.*

visible residual perfusion of the kidney on CTA or US, a delayed revascularisation attempt may be considered in selected cases. In a multicentre study, this approach had a technical success of 96%, with improvement of renal function observed in 40% of these patients after f/bEVAR.[1063]

Recommendation 130		**Changed**
After endovascular treatment for a complex abdominal aortic aneurysm, long term imaging surveillance is recommended; with computed tomography angiography within 30 days and one year and thereafter individualised.		
Class	**Level**	**References**
I	C	Consensus

Recommendation 131		**New**
After endovascular treatment for a complex abdominal aortic aneurysm, duplex ultrasound surveillance may be considered as an alternative to continued computed tomography angiography surveillance after the first post-operative year in selected patients.		
Class	**Level**	**References** ToE
IIb	C	Gargiulo *et al.* (2014),[1055] Perini *et al.* (2012),[1056] Heneghan *et al.* (2016)[1064]

Recommendation 132		**New**
Patients deemed at risk of bridging stent patency failure after endovascular treatment for complex abdominal aortic aneurysm may be considered for dual antiplatelet therapy in the early post-operative period.		
Class	**Level**	**References**
IIb	C	Consensus

Recommendation 133		**New**
Patients with target vessel obstruction after complex abdominal aortic aneurysm repair should be considered for prompt evaluation for possible revascularisation.		
Class	**Level**	**References**
IIa	C	Consensus

9. MANAGEMENT OF ILIAC ARTERY ANEURYSM

The most accepted definition of iliac artery aneurysm (IAA) is dilation of the vessel to more than 1.5 times its normal diameter.[82] In general, a common iliac artery (CIA) $\geq$ 18 mm in men and $\geq$ 15 mm in women, and an IIA $\geq$ 8 mm is considered aneurysmal.[82,1065] IAAs are commonly associated with aneurysmal dilation of the abdominal aorta as aorto-iliac aneurysms.[1066,1067] Isolated IAA is an aneurysm without an aneurysm of the infrarenal abdominal aorta. This definition includes aneurysms of the CIA, the IIA, the EIA, and combinations of those. Aneurysms of the EIA are rare.

Several classifications for isolated IAAs have been proposed.[1068–1070] Reber's anatomical classification into type I – IV appears well suited to compare outcomes of different anatomical entities (Fig. 11), while Fahrni's classification depends on neck suitability for endovascular repair, which may change with time, device, and operating technique.

The underlying pathology and type of isolated IAA is similar to AAA and includes degenerative aneurysm, pseudoaneurysm, penetrating ulcer, post-dissection aneurysm, mycotic aneurysm, and traumatic aneurysm.[1071]

Isolated IAAs are most frequently confined to the CIA (Reber I) and least frequent in the EIA[1066,1069,1072,1073] (Reber IV). Their overall frequency is reported in up to 7% of all aorto-iliac aneurysms and 12 – 48% of all isolated IAA are bilateral.[1066,1073,1074] The majority of patients with isolated IAA are male (90%) and diagnosed at the age of 70 years or older.[1072,1074,1075]

9.1. Surveillance of small iliac artery aneurysms and indications for repair

While most individuals with isolated IAA are asymptomatic, symptoms can result from local compression of the ureter, sacral plexus, or iliac vein.[1070,1076] Rupture has been reported in 4.3% (45/981)[826] and symptoms in 18% of isolated IAA.[1076] Physical examination may frequently overlook IAA, while US may identify 69% of cases,[1077] CTA is highly accurate in detecting IAA.[1070]

Data on the growth rate of IAA are scarce, with only retrospective studies, but it is thought to be similar to AAA, about 1 – 4 mm/year depending on aneurysm diameter.[1078–1080] Contemporary evidence from a large retrospective study has reported annual growth rates of 0.2 mm for isolated CIAAs 20 – 24.9 mm, 0.3 mm for CIAAs 25 – 29.9 mm, and 1.3 mm for CIAAs ≥ 30 mm.[1077] The incidence of rupture and its association with size and growth rate of the isolated IAA is not as well established as in AAA, with only case series available.

Most reported ruptured IAAs in the literature are larger than 50 mm, and rarely below 40 mm. A contemporary meta-analysis reported a weighted mean average diameter of 58 mm for ruptured IAA, with only two of 45 IAAs rupturing at < 40 mm diameter. A nationwide study from The Netherlands also reported very few IAAs rupturing below 40 mm (9/90) with a median diameter of 68 mm at the time of rupture.[826,1065,1066,1079,1081–1085]

Data on surveillance intervals for IAAs is limited, but recent recommendations suggest surveillance every three years for IAAs with diameter 20 – 25 mm, every two years for 25 – 29 mm and annually for ≥ 30 mm IAAs.[1077] Surveillance of a known IAA is preferably performed with US, with CTA reserved for those patients with larger aneurysms and or poor US visibility.

As solid data are lacking, the patients' operative risk as well as suitability for open and or endovascular repair should be considered, to determine the individual diameter threshold at which repair is considered. However, conservative treatment appears safe in most patients with a maximum diameter below 40 mm. A systematic review reported only two ruptures below the 40 mm threshold of 983 IAAs.[826,1081,1084] A retrospective multicentre study on the diameter of ruptured IIA aneurysms recommended surveillance of IIA aneurysms in elderly men until the diameter of 40 mm.[1065] This recommendation has been supported by a meta-analysis of IIA aneurysms.[1076]

Given the natural history of IAA with slow growth rates and the very low risk of rupture below 40 mm in diameter, the GWC considers it justified to raise the diameter threshold at which surgery should be considered to 40 mm.[826,1077] There are no data to suggest any gender differentiation of the indication for repair. Nevertheless, it may be reasonable to take gender and body size into account, in the same way as for AAA.

There are no available data on medical therapies in terms of BP control or treatment with platelet inhibitors, beta blockers or statins in patients with isolated IAA. Best medical treatment should therefore be according to recommendations for AAA (see Chapter 4).

Recommendation 134			New
For patients with an iliac artery aneurysm (common iliac artery, internal iliac artery, and external iliac artery, or combination thereof), imaging surveillance using ultrasound should be considered; every three years for aneurysms 20 – 24 mm in diameter, every two years for aneurysms 25 – 29 mm in diameter, and yearly for aneurysms ≥ 30 mm, taking into account life expectancy, suitability for future repair, concomitant aortic dilatation, and patient preferences.			
Class	Level	References	ToE
IIa	C	Steenberge *et al.* (2022)[1077]	

Recommendation 135			Changed
Patients with an iliac artery aneurysm (common iliac artery, internal iliac artery, and external iliac artery, or combination thereof) should be considered for elective repair at a diameter of ≥ 40 mm.			
Class	Level	References	ToE
IIa	C	Charisis *et al.* (2021),[826] Laine *et al.* (2017),[1065] Krupski *et al.* (1998),[1066] Chaer *et al.* (2008),[1072] Steenberge *et al.* (2022),[1077] Huang *et al.* (2008),[1079] Jalalzadeh *et al.* (2020),[1081] Fossaceca *et al.* (2015),[1083] Kasirajan *et al.* (1998),[1084] Kobe *et al.* (2018)[1085]	

9.2. Surgical treatment of iliac artery aneurysm

The aim of surgical treatment of IAAs is to exclude the aneurysm from the circulation to prevent further growth and rupture. Before the advent of endovascular repair in the early 1990s OSR was the mainstay of treatment of IAA. The steady shift towards endovascular techniques since 2000 has been associated with a significant decrease in operative morbidity and mortality,[1086] and a recent meta-analysis reported a peri-operative mortality rate of 0.7% for endovascular repair.[826] Furthermore, endovascular repair is associated with fewer complications and a shorter length of hospital stay.[1072,1073] While this trend was initially partly explained by differences in case mix, with a higher number of emergency cases in the OSR group, recent experience indicates significant advantages for endovascular repair in both elective and emergency settings.[1073,1086,1087] However, as pathology, anatomy, disease extent, and patient fitness differ widely between individual patients, both techniques should be available in centres managing patients with IAA.

IIA aneurysms due to their deep pelvic location are particularly challenging and contemporary meta-analytical data has reported operative mortality rates higher for OSR compared with endovascular techniques with 30 day mortality rates of 8.2% *vs.* 2.8%.[1076]

9.2.1. Open surgical repair. OSR is usually performed under general anaesthesia, using retroperitoneal or trans-abdominal access. Depending on the extent of the aneurysmal disease the reconstruction is done by iliac tube graft repair or by bifurcated graft repair including the infrarenal aorta, with or without revascularisation of the IIA. A less invasive technique in selected cases is ligation of the iliac artery with re-perfusion of the contralateral femoral artery and or IIA by a crossover bypass.[1088] The need for ligation of the IIA during OSR for IAA has been inconsistently reported.

Owing to the deep pelvic location, OSR of IAA can be technically challenging with an increased risk of iatrogenic injuries of veins, ureter, or nerve, resulting in peri-operative blood loss, morbidity including colonic ischaemia, and death.[1072]

9.2.2. Endovascular repair. Endovascular treatment of IAA originally involved embolisation of the IIA and stent graft coverage extending from the CIA to the EIA.[1089] Involving the infrarenal aorta and the contralateral iliac artery into the repair is often necessary to obtain a proper proximal seal.[1070,1072,1075] In contrast, OSR of isolated IAAs may be possible while leaving the infrarenal aorta and contralateral iliac arteries untouched.

Endovascular techniques have further evolved in recent years from routine embolisation of the IIA to side branch techniques preserving IIA patency.[1090] While the use of iliac branch devices (IBDs) to treat aorto-iliac aneurysms is well established, the use of the technique to treat IAAs has evolved with early mortality rates of just over 2% and encouragingly low rates of buttock claudication, erectile dysfunction, and bowel ischaemia[1091] if the anatomy is suitable. Furthermore Isolated IAAs treated with IBDs have demonstrated re-intervention and IBD occlusion rates of approximately 20% and 15% at five years of follow up.[1092] Results from aorto-iliac aneurysms indicate a high technical success rate and high midterm patency of the target vessel.[1093,1094] A contemporary meta-analysis of IBDs in aorto-iliac aneurysms reported 22% were used in isolated CIA aneurysms and 8% in isolated IIA aneurysms with high rates of technical success, low incidence of pelvic ischaemia and 0.4% 30 day mortality rate.[1095] The most common anatomical factor limiting the use of IBDs is an aneurysmal IIA.[1096]

Other, less well studied, alternative techniques of endovascular repair to preserve IIA perfusion in IAA have been proposed, including the bell bottom technique, the sandwich technique and hybrid repair including femoral crossover bypass;[1097] however, high quality studies on the management of an inadequate distal CIA landing zone are lacking.[1098]

Especially in ruptured isolated IAA the possibility to operate under local anaesthesia appears to be a significant advantage of endovascular repair. The necessity to convert to OSR is reported to be uncommon.[1083,1099]

In summary, the lack of comparative studies, and the different (mainly anatomical) prerequisites for open and endovascular repair, make them complementary in the treatment of IAA. Consequently, the decision about surgical technique for IAA repair should be based on individual considerations, such as anatomy, fitness, and the patient's wishes.

Recommendation 136		Changed
The choice of surgical technique for iliac artery aneurysm repair should be considered based on individual patient and lesion characteristics.		
Class	Level	References ToE
IIa	B	Buck *et al.* (2015),[1086] Yang *et al.* (2020),[1089] Illuminati *et al.* (2009),[1090] Giaquinta *et al.* (2018),[1091] Kouvelos *et al.* (2016)[1093]

9.2.3. Preservation of pelvic circulation. Interruption of IIA perfusion is normally well compensated for by collateral artery perfusion via pathways from the contralateral IIA, mesenteric and femoral arteries. If not, it may lead to symptoms including buttock claudication, colonic ischaemia, pelvic necrosis or erectile dysfunction.[1100] Buttock claudication is the most frequent complication of endovascular treatment of IAAs, with a reported frequency of up to 28%.[782,1072–1074,1093] Contemporary data from meta-analyses of isolated CIA aneurysms and IIA aneurysms reported similar rates of buttock claudication of 11.2% and 13.9%.[826,1076] Post-procedural sexual dysfunction, bowel ischaemia and SCI are rarely reported. The likelihood and severity of these complications are more frequent with bilateral IIA occlusion,[782,1093,1101] but cannot easily be predicted. Therefore, preservation of blood flow to at least one and ideally both IIAs is recommended if it does not compromise the primary treatment goal of aneurysm exclusion.

The availability of IBDs now allows preservation of IIA flow in most cases with suitable anatomy, leading to a reduced incidence of buttock claudication in the treatment of aorto-iliac AAAs and IAAs.[1093,1095,1102] Even in cases of IIA aneurysms without a proper landing zone within the main stem of the IIA, IBDs have successfully been used outside their IFU, landing distally in the gluteal arteries to preserve IIA flow to one of its major gluteal branches.[1103,1104] The superior gluteal artery can be used for distal stent graft sealing with early outcomes similar to IIA landing zones.[1105,1106]

Whenever embolisation of the IIA is necessary to exclude a CIA aneurysm, the embolising material should preferably be placed in the proximal portion of the IIA to maintain communication between its anterior and posterior divisions.[782,1100] Distal embolisation increases the risk of buttock claudication.[782,1100] In cases of bilateral IIA occlusion it has become common practice in many centres to stage the treatment to allow collateral development, although staging may increase the risk of aneurysm rupture.

In cases with extensive aortic coverage by stent grafts, with occlusion of segmental arteries, preservation of IIA flow plays an important role in prevention of SCI as this territory contributes to flow into the collateral network of the spinal cord.[1107,1108]

Recommendation 137		Unchanged
Preserving blood flow to at least one internal iliac artery during open surgical and endovascular repair of iliac artery aneurysms is recommended.		
Class	Level	References ToE
I	C	Bosanquet *et al.* (2017),[782] Jean-Baptiste *et al.* (2014)[1100]

Recommendation 138			Unchanged
For patients undergoing common iliac artery aneurysm repair in whom internal iliac artery embolisation or ligation is necessary, occlusion of the proximal main stem of the vessel is recommended if technically feasible, to preserve the distal collateral circulation to the pelvis.			
Class	Level	References	ToE
I	C	Jean-Baptiste *et al.* (2014),[1100] Bosanquet *et al.* (2017)[782]	

9.3. Follow up after iliac artery aneurysm repair

Data on follow up after endovascular IAA repair are scarce and when available the period of surveillance is short, with substantial numbers of patients lost to follow up. Surveillance is undertaken by DUS and CTA. Type 1 and Type 3 endoleaks have been reported and often lead to re-intervention. T2ELs are most common and probably under reported,[1076] but notably there were no reports of rupture related to untreated T2EL in a recent meta-analysis.[826] Secondary intervention rates were 17%[1085] but frequently not or under reported.[826] Evidence suggests that secondary intervention is more likely if stent graft coverage of the IIA origin is performed without concomitant embolisation.[1089] Furthermore, few have reported aneurysm related mortality, with the rate of 2.4% (7/288) likely to be an underestimate.[826]

Clearly the lack of robust follow up data for IAAs makes recommendations on follow up difficult. Longer term outcomes particularly for endovascular repair are needed. Until then, follow up should be in accordance with the recommendations for AAA (see Chapter 7).

10. MISCELLANEOUS AORTIC PROBLEMS

10.1. Mycotic abdominal aortic aneurysm

10.1.1. Definition and diagnosis of mycotic abdominal aortic aneurysm. Mycotic or infected AAAs are caused by septic emboli to the vasa vasorum, by haematogenous spread during bacteraemia or by direct extension of an adjacent infection leading to an infective degeneration of the arterial wall and aneurysm formation. The term mycotic aneurysm was coined by Osler in 1885, referring to the fungal like vegetation in endocarditis associated with infected aneurysms.[1109] Today, the term mycotic aneurysm is defined as all primary and secondary infective aneurysms, where bacteria are the most common causative pathogens. An alternative name for mycotic aneurysms, which has recently been proposed, is infective native aortic aneurysm.[1110]

In Europe and North America, staphylococci species, including both *Staphylococcus aureus* and *Coagulase negative* staphylococci, are the most common bacteria accounting for around 30 — 40% of mycotic AAAs. *Gram positive streptococci species* including *enterococci* and the *Gram-negative* Enterobacteriaceae species *(i.e., Escherichia coli*) and *Salmonella* species account for roughly 10 — 20% of MAAs each. In East Asia, however, *Salmonella* species are the dominant causative microbes, reported in up to 60 — 70% of mycotic AAAs. A culture negative rate is reported in the range of 20 — 30%.[1111]

The incidence of MAA is 0.5 — 1.53% of all aortic aneurysms in Western countries and reportedly higher in East Asia.[1112—1114] Most patients are male and tend to be younger (mean age 69 — 70 years) than those with a degenerative non-infected aneurysm (74 — 78 years).[89,1115,1116] Left untreated, beyond the septic complications, the natural outcome of a mycotic AAA is that of a rapid expansion, rupture, and death.

Diagnosis of a mycotic AAA is based on a combination of (1) clinical presentation, (2) laboratory tests and microbiology, and (3) radiological findings (Table 23). In addition, the presence of peri-aortic infection during surgery is diagnostic. A typical medical history is often seen, with the presence of concomitant infections (e.g., osteomyelitis, urinary, tuberculosis, gastroenteritis, and soft tissue) and immunosuppressive disease or medications (e.g., cancer, renal failure with dialysis, human immunodeficiency virus, diabetes, or steroid treatment).[1114—1121]

CTA represents the first line imaging technique,[1122] which can be supplemented with molecular imaging if necessary, e.g., 18-FDG PET or WBCS.[1123,1124]

A recent Delphi consensus statement proposed a diagnostic algorithm for mycotic AAA, based on a combination of the three criteria from Table 22: Definite diagnosis: 3/3 clinical criteria and no differential diagnosis being more probable, or 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 mycotic AAA; Probable diagnosis: 2/3 clinical criteria and no differential diagnosis being more probable; Not probable diagnosis: 1/3 clinical criteria.[1110]

Recommendation 139			Unchanged
The diagnosis of a mycotic abdominal aortic aneurysm is recommended to be based on a combination of clinical, laboratory, and imaging parameters.			
Class	Level	References	ToE
I	C	Sörelius *et al.* (2016),[1116] Jutidamrongphan *et al.* (2022)[1122] Sörelius *et al.* (2019)[1125]	

10.1.2. Management of mycotic abdominal aortic aneurysm. Early diagnosis, immediate administration of systemic antibiotics, and timely surgical treatment is crucial to improve early outcomes.

Empirical antibiotic treatment against *Staphylococcus aureus* and Gram negative rods, such as *Salmonella spp*. should be initiated as soon as cultures have been secured. As soon as possible targeted antibiotic therapy (depending on the microbiology) is started, alternatively continued empiric treatment in cases with negative blood and tissue cultures.

Due to the unpredictable and malignant natural course of mycotic AAA, with rapid expansion and high rupture risk (44% present with rupture,[1125,1126] prompt repair should be considered irrespectively of aneurysm size. The timing of

Table 23. Suggested diagnostic criteria for mycotic abdominal aortic aneurysms.	
Clinical presentation	Abdominal and or back pain, fever, sepsis or shock, concomitant infection
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 ribonucleic acid polymerase chain 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 presentation	Presence of peri-aortic infection

the surgery is debated. Reports of favourable outcomes in patients treated by delayed surgery after an initial period of systemic antibiotics, have led to such a strategy being proposed by some.[1127,1128] However, there is likely to be selection bias in those reports and the high growth and rupture rate observed for mycotic AAA makes deferred surgery risky unless rigorous surveillance is in place.

Recommendation 140		Unchanged
Patients with a suspected mycotic abdominal aortic aneurysm are recommended for treatment with intravenous antibiotics; empirical antibiotic treatment against *Staphylococcus aureus* and Gram negative rods, initiated as soon as cultures have been secured, followed by continued targeted therapy depending on the microbiology or continued empiric treatment in cases with negative cultures.		
Class	**Level**	**References** ToE
I	C	Sörelius *et al.* (2016),[1116] Sörelius *et al.* (2019),[1125] Shirasu *et al.* (2022)[1129]

Recommendation 141		Unchanged
Prompt surgical treatment of mycotic abdominal aortic aneurysms is recommended, irrespective of aneurysm size, due to the high rupture risk.		
Class	**Level**	**References**
I	C	Consensus

Despite the lack of evidence, OSR has long been regarded as the gold standard for definitive treatment of mycotic AAA. OSR includes resection of the aneurysm, extensive local debridement, and revascularisation by extra-anatomic bypass or *in situ* reconstruction. Options for *in situ* conduits include spiral graft made from long saphenous vein, autologous or heterologous cryopreserved femoral veins (so called neo-aorto-iliac system),[1130,1131] cryopreserved arteries,[761] bovine pericardium,[1132] or if unavailable prosthetic grafts (PTFE, Dacron, silver, or antibiotic soaked Dacron grafts).[1121,1133–1135] Multiple intra-operative samples should be obtained for culture. Extensive debridement should occur, and the infective process should be separated from the graft with pedicled omentoplasty.[1129] Mortality rates of up to 5 – 49% after *in situ* grafting *vs*. 24 – 50% after extra-anatomical bypass have been reported.[761,1121,1130,1133,1134,1136–1138] Infection related complications may occur in 0 – 20% after *in situ* reconstruction and older data suggest an equally high complication rate after extra-anatomic bypass, with the most feared being late aortic stump blow out in up to 20%.[1139] No reliable comparative data exist between the various open surgical techniques.

In the last 20 years mycotic AAAs have increasingly been treated successfully by endovascular means. EVAR has been regarded with scepticism because of major concerns about leaving the infected tissue in place, including the aneurysm itself, and the risk of persistent or recurrent infection. On the other hand, EVAR is a less invasive alternative to OSR of mycotic AAA, enabling treatment of fragile and comorbid patients with challenging aneurysm anatomy and avoidance of major surgical trauma (aortic cross clamping, heparinisation, and massive blood transfusion). In emergency situations EVAR may be a bridge to later definitive surgery, and for those unfit for OSR be a permanent or palliative treatment.[1127] A large European multicentre study including 123 patients with 130 mycotic AAAs (38% rupture and 52% suprarenal or thoracic) showed that EVAR may offer a durable treatment (55% five year survival) if associated with long term antibiotic therapy (6 – 12 months or possibly lifelong)[1114] but additional open and percutaneous procedures may be necessary to remove secondary lesions.[1116,1118] Late infection related complications do occur, especially within the first year after surgery, and are often fatal (European study 19% of total cohort), especially in patients with non-*Salmonella* positive blood cultures (41% five year survival), with immunodeficiency (40% five year survival), with peri-aortic or intrathrombus gas on preoperative CT scan (36% five year survival)[1114,1140] or with fever or rupture at the time of the operation.[1116,1127]

No direct comparative studies exist between OSR and EVAR for mycotic AAAs. A Swedish nationwide propensity score matched analysis of 132 patients with 144 mycotic AAAs, showed a significant early survival benefit for EVAR (up to four years) with no late disadvantages in terms of rates of late infection or aneurysm related complications or survival,[1116] suggesting that endovascular repair is an acceptable alternative to OSR. In a systematic review, including 963 patients from 28 studies, EVAR (*vs*. OSR) was associated with a lower 30 – 90 days mortality rate for both paravisceral and infrarenal mycotic AAAs, while no difference was seen between the techniques after five years.[1125] In a Japanese nationwide study, including 862 patients with mycotic AAAs, persistent or recurrent aneurysm related infections were significantly more frequent after EVAR than OSR (OR 2.8); however, after propensity score matching no differences in three year all cause and aorta related mortality was seen.[1141] In a recent meta-analysis, including 1 203 patients from 14 studies, the pooled recurrent infection rate was significantly higher after EVAR than OSR (RR 2.4), while infection related rupture or death, peri-operative death, one year death, and re-admission or re-intervention did not differ between the two groups. The conflicting literature highlights the problem of biased retrospective single centre studies. Not least in terms of selection bias, where fit patients are more often selected for open repair, while less fit, unstable patients, or those with challenging anatomy are treated endovascularly to a greater extent.[1129]

The antimicrobial regimen should be formulated on a case by case basis in close collaboration with infection specialists based on clinical, laboratory parameters, and imaging studies. Surveillance and duration of antibiotic therapy (ranging from 4 - 6 weeks to lifelong) are influenced by the microbiology, type of surgical repair, and immunological status of the patient.

In summary, mycotic AAA is a rare and life threatening disease. Early detection and treatment with antibiotics followed by surgical repair is central to their management. However, because of the variability in presenting symptoms and condition, anatomical complexity, and bacteriology, as well as the lack of strong evidence, an individualised approach is recommended, with EVAR being an acceptable alternative to OSR. Regardless, long term clinical and radiological surveillance on an individual basis is advocated. Finally, given the rarity and complexity of mycotic AAA, its management should be centralised to high volume centres with available multidisciplinary expertise (see Chapter 2).[1110]

Recommendation 142		New
Patients with mycotic abdominal aortic aneurysms are recommended to be referred to high volume vascular surgical centres, for multidisciplinary management.		
Class	Level	References
I	C	Consensus

Recommendation 143			Unchanged
The choice of surgical technique for the treatment of a mycotic abdominal aneurysm should be considered based on individual patient and lesion characteristics.			
Class	Level	References	ToE
IIa	C	Sörelius *et al.* (2016),[1116] Sörelius *et al.* (2019),[1125] Shirasu *et al.* (2022),[1129] Hosaka *et al.* (2021)[1141]	

Recommendation 144			Unchanged
Patients who have undergone mycotic abdominal aneurysm repair should be considered for an individualised post-operative antibiotic regimen and surveillance strategy, based on patient factors, microbiology, and the surgical technique used.			
Class	Level	References	ToE
IIa	C	Sörelius *et al.* (2016),[1116] Sörelius *et al.* (2019)[1125]	

10.2. Inflammatory abdominal aortic aneurysm

10.2.1. Definition and diagnosis of inflammatory abdominal aortic aneurysm. Inflammatory AAA, first labelled by Walker and colleagues in 1972,[1142] represents 5 – 10% of all AAAs.[1143,1144] Patients with inflammatory AAAs are about 5 – 10 years younger than patients with degenerative AAAs,[1145–1147] predominantly males (M/F ratio 6 – 30/1) and heavy smokers (85 – 90%), and often have hypertension, coronary artery disease and PAOD.[1146,1148]

Most inflammatory AAA belong to the group of chronic peri-aortitis (idiopathic peri-aneurysmal retroperitoneal fibrosis) and are characterised by (1) marked thickening of the aneurysm wall, (2) shiny white peri-aneurysmal and retroperitoneal fibrosis, and (3) dense adhesions of adjacent intra-abdominal structures.[1149,1150]

The pathogenesis of inflammatory AAA remains unknown. Autoimmune mechanisms are likely to be important in inducing this chronic inflammatory reaction, either by a local disease process based on an inflammatory reaction to components of atherosclerotic plaques or as a manifestation of a systemic disease.[1151] Based on immunological studies, a classification of inflammatory AAAs as immunoglobulin G4 (IgG4) related and IgG4 non-related has been proposed, emphasising an immunological role in the development of the disease.[1152] IgG4 related inflammatory AAAs which constitute approximately 50% of all inflammatory AAAs, risk developing IgG4 related systemic disease in other organs but rupture less frequent.[1152] Evidence of a genetic predisposition has also been demonstrated,[1153] but ultimately, the aetiology may be multifactorial.

The diagnosis of inflammatory AAA is based on a combination of clinical, laboratory, and imaging parameters.[1154] Inflammatory AAAs are associated with a higher frequency of aneurysm related symptoms (65 – 90%) than degenerative AAAs. A triad of chronic abdominal, back, flank or pelvic pain (50 – 80%), weight loss (20 – 50%), and elevated systemic inflammatory markers such as erythrocyte sedimentation rate, C reactive protein levels, and white blood cell count (60 – 90%) is highly suggestive of an inflammatory AAA.[1146] Clinical findings include a tender pulsatile AAA (15 – 71%)[1146,1155,1156] and ureteral obstruction causing hydronephrosis (10 – 50%)[1157] and chronic renal dysfunction (20%).[1158]

CTA remains the method of choice to detect the inflammation around the enlarged aorta with thickening of the adjacent tissues and potential entrapment of adjacent

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organs: duodenum and sigmoid colon (60%), ureteral obstruction (20 – 44%) with hydro-uretero-nephrosis (15 – 30%) and left renal or caval vein involvement (18 – 21%).[1159,1160] CTA detects the typical anatomical feature, the mantle sign; a thickened wall from chronic inflammatory cells and dense peri-aneurysmal fibrosis sparing the posterior wall, with possible involvement of adjacent structures such as the duodenum, ureters, left renal vein and inferior vena cava.[1146,1161] There is, however, no consensus on how to measure the diameter of an inflammatory AAA, whether it should include the thickened aortic wall or not,[1160] which complicates the decision making on the possible need for surgery. Including the peri-aortic inflammation or oedematous wall, however, risks greatly overestimating the diameter, and thereby forcing surgical repair of a *de facto* small AAA. Due to the increased risk of surgical complications and lack of increased risk of rupture, it is not advisable.

^{18}F-FDG PET/CT is a sensitive and specific imaging tool to detect and monitor the peri-aortic inflammation[1162–1164] and diffusion weighted MRI has emerged as a potential additional tool to diagnose and follow up inflammatory AAAs.[1165]

In the differential diagnosis mycotic AAA should be ruled out, and is facilitated by negative bacterial blood cultures, negative QuantiFERON-TB Gold test (tuberculosis), negative serological tests (syphilis, *Coxiella, Bartonella, Brucella*), negative indium 111 tagged white blood cell scan, and the typical morphological feature on CTA.

Recommendation 145		New
When measuring the diameter of inflammatory abdominal aortic aneurysms to determine the indication for repair, the peri-aortic inflammation or wall oedema should not be included.		
Class	**Level**	**References**
III	C	Consensus

10.2.2. Management of inflammatory abdominal aortic aneurysm.

The optimal management of patients with inflammatory AAAs remains uncertain and it is recommended that all patients with inflammatory AAA are managed and closely followed by a multidisciplinary team.[6]

Non-operative medical management with corticosteroids should be considered for symptomatic aneurysms with a diameter below the threshold for repair but severe pain and weight loss, associated with intense hydronephrosis, and a mantle sign suggesting peri-operative difficulties.[1166] The optimal dose and duration of medical treatment are still unclear since controlled clinical trials are lacking on the long term efficacy of steroids in inflammatory AAAs. Nonetheless, based on the recommended therapy for primary vasculitis, high dose corticosteroids therapy (30 – 80 mg/day prednisone equivalent) should be initiated for induction of remission. Once the disease is controlled, the glucocorticoid dose should be reduced to a target dose of $\leq$ 5 – 10 mg/day after one year.[1167] In addition to conventional immunosuppressants, adjunctive therapy (azathioprine, cyclophosphamide and methotrexate) may be required in selected patients as steroid sparing agents because of the side effects of steroids or in steroid refractory cases.[1168–1171] Rituximab was shown to be effective in IgG4 related diseases in an open label pilot trial.[1172]

Tamoxifen (a selective oestrogen receptor modulator) has been used in the treatment of idiopathic retroperitoneal fibrosis, based on its usefulness in pelvic desmoid tumours. In a prospective single centre study, 15/19 patients treated with tamoxifen, 20 mg orally twice daily, reported substantial resolution of symptoms, improved acute phase reactants and signs of regression on gallium and CT scanning after a median treatment duration of 2.5 weeks.[1173] Tamoxifen in combination with steroids has been suggested to be effective in inflammatory AAAs.[1170]

Acute phase reactants (erythrocyte sedimentation rate, C reactive protein) alone are not reliable for follow up as they are often not concordant with the metabolic assessment of the disease and normalisation of erythrocyte sedimentation rate occurs earlier during follow up.[1146,1156,1174] A prospective trial of retroperitoneal fibrosis imaging has shown that ^{18}F-FDG PET may help to guide decisions about initiation or cessation of steroid treatment. Patients with a maximum standard uptake value $\geq$ 4 are 10 times more likely to respond to steroid therapy than those with a value $<$ 4.[1175]

Recommendation 146			Unchanged
All patients with symptomatic inflammatory abdominal aortic aneurysms should be considered for medical anti-inflammatory treatment, with corticosteroids being the initiation therapy of choice.			
Class	**Level**	**References**	**ToE**
IIa	C	Paravastu *et al.* (2009),[1157] Vaglio *et al.* (2011),[1168] van der Bilt *et al.* (2016),[1169] Skeik *et al.* (2017)[1171]	

Surgical treatment of inflammatory AAAs poses a different challenge to surgeons compared with standard degenerative AAAs due to the increased risk of iatrogenic bowel, caval, iliac vein, and ureteral injuries during OSR and the increased inflammatory response to endoprosthesis implantation. Patients operated on for inflammatory AAAs have higher early mortality and complication rates but equivalent long term outcomes compared with a matched cohort of patients with degenerative AAAs.[1176]

The risk of inflammatory AAA rupture is reported to be low ($<$ 5%).[1150] Hence, the same diameter threshold at which repair is considered as for degenerative AAAs is indicated, and only rarely may surgical treatment be indicated on symptomatic refractory cases despite medical treatment, to control the inflammatory process.[1177]

OSR can be extremely challenging due to the high peri-aortic fibrotic adhesion to the duodenum, left renal vein,

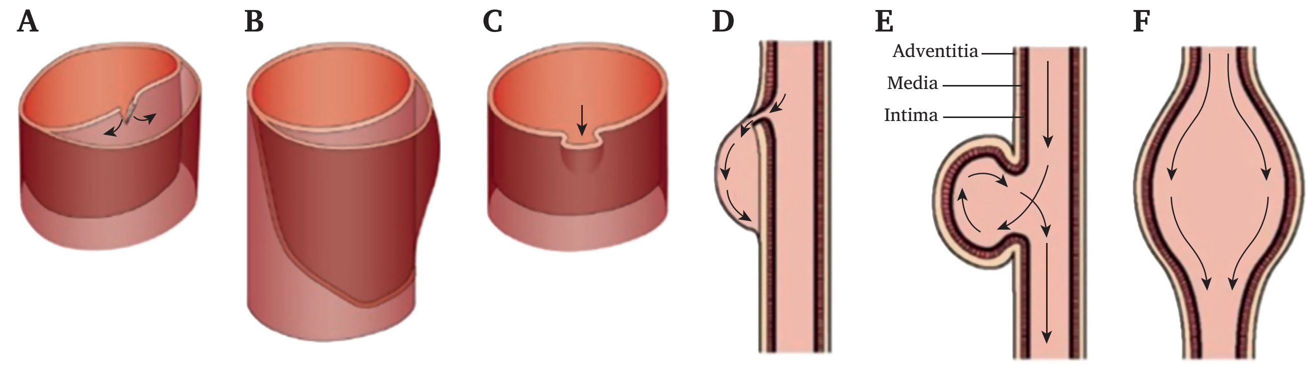

Figure 12. Illustration of (A) aortic dissection, (B) intramural haematoma, (C) penetrating aortic ulcer, (D) pseudoaneurysm, (E) saccular aneurysm, (F) fusiform aneurysm.

inferior vena cava, and ureters.[1177] Intra-operatively, inflammatory AAAs appear white and shiny.[1142] Extensive adhesiolysis of peri-aneurysmal structures should be avoided to limit the risk of iatrogenic injuries. Peri-aortic fibrosis provides a hostile operative field which explains the reported higher intra- and post-operative morbidity and mortality (6 – 11%) after open inflammatory AAA repair.[1157] A modified transperitoneal approach with limited dissection[1148] is believed to reduce the risk of iatrogenic injury and more safely gain proximal and distal control of the aneurysm distant from the thickened parts of the aneurysmal wall,[1144] leaving the duodenum attached to the thickened peel. For that purpose, suprarenal aortic cross clamping may be required in up to 40% of cases,[1178] as well as extended reconstruction of the external iliac or femoral arteries. Moreover, pre-operative ureteral stent placement is required in a majority of patients (90%) to release hydronephrosis and help identification of the ureter during surgery.[1155]

After OSR, regression of peri-aneurysmal inflammation and fibrosis is observed in up to 86%, and regression of associated hydronephrosis in up to 80%[1148,1178] but can take several years to complete. Graft related complications are described in 9%, including para-anastomotic pseudoaneurysms and GEF.[1179]

In anatomically suitable patients, EVAR should be considered as a first line treatment option because of the observed lower 30 day mortality rates (2.4%)[1157] and fewer major complications.[1155,1180,1181] In most cases, peri-aneurysmal fibrosis post-EVAR resolves at a slower rate compared with OSR.[1155,1182,1183] With respect to hydronephrosis, it is unclear whether EVAR alone has any beneficial effect and it could be a slow process. Hence, the initial short term benefit should be counterbalanced with possibly higher rates of hydronephrosis in need of double J stenting over time.[6]

Hydronephrosis and peri-aortic fibrosis may persist and even progress despite OSR or EVAR Therefore, lifelong surveillance[1157,1159] and continued immunosuppressive therapy[1168,1170] remain warranted after inflammatory AAA repair, and pyelostomy, or lysis by means of open surgery, may be required.

Recommendation 147			Unchanged
Patients with an inflammatory abdominal aortic aneurysm should be considered for repair at an aneurysm diameter of ≥ 55 mm, with endovascular repair in preference to open surgical repair if the anatomy is suitable.			
Class	**Level**	**References**	**ToE**
IIa	C	Stone *et al.* (2012),[1155] Paravastu *et al.* (2009),[1157] Duque Santos *et al.* (2018),[1180] Ockert *et al.* (2006)[1181] Kakkos *et al.* (2015)[1182]	

10.3. Penetrating aortic ulcer, pseudoaneurysm, intramural haematoma, local dissection, and saccular aneurysm

Penetrating aortic ulcer (PAU) is defined as ulceration of an atherosclerotic plaque that penetrates through the aortic intima resulting in a variable amount of haematoma within the aortic wall (Fig. 12). These lesions typically occur in elderly patients with systemic atherosclerosis and associated comorbidities. Based on a literature review, the estimated incidence is 1% in the vascular population, with abdominal PAU (11 – 24%) being less common than thoracic PAU (76 – 86%) but multiple lesions and associated aneurysms may be noted.[1184] Progression of PAU may lead to intramural haematoma (IMH), pseudoaneurysm formation (dilatation of the aorta due to disruption of all wall layers, which is only contained by peri-aortic connective tissue, also called false aneurysm), rupture (extra-aortic haematoma), and lower limb embolisation.[1184,1185] PAU are symptomatic in 18 – 70%, causing pain (52%) or acute lower limb ischaemia because of distal embolism (12%) or rupture (4 – 7%).[1184,1186,1187]

Saccular AAAs are regarded as a separate entity defined as spherical aneurysms involving only a portion of the aortic circumference.[1188] Infection should always be excluded, and if present managed accordingly (see section 10.1). The optimal management of non-infected saccular AAA, including when to intervene, requires further research and should currently be based on individual risk assessment.

Owing to the uncertainty about a possible increased rupture risk[1188,1189] early treatment, with a lower diameter threshold for elective repair than for standard fusiform AAA, may be considered.

Isolated abdominal aortic dissections (IAADs) are rare and much less common than abdominal aortic dissection associated with thoracic aortic dissection.[1190] The dissection is related to a tear in the intimal layer and subsequent blood flow through the tear into the media creating a false lumen. The entry tear generally originates below or at the level of the renal arteries (82%).[1191] A concomitant AAA is present in 41% of patients with symptomatic IAAD.[1190] IMH represents blood in the aortic wall without an intimal tear or entry point on imaging[936] and rarely exists in the abdominal aorta alone. If IAAD, IMH, or pseudoaneurysms are detected in the abdominal aorta, trauma, iatrogenic injury or PAU as an underlying cause should be excluded.[1192] The most common complaint is abdominal, back, or flank pain (57 – 62%), sometimes associated with acute lower limb ischaemia 5%.[1190,1193]

Both CT and MRA enable the diagnosis of PAU, IMH, and IAAD with a high degree of accuracy. PAUs are characterised by a contrast filled crater that communicates with the aortic lumen. IMH is a crescentic area of smooth high attenuation within the aortic wall, detected on unenhanced CT. Intramural blood pools are frequently observed but are not associated with a poor prognosis and should be distinguished from ulcer like projections.[1161] Dissection presents as a linear filling defect in the aortic lumen with the true lumen often smaller than the false lumen. The craniocaudal extent of a PAU is much shorter than an IAAD or primary IMH.

Serial imaging surveillance by cross sectional imaging (CTA or MRA) is justified since the natural course of these pathologies remains unknown[1187,1194] with reportedly highly variable growth rates.[1195,1196]

Complicated PAU refers to a co-existing extra-aortic haematoma (pseudoaneurysm), embolisation symptoms and recurrent pain.[1194,1196] Likewise, complicated IMH or IAAD means the presence of recurrent pain, expansion of the IMH, peri-aortic haematoma, intimal disruption, or malperfusion.[1197]

Although the natural history of these processes has not been clearly described, for every patient with PAU, IMH, or IAAD medical management should be initiated and is essentially based on of the same concept used for type B aortic dissections, with reduction of the BP, management of atherosclerotic risk factors and optimal pain control.[936] A complicated PAU, IMH, or IAAD requires invasive treatment, as do IAADs which are associated with concomitant aneurysms even for lesions with a diameter $<$ 50 mm[1191,1193,1198,1199] although some have advocated a more aggressive approach if the overall aortic diameter is $>$ 30 mm.[1191,1198,1199] In a systematic review, including 482 patients with IAAD from 17 studies, patients with conservative treatment had an all cause 30 day mortality rate of 1%, a long term mortality rate of 5% (after 43 month follow up), and an intervention rate during follow up of 18%. Patients with OSR had a 30 day mortality rate of 9%, a long term mortality rate of 12%, and a re-intervention rate of 9%. Patients with endovascular repair had a 30 day mortality rate of 2%, a long term mortality rate of 5%, a re-intervention rate of 6%, and a persistent endoleak rate of 4%.[1200]

The focal nature of these pathologies renders them ideal targets for endovascular repair with stent grafts. This can be achieved with high technical success rates in complicated cases, but the procedure may be associated with ahigh in hospital mortality rate (10%) because of the frailty of the population affected.[1194,1201]

Recommendation 148		Changed
Patients with an uncomplicated* penetrating aortic ulcer, isolated dissection, or intramural haematoma of the abdominal aorta should be considered for conservative management with best medical treatment and continued surveillance.		
Class	**Level**	**References**
IIa	C	Consensus

* No expansion, co-existing peri-aortic or extra-aortic haematoma (pseudoaneurysm), embolisation symptoms, recurrent pain, and or malperfusion.

Recommendation 149		Changed
Patients with a pseudoaneurysm or complicated* penetrating aortic ulcer, isolated dissection, or intramural haematoma in the abdominal aorta should be considered for surgical treatment, preferably by endovascular means.		
Class	**Level**	**References**
IIa	C	Consensus

* Expansion, co-existing peri-aortic or extra-aortic haematoma (pseudoaneurysm), embolisation symptoms, recurrent pain, and or malperfusion.

Recommendation 150		Unchanged
Early surgical treatment (open or endovascular) may be considered for penetrating aortic ulcer and saccular abdominal aortic aneurysms, with a lower diameter threshold for elective repair than for a standard fusiform abdominal aortic aneurysm.		
Class	**Level**	**References**
IIb	C	Consensus

10.4. Concomitant malignant disease

The reported incidence of concomitant malignant diseases and AAA varies between 3% and 17%.[1202,1203] Cancer may be detected incidentally on CTA for AAA assessment or the aneurysm may be found during investigations for symptomatic malignancy. It represents a challenging issue in terms of treatment priority, timing, and expected outcome.

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Most published papers consist of small case series. Hence, decisions should be made based on clinical judgement applied individually in a multidisciplinary setting. Being a prophylactic procedure AAA repair is only worthwhile if the lifetime risk of rupture exceeds the risk of treatment in patients with a reasonable life expectancy. The prognosis of concomitant cancer is therefore central in the decision making process together with other comorbidities (age, physiological wellbeing) and patient preference. Other considerations are a perceived increased risk of AAA rupture following abdominal cancer surgery[1204,1205] *vs*. a significant delay in the treatment of cancer if AAAs are treated by OSR first, and the risk of graft infection.

Cytotoxic chemotherapy did not increase aneurysm growth compared with patients not undergoing treatment for malignancy in retrospective analyses[1206,1207] but a retrospective single centre analysis including 217 patients with AAA with 238 synchronous malignancies suggested that antimetabolites as part of chemotherapy may increase the annual AAA growth rate and closer monitoring of these patients with AAA was suggested.[1208] Furthermore, only nine patients with AAA and concomitant cancer receiving chemotherapy have been reported in the literature needing urgent aneurysm surgery, which could possibly be explained by under reporting or representing the normal biological variability observed in aneurysm disease.[1202,1206,1209,1210]

Two meta-analyses, focusing on management of AAA and concomitant abdominal neoplasms, included different mostly retrospective case studies but came to the same conclusion; treat what is most threatening or symptomatic first (large AAA, obstructing colonic cancer, bleeding gastric cancer, etc.).[1211,1212]

Since OSR of AAA prior to resection of a gastrointestinal cancer may result in a delay of months in comparison to days post EVAR,[1204,1211–1214] the AAA should preferably be considered for EVAR if anatomically suitable followed by staged cancer surgery within two weeks. EVAR also has an evolving role during combined interventions.[1215] This would allow for a minimum delay in the treatment of both the aneurysm and the cancer, as well as a reduced risk of graft infection. A high procedure related mortality and morbidity has been observed when open AAA repair is carried out prior to gastrointestinal cancer resection, often weeks or months later, as opposed to cancer surgery first: 19% and 42% *vs*. 9% and 26%, respectively.[1216]

If both lesions are life threatening (e.g., large aneurysm with advanced obstructing malignancy), and the anatomy is not suitable for endovascular repair, or if the patient is young[1217] a synchronous open approach may be chosen, providing great attention to detail (patient selection, blood supply to avoid bowel necrosis, irrigation, and omental wrap to avoid infection) understanding that cumulative morbidity and mortality are higher in these single stage operations.[1216]

The overall survival rates post EVAR in patients treated for concomitant cancer are naturally poorer because of progression of the neoplastic disease and are influenced by type, stage, and grading of the malignancy: 58% at four to five years for colorectal cancer[1215,1216] and 15% at three years for lung cancer.[1218] In lung cancer and pancreatic cancer, staging is crucial before considering AAA treatment because the overall survival correlates closely with the stage of these cancers.[1218,1219]

As with any patient with severe concomitant comorbidities and underlying chronic disease with a poor prognosis, management of rAAA in a patient with advanced cancer disease, previously deemed inappropriate for elective repair, should be discussed with the patient and the family, with emphasis on the futility of attempting repair, and the patient's wishes should be made clear to family or other parties involved.

Overall, there is an increased risk of DVT and pulmonary embolism after OSR of AAA,[1220] but in patients with AAA and concomitant cancer also of limb thrombosis post-EVAR (up to 7.4%), possibly because of hypercoagulability, thrombophilia, para-neoplastic syndrome, chemotherapy, and lithotomy position.[1211–1214,1221] Prolonged LMWH prophylaxis up to four weeks should be considered post-operatively in patients with concomitant cancer.[1222]

Recommendation 151		Unchanged
Patients with an abdominal aortic aneurysm and concomitant cancer are not recommended a different indication (threshold diameter) for prophylactic aneurysm repair than patients without cancer, including cases of chemotherapy.		
Class	Level	References ToE
III	C	Martin *et al.* (2015),[1206] Maxwell *et al.* (2021),[1207] Kumar *et al.* (2016)[1212]

Recommendation 152		Unchanged
Patients with a large or symptomatic abdominal aortic aneurysm with an indication for repair and concomitant malignancy should be considered for a staged surgical approach, with endovascular repair first, to allow for treatment of the malignancy with minimal delay.		
Class	Level	References ToE
IIa	C	Kouvelos *et al.* (2016),[1211] Kumar *et al.* (2016)[1212]

Recommendation 153		Unchanged
Patients with concomitant cancer should be considered for prolonged low molecular weight heparin prophylaxis for up to four weeks after abdominal aortic aneurysm repair.		
Class	Level	References ToE
IIa	C	Pawlaczyk *et al* (2016),[1220] Felder *et al.* (20190[1223]

10.5. Genetic syndromes

Although classic cardiovascular risk factors are the leading cause of AAA, in young patients (< 60 years) and in those

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with a positive family history or with physical features associated with monogenetic syndromes (loose skin, joint hypermobility, multiple or atypical vascular aneurysms), a specific diagnostic approach is needed to look for underlying genetic or connective tissue disorders, or both. More than 30 heritable conditions have been described that can potentially manifest with aortic or arterial aneurysms. The same heritable aortic disease usually associated with the thoracic aorta can also affect the abdominal aorta, but to a much lesser extent, such as Marfan syndrome, vascular Ehlers—Danlos syndrome (vEDS), Loeys—Dietz syndrome, arterial tortuosity syndrome, and aneurysm osteoarthritis syndrome.[1224]

Mutations in genes encoding for extracellular matrix components (e.g., Fibrillin 1, Collagen Type III Alpha 1 Chain, Collagen Type IV Alpha 5 Chain); the smooth muscle cell contractile apparatus (e.g., actin alpha 2 smooth muscle aorta, protein kinase cyclic guanosine monophosphate dependent type I); transforming growth factor beta 3 signalling pathway (e.g., TGFBR 1, 2, Small Mothers against decapentaplegic homolog 3 [Smad3], TGFB3) are known to be associated with increased risk of abdominal aortic pathology and aneurysm formation. Variability in clinical presentations among individuals with identical mutations can be significant.[1225,1226]

Appropriate genetic counselling and testing of the patient and family members should be initiated early, not only to establish proper medical and surgical management in the individual patient but also to uncover implications for family members. Genetic assessment involves screening, diagnosis, and counselling for individuals at risk of or affected by connective tissue disorders. After assessment of personal and family history for features of a suspected condition, genetic counselling is provided to patients who meet the criteria, to share genetic risk information and discuss the benefits, risk, and limitations of genetic testing if indicated for the patient and or at risk relatives.[1227] Comprehensive gene panel testing is becoming more common and readily available. The next generation sequencing test is a simple blood test designed to detect mutations in the coding region of most genes associated with connective tissue disorders. Diagnostic vascular imaging should not only focus on the known pathological features but also provide a complete overview of the cerebral, thoracic, and abdominal vasculature using whole body MRA and transthoracic echocardiography.[1228]

Management strategies, including imaging surveillance (CTA, MRA, and DUS), medical treatment, or surgical intervention, for the individual patient should be discussed within a multidisciplinary aortic team. An individual approach is paramount since the rupture risk is higher at smaller aortic diameters in for example Loeys—Dietz syndrome (TGFBR1,2) and aneurysm osteoarthritis syndrome (Smad3) than in Marfan (Fibrillin 1) patients, and surgical repair is more challenging in vEDS owing to the increased arterial wall fragility than in Marfan's syndrome and Loeys—Dietz syndrome. Thus, the diameter threshold at which repair is considered should be individualised and largely depends on the underlying genetics.

If surgical treatment is considered OSR is generally to be preferred using specific repair techniques due to vessel friability, for example delicate and atraumatic handling of tissues and sewing of anastomoses with pledgeted sutures, and use of supporting cuffs and glues. More recently, particularly in patients with an increased surgical risk because of redo procedures or in emergencies as a bridging procedure, a gradual move towards endovascular repair has been observed.[1229] However, due to the basis of these diseases, with vascular fragility and high risk of continued aneurysm development with uncertain durability of endovascular treatment in this patient group, this approach cannot be recommended for routine use in the elective treatment of AAA with underlying genetic causes.

Vascular EDS (Collagen Type III Alpha 1 Chain) is a dominant, inherited, rare, and most serious of the connective tissue disorders with inherent vessel friability that causes arterial dissection and ruptures with a high mortality rate. Treatment with the cardioselective beta blocker celiprolol, with β2 agonist vasodilatory properties was shown in a RCT Beta blockers in Ehlers-Danlos Syndrome Treatment (BBEST) trial, including 53 patients with vEDS, to be associated with a three fold decrease in arterial rupture after 47 months of follow up (HR 0.36; 95% CI 0.15 – 0.88).[1230] The protective effect of celiprolol was confirmed in a retrospective cohort study, including 144 patients with vEDS of whom $>$ 90% were treated with 400 mg/day celiprolol. After a median of 5.3 years of follow up the overall survival was high (72%) and more than two thirds of patients remained clinically silent, despite a large number (51%) with previous arterial events. Treatment with celiprolol was associated with a dose dependent significantly better survival.[1231] In a cohort of 45 patients with vEDS on celiprolol, the annual risk of major vascular events was 4.7%, similar to the treatment arm of the BBEST trial (5%) and lower than in the control arm of the same trial (12%).[1232]

A recent large retrospective analysis of 126 patients with confirmed molecular diagnoses from The UK National Diagnostic Service for Ehlers—Danlos syndrome (EDS) showed that those patients on a long term angiotensin II receptor blocker and or beta blocker had fewer vascular events than those not on cardiac medication who received the same lifestyle and emergency care advice during a mean five years follow up.[1233] The potential beneficial effect of angiotensin II inhibitors in vEDS needs to be verified in controlled studies.

Experience of invasive treatment is limited to case reports and small case series.[1234,1235] A recent international consensus report on the diagnosis, natural history, and management of vEDS concluded that contained ruptures may be treated conservatively, with close monitoring to detect recurrent bleeding. Non-contained ruptures, clinically unstable aneurysms (pre-rupture), and false aneurysms often require intervention. Depending on the location, endovascular treatment (embolisation of the bleeding artery), or open surgery (aorta and iliac vessels) may be indicated although invasive procedures may provoke further morbidity. These patients are best managed by multidisciplinary teams (vascular surgeons, cardiologists, cardiothoracic surgeons, geneticists, and other specialists) in tertiary centres of excellence with expertise in managing connective tissue disorders, including genetic family assessment.[1229,1236] International multicentre collaborations such as the European Reference Network on Rare Multisystemic Vascular Diseases (http://vascern.eu/)[1237] will play an important role in improving the knowledge of the management of this rare disease.

Recommendation 154		Changed
Patients with an abdominal aortic aneurysm with a suspected underlying genetic cause, such as early onset (< 60 years) or positive family history of aneurysmal disease, or with physical features associated with monogenetic syndromes, are recommended for genetic evaluation.		
Class	**Level**	**References** ToE
I	C	van der Linde *et al.* (2013),[1224] Brown *et al.* (2013)[1227]

Recommendation 155		Unchanged
Referral to a multidisciplinary aortic team at a highly specialised centre is recommended to manage patients with an aortic disorder suspected of having an underlying genetic cause.		
Class	**Level**	**References**
I	C	Consensus

Recommendation 156		New
Patients with vascular Ehlers–Danlos syndrome are recommended prophylactic treatment with celiprolol.		
Class	**Level**	**References** ToE
I	B	Ong *et al.* (2010),[1230] Baderkhan *et al.* (2021)[1232]

Recommendation 157		Unchanged
In young patients with suspected connective tissue disorders and an abdominal aortic aneurysm, open surgical repair is recommended as first option.		
Class	**Level**	**References**
I	C	Consensus

Recommendation 158		New
For patients with abdominal aortic aneurysms with an underlying genetic cause, the threshold diameter for considering repair should be individualised, depending on the underlying genetics and anatomy.		
Class	**Level**	**References**
I	C	Consensus

11. SHARED DECISION MAKING WITH SUPPORTING INFORMATION FOR PATIENTS

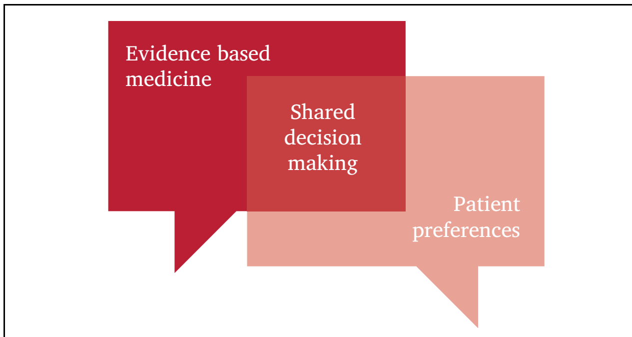

Figure 13.

11.1. Shared decision making

No decision about me, without me.

SDM is a process focusing on best quality patient centred healthcare, which respects patients' views, preferences, and autonomy.[1238]

The concept of SDM is 40 years old and practiced when more than one treatment option is available, and the patient and healthcare professionals jointly evaluate the available evidence and treatment options in order to undertake decisions together in formulating a care plan.[1239] SDM is particularly challenging in surgical disciplines where treatments are often irreversible and can have unintended harms with consequent impact on the future lifestyle of the patient. These harms are not as readily withdrawn as is the case for drug therapy. The benefits of SDM may be more apparent for patients (including reduced decisional conflict and improved satisfaction with their clinical care) than for surgeons, but SDM has been proven to have important overall healthcare benefits including improving the patient–clinician relationship, increasing patient compliance, and reducing healthcare costs by avoiding unwanted treatments. Prophylactic procedures in vascular surgery, including AAA screening and elective repair, are important areas in which to implement SDM.[1240] Implementation of SDM is likely to involve changes in the decision making process for the majority of both patients and clinicians. This chapter will summarise the available evidence concerning SDM relating to elective AAA repair and briefly consider the use of decision aids for AAA screening and surveillance. The application of SDM to ruptured AAA is not discussed because of the high pain levels and varying cognition of these patients in the emergency situation.

Currently there is little evidence regarding the implementation of SDM into clinical practise for elective AAA repair, AAA screening or surveillance but the available evidence has been summarised in a scoping review.[1241] Fifteen RCTs assessing strategies for the facilitation of SDM across differing surgical specialties[1242] consisted of provision of information to patients and the time needed to support patient–clinician communication to reach an agreed care plan. For communication a three talk model has been proposed: (1) discuss the choices; (2) discuss the options; (3) reach a joint decision for the care plan. It also is necessary to identify the best way of presenting the information and evidence to patients.[1239] This can be reduced to a two talk model if patients are provided with good quality information before the first consultation.

11.1.1. Preference for shared decision making. Several studies have evaluated patient preference for SDM for elective AAA repair. Reported patient preference for SDM in AAA repair ranges from 58% in a Dutch study to 95% in a study in the USA.[1243,1244] However, studies evaluating whether patients felt that they had been involved in deciding their care plan suggest that SDM had been implemented in less than half.[1244–1246]

There are no studies on surgeon preference for SDM for elective AAA repair. There is only a single study that has evaluated objectively whether vascular surgeons implemented SDM in their consultations; with 19/54 patients facing repair of an asymptomatic AAA, adequate SDM was identified in only 7/19 of these consultations.[1244] This is broadly consistent with a systematic review across the surgical specialties where 7 – 39% of cases were found to include adequate SDM, although surgeons perceived SDM to have been implemented in almost half of cases.[1247]

11.1.2. Patient choice *vs.* clinician choice. A recent survey from the USA, including 99 patients in Veterans Affairs hospitals facing elective AAA repair and considered suitable for both EVAR and open repair, revealed that 41% had received no information about open repair, 37% had received no information about EVAR, and the issue of conservative management was not even addressed.[1246] In a companion paper, the lack of information about alternative treatments was given as an important reason for the treatment received.[1248] Information about alternative treatments should be provided to the patient, even when they are not available at the consultation centre or fall outside the expertise of the consulting clinician.

11.1.3. Patient information, with definitions of decision aids and decision support tools. To engage in shared decision making patients need good quality unbiased information, where does this come from? The survey of 99 patients showed that 41% of the patients got no information, about one in six received information from primary healthcare and one in 10 obtained information from the internet, other sources included friends, family, and television.[1246] Assessments of the available information for patients through online sources has been rated as poor.[1249,1250] A more recent study identified that patient directed online information about open surgery and EVAR was mainly text, lacking in visual information, and needed an advanced reading age for its comprehension.[1251] There is scant information about conservative management and rupture risk.

Patients, especially older ones, are particularly interested in the short term outcomes, complications and speed of recovery after AAA repair.[1248] The information wanted by patients may, however, differ from the information currently available to clinicians. An ongoing work, of the development of a Core Outcome Set for elective AAA repair is discussed in section 2.1.

Decision aids are interventions that support patients by making their decisions explicit, providing information about options and associated benefits and harms, and help clarifying congruence between decisions and personal values. Decision aids should contain infographics and can be provided in leaflet, card, or digital formats. Their use has been shown to improve patient knowledge, risk perception and to enable value congruent choices across clinical medicine: their use is not associated with any harms.[1252]

Decision support tool (DSTs) is another name for a patient decision aid used by some investigators but the term can also be used for clinician based use in supporting the ongoing social and clinical care needs of dependent patients. When this term is used in the context of SDM, it should be prefaced by patient.

Both decision aids and patient DSTs differ from other patient resources as they are designed to provide information regarding potential treatment options without instructing patient behaviour, and should be available to patients either before the first consultation with a vascular surgeon or before deciding to attend for AAA screening. They may be provided in leaflet, card or digital format and should contain infographics. Recent work indicates that vascular surgery patients prefer digital decision aids.[1253]

11.1.4. Decision aids to improve patient knowledge. There are several reports, including three RCTs, on the use of preconsultation AAA specific decision aids for patients facing elective AAA repair.[1254–1256] Decision aids improved information provision as ascertained by patient reported perceived knowledge and objective assessment.[1254,1255] The first RCT reported a sustained increase in perceived and objective levels of aneurysm related knowledge. However, despite this increase in knowledge, the decision aid did not improve objective markers of decision making (i.e., reduce decisional conflict scores).[1254] Subsequently two further randomised trials have reported on the use of DSTs. The OVIDIUS trial (Operative Vascular Intervention Decision making Improvement Using SDM tools), a Dutch stepped wedge cluster randomised trial, showed that patients with AAA demonstrated significantly higher knowledge scores after introduction of these decision aids (median increase score of 40% for patients with AAA, $p < .005$.[1257] The proportion of participants opting for conservative management strategies after implementation of DSTs increased significantly from 7.4% to 28.8% but the proportion of participants opting for EVAR *vs.* open repair did not change significantly after DST implementation. The PROVE AAA cluster randomised trial in the USA (PReferences for Open *vs.* Endovascular repair of AAA) reported that patients exposed to a decision aid were more likely to receive their preferred AAA repair type, with

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95% in agreement in the decision aid group in comparison to 86% in agreement in the control group.[1258] There were equivalent proportions of patient preference for EVAR and open repair across both DST and control groups, with 79% and 76% expressing a preference for EVAR respectively; conservative management was not considered as an option. In a sub-study of the AAA IMPROVE trial, it was reported that the rate of preference for open repair was twice as high in non-retired (still working) *vs*. retired participants.[1248]

The decision whether to accept an invitation to AAA screening is a situation where the invitee needs information or a decision aid to support their decision. Decision aids for AAA screening are available in Canada (https://decisionaid.ohri.ca/AZsumm.php?ID=1428)[1259] and England (https://www.nhs.uk/conditions/abdominal-aortic-aneurysm-screening/)[1260] but the role of these in helping the patients reach a decision or in reducing decisional conflict has not been formally assessed. A recent study has addressed patients' preferences concerning surveillance intervals for small AAA and 78% found a decision aid to be useful in forming their preferences.[1261]

11.1.5. Implementation. The National Institute for Health and Care Excellence (NICE) [NG197] guidelines recommend appointment of a senior healthcare professional as a service user champion to increase accountability and responsibility in the implementation of SDM.[1262] This recommendation for involvement of high level leadership is alongside the development of quality improvement projects to deliver SDM. However, there is no evidence to support this recommendation and the practicalities of delivering it will probably differ across healthcare systems.

To summarise, SDM is essential to providing best quality care, but it is not embedded in current vascular surgical practice. Therefore, it is important that SDM is always considered in the encounter with the patient or their relatives and carers. For this, the provision of good information is key, and the use of DSTs should be considered to further assist patients in decisions.

Recommendation 159			New
Shared decision making should be facilitated during conversations around abdominal aortic aneurysm screening, surveillance and the management of large asymptomatic abdominal aortic aneurysms being considered for repair.			
Class	Level	References	ToE
I	B	Machin *et al.* (2023)[1241]	

Recommendation 160			New
Use of decision support tools to assist patients in their decisions about the management of abdominal aortic aneurysms being considered for repair should be considered.			
Class	Level	References	ToE
IIa	A	Knops *et al.* (2014),[1254] Stubenrouch *et al.* (2022),[1257] Eid *et al.* (2022)[1258]	

11.2. Information for patients

This information has been developed by the ESVS. In order to provide guidance for healthcare professionals involved in the care of patients with AAA the ESVS produces guidelines and recommendations. The ESVS guidelines committee for AAA has produced a full set of guidelines for professionals, which is the main part of this document.

The next part of the document contains the same information but presented in a format for non-experts, with the aim of providing unbiased information to patients and their relatives and carers, to facilitate shared decision making. Details of the process used to develop this information, and how strong the evidence is for each piece of information, are given at the end of this section. Where very good evidence for the management of people with AAA has been found, it has been included in the information presented here.

What is an abdominal aortic aneurysm? An abdominal aortic aneurysm is a swelling or ballooning of the main artery in the body as it takes blood through the belly to supply the legs (Fig. 14A). These aneurysms are very rare before the age of 60 years. They are more common in people who have smoked (current smokers or ex-smokers) than in those who have never smoked. They are also more common in men than in women. A minority of patients may have a strong genetic cause for the abdominal aortic aneurysm.

Most aneurysms do not cause any symptoms and patients with an aneurysm usually do not realise they have one until it is found by a doctor as a result of invitation to an aneurysm screening programme, other medical tests, or in the event that the aneurysm bursts.

How is an abdominal aortic aneurysm diagnosed? Occasionally, an AAA is found by a doctor while examining the tummy of a patient. However, this is not always reliable. A better way to confirm the presence of an AAA is by an ultrasound scan of the abdomen. This ultrasound scan does not involve any radiation and is quick and simple. In most cases an AAA remains unsuspected before it is found, either as part of a screening programme or from an ultrasound, or other scan, undertaken for a different complaint.

What about screening for abdominal aortic aneurysm. Offering ultrasound screening to groups at higher risk of having an aneurysm reduces the risk of dying from a burst AAA. It does this because there are safe and effective ways to treat or repair the AAA before it bursts. This increases the number of AAA repairs performed, but since it saves lives and costs less than treating burst AAAs, it can provide a cost-effective health strategy by finding aneurysms before they burst. Offering screening does increase the number of people who require operations to repair an AAA, but these operations are much safer than leaving an aneurysm alone. Screening has been shown to be cost-effective in men aged 65 and older, but presently there is little information about whether higher risk groups of women would benefit from screening.

- We recommend that all at high risk of AAA should be offered a one time ultrasound screening examination

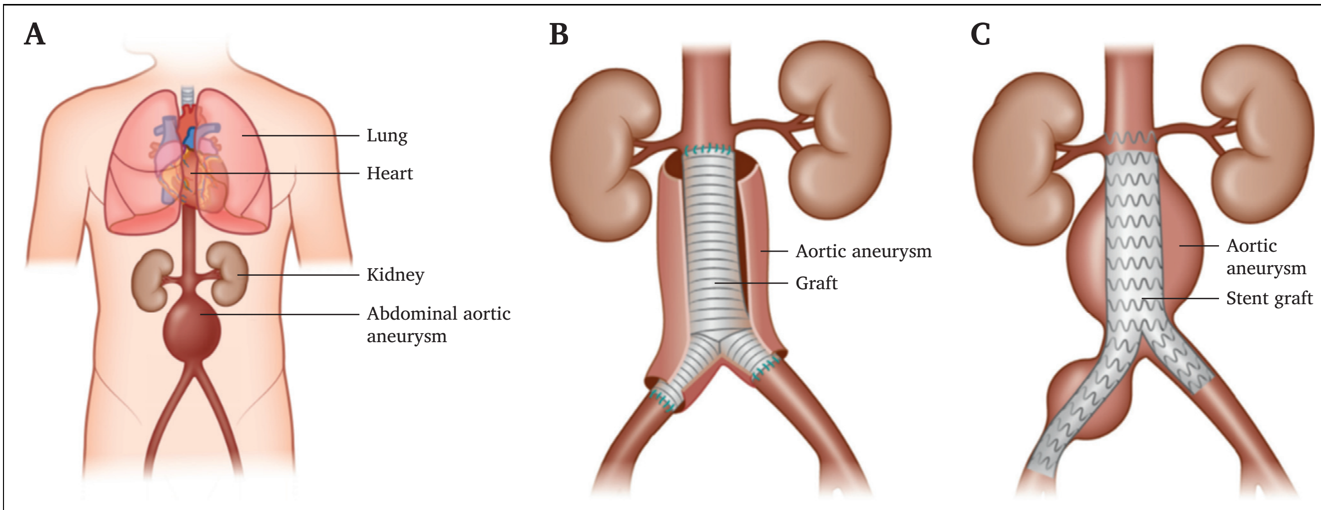

Figure 14. (A) (Simple) abdominal aortic aneurysm (AAA). (B) Open surgery for an abdominal aortic aneurysm (open AAA repair). The affected segment of the aorta is replaced with a material graft stitched in place. (C) Endovascular AAA repair. A stent graft is placed inside the aneurysm to reline the aorta and prevent the aneurysm bursting.

of their tummy to look for the presence of an abdominal aortic aneurysm. Higher risk groups that should be offered screening are elderly ($\geq$ 65 years) men in general, men and women with an immediate family relative with an aneurysm (in the abdominal aorta or another artery).

What happens if I am diagnosed with an abdominal aortic aneurysm? If you are diagnosed with AAA you will be told whether it is small (between 30 mm and 54 mm in diameter) or large (55 mm or bigger). The size of an aneurysm is usually measured on an ultrasound scan from the front to the back. If it is measured by a different imaging method, the size is usually slightly bigger than reported from the ultrasound scan. However, it is the ultrasound measurement that is the most important one.

- While your AAA remains small, it is very unlikely to cause you any problems. You should have the size of your AAA monitored on a regular basis with an ultrasoundscan (surveillance), this may only be needed every three years for the smallest aneurysms.

If I have an abdominal aortic aneurysm what is the risk of it bursting? If your AAA is small, the risk of it bursting is extremely small. The risk of aneurysm bursting increases as the size of the aneurysm increases. For a 30 mm AAA the risk of it bursting within one year is about one in 2 000 (0.005%) for men and one in 500 (0.02%) for women. For a 50 mm aneurysm the risk is about one in 150 (0.66%) for men and one in 30 (3.3%) for women. It is known that the risks of aneurysm rupture increase for aneurysms larger than 55 mm.

- For larger AAAs, the risk of surgical repair are considered to be lower than the risks of rupture. Therefore, most patients with a large AAA are offeredrepair.

We are less certain of the risk of rupture of AAA between 55 – 70 mm but the risk may be up to one in 10 (10%) per year, increasing to about 30% for even larger aneurysms.

What can I do to stop an aneurysm growing larger? At the moment, there are no treatments (drug, diet or exercise) will stop your AAA getting bigger. However, if you are a smoker your aneurysm to grow more quickly.

- Stopping smoking will reduce the chance of your aneurysm growing quickly.

If I have an aneurysm will it affect other parts of my body or my general health? Having an AAA is often a warning signal of disease in other blood vessels, including those supplying the heart. This is not a direct effect of having an aneurysm. It is just that the same things that cause aneurysms such as smoking also cause disease in other blood vessels. Therefore, your doctor may recommend that, in addition to improving your physical fitness, you take one or more drugs to reduce your chance of having heart problems or a stroke in the future.

- We recommend that all people diagnosed with an AAA should be prescribed a cholesterol lowering drug (statin) to reduce the risk of other cardiovascular diseases. Physical exercise is not contraindicated and is encouraged.

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. For many patients AAA repair may not be needed in their lifetime.

- We recommend that for men, if their AAA grows to the size of 55 mm or more, they should be referred to a surgeon for consideration of surgery to repair it.

It is known that aneurysms in women are more likely to suffer a burst AAA at smaller sizes than men, but surgery to repair an aneurysm is riskier for women than for men.

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Therefore, repair of an AAA in women is often considered at slightly smaller AAA size than in men. In some countries there are restrictions on driving if you have a large AAA and you should check with the office issuing your driver's licence.

What happens if I am referred to a vascular surgeon to discuss surgery? When you are seen by a vascular specialist to discuss your AAA, the main question that will be considered, is whether you would benefit from an operation or not. Not everyone with an AAA would benefit from having it repaired. This is because of the risks associated with age and general health of the patient. If the risks associated with AAA repair are greater than the risk of the aneurysm bursting, then surgery is not recommended, although this may be reconsidered if the situation changes. If AAA repair is considered, the patient is likely to be sent for a Cat scan, which provides more detailed information about an AAA. This involves the injection into a vein in your body of dye that can be seen on the scan. This dye clearly reveals the details of the arteries and the aneurysm. A CTA scan involve a small amount of radiation but is a good method for seeing the blood vessels and parts of the aneurysm that cannot be seen on ultrasound (such as the parts of the aorta in your chest).

Two forms of surgery are commonly performed: open operations and endovascular (keyhole) operations.

We recommend that in people who are fit for both open repair and endovascular repair (keyhole surgery), the decision about which type of operation to have should be based on the personal preference of the patient. This decision should be made in consultation with a vascular surgeon. Factors included in the decision making process include the shape of the aorta (is it suitable for keyhole surgery?) and the general health of the patient (what are the risks of surgery?). In patients who are at slightly higher risk of AAA repair, because they have other health problems, we recommend that endovascular repair should be performed.

For men, the risk of dying from a complication during or immediately after planned surgery is about one in 29 (3.4%) for open repair and one in 140 (0.7%) for endovascular repair. Risks of surgery are higher in women, about one in 18 (5.6%) for open repair and one in 45 (2.2%) for endovascular repair.

How is an operation to repair an abdominal aortic aneurysm performed? An open operation to repair an abdominal aortic aneurysm is performed through a large cut in the tummy. The aorta is identified at the back of the tummy and the blood flow through the aorta temporarily stopped. The aneurysm is then replaced with a material graft that is stitched in place and the blood flow through the aorta then restored (Fig. 14B).

An endovascular operation is carried out through smaller cuts or punctures in the groin. Using Xray control a spring loaded graft (also called stent-graft) is passed up from the arteries in the groin into the aorta (Fig. 14C). Once the graft is in the right place it is released. Often three or four graft pieces are required but once completed the endovascular graft takes the strain off the wall of the aneurysm. Not everyone can have an endovascular aneurysm repair. One of the things surgeons assess, when seeing patients with abdominal aortic aneurysms, is their suitability for an endovascular repair. About 70% to 80% of people with aneurysms are suitable for an endovascular repair.

What are the main advantages and disadvantages of an open and an endovascular abdominal aortic aneurysm repair?

Type of AAA surgery	Advantages	Disadvantages
Endovascular repair, keyhole	Smaller cuts Can be done under local anaesthesia Shorter hospital stay Quicker recovery Lower risk of death after the operation	Needs close monitoring after repair (surveillance) Increased radiation burden Higher risk of further operations to prevent rupture
Open surgical repair	Lower risk of further operations in the future Lower radiation burden Reduced need for further scanning in future Possible better long term survival	Big cut in the tummy Needs general anaesthesia Longer hospital stay Slower recovery Five times higher risk of death after the operation

One year after the operation there is no difference in patient quality of life between the two types of AAA repair. Three years after the operation there is no difference in survival by type of AAA repair.

What happens if I am not fit enough to have an operation to repair my aneurysm? In some people the risks of surgery to repair an aneurysm are higher than usual. For example, people with lung disease or kidney problems are more likely to suffer complications after surgery than those without.

- When the risk of surgery is greater than the risk of an aneurysm bursting surgeons will normally recommend that an operation is delayed until the aneurysm gets bigger or that it is not done at all.

There is very limited evidence about the best way to care for you, if your physical fitness for surgery cannot be improved. In patients who are unfit, having an aneurysm repaired is likely to stop it bursting, but there is no evidence that such an operation will prolong life.

- If you are a smoker, then stopping smoking will reduce the risk of your aneurysm growing and bursting.

If the patient chooses ahead with an aneurysm repair, the average risk of dying from the operation is about 7% (1 in 14, compared with between 1 in 50 or 1 in 100 in physically fit patients). It should be noted that this average risk is for all unfit patients. Many people will have risks higher than this and a decision about surgery will have to be made based on the advice from a surgeon and an anaesthetist at the time an operation is being considered on an individual basis.

New treatments to stop both small and large AAAs increasing in size and bursting are being developed and assessed, but there is no good evidence yet.

What happens if an aneurysm bursts? If an aneurysm bursts (ruptures) this is a medical emergency. If you have an aneurysm and suddenly develop severe back or abdominal pain, or collapse it is important to seek medical help immediately and make sure you inform the doctors and nurses treating you that you have an AAA. Unfortunately, many people do not survive aneurysm rupture. In those people who reach hospital an emergency operation can be performed. This is much higher risk than planned surgery. About one in three people who have an operation for a ruptured AAA will not survive. Many people who do survive will take many months to recover or suffer long term physical disability. Given these risks some patients choose not to have a ruptured aneurysm repaired despite the fact that almost all patients with a ruptured aneurysm will die from this within a few days, without an emergency repair.

Ruptured aneurysms can be treated using the same operations as for planned surgery.

- Based on recent evidence we recommend that patients with a ruptured aneurysm who are suitable for an endovascular repair should have this as the first option wherever possible.

Rare causes of abdominal aortic aneurysm. Most aneurysms are caused by a combination of factors, such as an individual's genetic background, that predispose certain groups to the development of an AAA and environmental factors, such as smoking, that in combination lead to damage of the structure of the aortic wall and the formation of an aneurysm. In some rare cases an AAA can be caused by other factors, including infection and genetic causes. It is harder to recommend treatments for these rare aneurysms because we generally know less about diseases that are uncommon.

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. If your doctor thinks your AAA is due to one of these causes, they will tell you this and explain what treatment would be best for you. If there are strong genetic causes, patients will be advised and treated by a joint team of clinical geneticists and vascular surgeons. Open repair may provide a better treatment if repair is recommended.

How was this information developed and what should I know before reading the full document? The above information is a summary of the overall guidelines for clinicians, which has been produced by the ESVS AAA Guidelines Committee. This committee was set up to review all the available medical evidence about AAAs and make recommendations about how they should be managed. As part of this process all pieces of evidence are considered. A decision is then made by the committee whether the evidence is strong enough to make a firm recommendation that all doctors should follow. In case of only limited evidence a weak recommendation to be considered is made. In some areas there is no, or little, evidence available on which an expert consensus recommendation can be made.

The committee therefore makes a decision about whether one particular treatment is one that experts would agree is the best. For each treatment being considered the committee then awards a grade from A (best quality evidence) to C (no real evidence) as well as a class of recommendation from I (strong recommendation and an agreement among experts that the treatment is beneficial, useful or effective) to III (agreement that the treatment is not effective, or even harmful).

This section on information for patients has been put together and reviewed by patients.

Where can I get more information? You can ask your local screening programme or vascular surgeon. There is lots of information available on the internet, but it is not always accurate, may not cover all the treatment options and can be difficult to read.

Listed below are some links to online information for patients.

Canada https://decisionaid.ohri.ca/AZsumm.php?ID=1428

NHS https://www.nhs.uk/conditions/abdominal-aortic-aneurysm-screening/

The Netherlands: https://sdm-library.medify.eu/surgery/index_keuzehulp-aneurysma_nl.html (also in English)

Sweden: https://assets.ctfassets.net/e8gvzq1fwq00/6ITYvSaZj1Qo5MHvY1XUcU/38860a8cedf42d4c332b89fcd43b9ed0/Aortasjukdomar_2019_WEB_Final.pdf

12. UNRESOLVED ISSUES

The GWC identified key issues relating to the management of abdominal aorto-iliac artery aneurysms that need to be addressed to better define future guidelines. These include the following:

General issues

The vast majority of evidence supporting recommendations in these guidelines originate from Europe and North America, and it is unclear how or if this can be transposed to different geographical, ethnic and social settings. Also, the evidence base derives mainly from male dominated or male only studies, while data on women are insufficient.

A general problem in the aortic field is a lack of high quality data. Frequently we only have retrospective, single centre, data to rely on, and many recommendations are consequently based on Level C evidence and are to be considered more like expert consensus recommendations. The interpretation of such data is challenging for many reasons. Single centre reports are typically subject to publication and confirmation bias. Industry involvement introduces a commercial special interest, which further risks the objectivity of the data. The output of unbiased high quality data is therefore a generally high priority issue within the AAA area.

Artificial Intelligence techniques such as machine learning holds great promises to manage, analyse, and use large datasets to develop applications within the healthcare sector. This includes automated imaging analyses, diagnostics, planning and follow up. Continuous follow up of new EVAR devices for early detection of failure is perhaps of most interest in the near future. In the longer term, we predict an even greater and pervasive impact, and it is important that vascular surgeons are involved in the continued development of the field.[1263]

Service standards

How should the future care of patients with aorto-iliac aneurysmal disease be organised? Particularly important but also controversial are the issues of centralisation and surgical volume. There is clearly a strong relationship between volume and outcome, but whether this can be further refined by adjusting to individual and centre outcomes is unknown. The volume—outcome relationship for OSR is naturally linked to the peri-operative period and is thus easy to study. For EVAR, the long term durability is of greater importance, but also more difficult to study and therefore more difficult to determine.

Related to that, how can open surgical skills be acquired and maintained as more cases are treated with endovascular technology, especially since surgical volume seems to be paramount to OSR outcomes (*vs.* EVAR). Should open surgery be centralised in the near future? The decreased exposure and simultaneous increase in complexity of cases reserved for open aortic surgery has also created a conundrum for vascular surgery training. Whether simulation training and or dedicated programmes can effectively compensate for the decrease in OSR training remains uncertain.

Screening

The changing epidemiology has challenged the future of AAA screening. The combination of decreasing smoking prevalence and improved cardiovascular prevention has generally resulted in a significant decrease in the prevalence of AAA. On the other hand, longevity is increasing, and may be accompanied by the development of AAAs at an older age. If targeted screening for high risk groups, or adjusting the timing of screening, can improve cost effectiveness of general screening, remains unknown. Also, strategies to improve screening uptake should be explored in future research.

Secondary cardiovascular prevention combined with AAA screening could have a major impact on the overall health promoting effect of an AAA screening programme and needs to be evaluated properly. In addition, extended screening programmes, targeting multiple disease processes, needs further assessment.

Management of patients with a small abdominal aortic aneurysm

There is no consensus on how to place callipers in ultrasound assessment of AAA. The choice of method has a major impact on diagnostics, follow up routines and treatment decisions. Although having a uniform measurement method is desirable, none of the existing methods seems superior in all aspects.

Radiation exposure has emerged as a potentially major occupational hazard in modern vascular surgery, causing safety concerns for healthcare workers and patients. This is most relevant for high radiation environments such as EVAR and even more so for complex EVAR. How to improve radiation safety behaviour is a key question demanding great attention. Furthermore, new upcoming techniques that allow endovascular navigation without Xray based fluoroscopy have shown promising preliminary results. If or when these techniques can be transferred into clinical practice on a wide scale is as yet unclear.

No specific medication has been shown to unequivocally reduce growth or decrease rupture risk. Statins may have this effect, but since they are already recommended for secondary cardiovascular prevention in patients with AAA, placebo controlled studies are not possible. The most promising drug candidate at the moment is metformin, the world's most widely used antidiabetic drug. There are several ongoing RCTs evaluating the effect of metformin on AAA growth, including the Metformin for Abdominal Aortic Aneurysm Growth Inhibition (MAAAGI) trial,[1264] the Metformin Aneurysm Trial (MAT),[1265] the Limiting AAA with Metformin Trial (LIMIT),[1266] and the Metformin Therapy in Non-diabetic AAA Patients (MetAAA Study)[1267] but no results are yet available. Drug coated balloons or EVAR devices for delivering drugs to the aortic aneurysm is a technology still in its infancy, but which holds great potential to dramatically change the treatment of small AAAs.[1268]

The impact of cardiovascular secondary preventive medical treatment in patients with AAA and refinement of pre-operative assessment should be studied in close collaboration with other societies and guideline groups. Specifically, there is reason to clarify AAA specific LDL target values[1269] and BP limits.[210,1270] Furthermore, the risk benefit ratio for platelet inhibitors in patients with AAA is debated and needs to be clarified.

The optimal size threshold for repair in men remains unclear. The RCTs only show that surgical repair is not worthwhile < 55 mm. This has been taken as proof for 55 mm as generally accepted threshold for when repair should be considered. However, the fact is that the evidence for it is weak, and accumulated data indicate that the limit should perhaps be higher. Effort should be put into defining a more patient specific threshold for repair. The development of better predictive tools for individual rupture risk including bio-markers, functional imaging, and morphology based indicators should be the subject of long term research projects.

The size threshold for considering AAA repair in women is even more an area of uncertainty requiring further research. The Women's Aneurysm Research: Repair Immediately Or routine Surveillance (WARRIORS) trial is an upcoming international RCT evaluating whether women with small asymptomatic AAAs would benefit from being offered EVAR at smaller diameters than men and smaller diameters than recommended in current clinical guidelines.

Surgical treatment of abdominal aortic aneurysm

The rapid technological development is an inherent challenge within the endovascular field. Constant upgrades and modifications and with several actors involved, make it extremely difficult to get reliable data about durability, which is of the utmost importance. Device related complications or problems are rare and difficult to detect and study in single centre environments. RCTs although representing the highest LoE will eventually become outdated under these circumstances, and therefore cohort data and registry data will be the main means of continuously updating our knowledge. The behaviour of the later generations of low profile stent grafts is an ongoing research area of great importance.

Responsible introduction of new products is important, for ethical reasons as well as for the credibility of our vascular surgical discipline. In particular, device related complications should be studied in large collaborations. RCTs comparing devices are very difficult and may already be outdated once the results become available. Low profile stent grafts, disruptive technologies based on polymer sealing, or adjuncts like anchors or chimneys are examples of such technologies. Although regulatory bodies have recognised the need to be more stringent, it is a responsibility of clinicians to contribute to registries or collaborative studies, particularly when novel or disruptive technologies are involved. Automated systems of active surveillance related to the use of devices would be a good topic for further research. The new EU MDR will affect the access to and development of devices in Europe, the question is how? The risk of stagnation of development is palpable, but that is perhaps not only a bad thing. However, it is important that the regulatory framework does not cause the European market to be deprived of modern treatment options and fall behind in access to innovative solutions.

The benefit of pre-emptive embolisation of side branches or non-selective sac embolisation during EVAR has been investigated, including with RCTs, but the true benefit remains elusive. Also, cost effectiveness and safety have not been sufficiently explored. Additional high level evidence focusing on hard endpoints such as survival or rupture are necessary to justify a broad change in practice.

The use of permissive hypotension has been advocated for the management of ruptured AAA. However, the proof of benefit is mainly derived from trauma studies, with significantly different characteristics. Further studies on the use of permissive hypotension, the ideal BP target, or the benefit of actively lowering BP in the setting of ruptured AAA would be desirable. Furthermore, aortic balloon occlusion, or endoclamping, has been proposed as a way to preserve vital organ perfusion before and during ruptured AAA repair. However, evidence is insufficient to support routine use. Further investigation should aim to clarify the ideal target populations and timing of aortic balloon occlusion in this context.

Radiation exposure has emerged as a potentially major occupational hazard in modern vascular surgery, causing safety concerns for healthcare workers and patients. This is most relevant for high radiation environments such as EVAR and even more so for complex EVAR. How to improve radiation safety behaviour is a key question demanding great attention. Furthermore, new upcoming techniques that allow endovascular navigation without Xray based fluoroscopy such as Fiber Optic RealShape (Philips, Eindhoven, The Netherlands)[1271,1272] and electromagnetic tracking systems,[1273—1275] have shown promising preliminary results. If and when these techniques can be transferred into clinical practice on a wide scale is yet unclear.

Post-operative follow up

Although stratification of follow up based on the estimated risk of complications up to five years is recommended, the exact frequency of imaging remains debatable, as the current practice of yearly exams for higher risk patients is based on very little evidence. Moreover, the frequency of surveillance after five years is very scarcely supported by evidence and may not be influenced by prior risk estimates. Since conducting RCTs in this area is very challenging, collaborations using large, high quality registries may be the preferred methodology.

The management of endoleak, particularly T2EL and occult endoleak with sac growth, is a major clinical problem. Given the high failure rate of current endovascular strategies for resolving T2EL, no preferred strategy is currently recommended. Future research on methods for improved identification of hazardous T2EL and effective endovascular methods of repair is necessary. Furthermore, a clear strategy to improve the diagnosis and classification of visible endoleaks and reveal occult endoleaks is warranted. In this context, dynamic CTA is a promising new technology that needs to be evaluated in clinical practice. The proposed step up diagnostic strategy present in section 7.4.3. requires validation and refinement. The clinical relevance of non-shrinking AAA after EVAR and possible link with late overall survival is another related topic that deserves further research.

With the development of artificial intelligence algorithms based on image analysis, it is possible that risk prediction after EVAR, may become more precise and less dependent on subjective analysis or expectations. Integration of additional aspects (genetic, environmental, pharmacological) may refine risk prediction even further. Recently, a European artificial intelligence based multicentre study (VASCUL-AID) was initiated, which evaluates predictors for AAA progression (www.vascular-aid.eu).

Complex abdominal aortic aneurysm

In juxtarenal, pararenal or type IV TAAA the indications for repair are less clear than for standard infrarenal AAA. The risk of rupture is assumed to be similar, but this is not well demonstrated while the operative risk is generally considered higher. Better quality evidence is necessary to support treatment decisions.

While preference should be given to customised endovascular solutions, these may not be readily available and off label solutions may be the only alternative to OSR. The role of these procedures, their durability and specific complications require further evidence. In the long term, it would also be desirable to move away from time consuming and costly customised solutions in the elective situation, and the development of universal and durable off the shelf solutions is warranted.

The issue of cost effectiveness of complex AAA repair in general, and of complex endovascular repair with specially designed stent grafts (CMD) in particular, needs further analysis.

More research is needed to better understand the reason for the reported poor long term survival after fEVAR; is it merely a study methodological phenomenon due to uncompensated patient selection biases or is endovascular treatment associated with as yet unknown adverse long term effects?

Iliac aneurysm

Currently, iliac aneurysms are considered as a whole, without specifying anatomical locations. However, it is possible that rupture risks differ from common iliac to external or internal iliac aneurysms.

Miscellaneous aortic problems

Rare diseases require multicentre and probably international collaborations. Therefore, we support the creation of international registries for mycotic AAA, Inflammatory lAAA, PAU, IMH, pseudoaneurysms, saccular aneurysms, and isolated dissection, focusing on epidemiology, medical treatment, indications for treatment, surveillance in patients with genetic disorders, and outcome after OSR and EVAR.

Patient selection for bridging or definitive endovascular management of mycotic aneurysms remains uncertain. Further research focusing on identifying the ideal endovascular candidate is necessary. Also, the optimal medical and operative strategies for inflammatory AAA are still unclear. This includes corticosteroid doses, alternative anti-inflammatory medications, and operative management, although EVAR is generally preferred due to the iatrogenic risk of OSR. The natural history and rupture risk of saccular aneurysms is largely unknown. Longitudinal data on this special type of aneurysm would help clarify and avoid possible overtreatment.

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APPENDIX A. SUPPLEMENTARY DATA

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